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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.
MEDICAL EMERGENCY TEAM ANALYSIS

Medical Emergency Teams (METs) or Rapid Response Teams (RRTs) were introduced after being incorporated into the Institute for Healthcare Improvement (IHI) 100,000 Lives campaign in 2005. This was furthered by the Joint Commission’s 2009 National Patient Safety Goal 16 - to improve the identification and response to clinical deterioration. METs differ from traditional code teams in a number of ways: they aim to improve the medical safety of ward patients with unexpected clinical deterioration, aid in the diagnosis and treatment of rapid cardiac, pulmonary and neurologic dysfunction; and may even be used to address end-of-life issues with patients and families. METs are initiated by any member of hospital staff when a patient fulfills predefined criteria. Hospitals are now implementing the second step: measuring their effectiveness. Five single small center trials supports METs, however the MERIT trial failed to demonstrate a benefit of patient morbidity and mortality. A Cochrane review failed to demonstrate a benefit. The adverse effects of METs include: additional cost, desensitization to emergencies, ICU personnel away from unit and a decreased sense of responsibility for ward staff. At Atlanticare Regional Medical Center (ARMC) there is anecdotal evidence among the Internal Medicine Residents that METs are called with a reproducible predictability according to nursing shift change (07:00 and 19:00), nursing medical delivery times and patient assessment. Data of MET calls from the ARMC Mainland Campus were collected from August through November 2011. Date and time of each MET were obtained from operator records. Time of day was grouped into one-hour and fifty-nine minute segments, (6:00am to 7:59am, 8:00am to 9:59am, etc). From August through November 2011 there were a total of 300 MET calls and 7015 admissions (43 METs per 1000 admissions). When combined over the four month period, the average number of METs per time segment was 25 with a standard deviation of 12.3. To assess distribution of MET calls Z-test was used. The number of METs followed a cresendo-decresendo pattern increasing shortly after nursing shift change. The peak occurred between 10:00am and 12:00pm (46 MET calls, Z-score = 1.71, p-value = 0.04); the time associated with medical delivery and nursing assessment. The nadir occurred between 12:00am and 1:59am (7 MET calls, z-score = -1.46, p-value = 0.07). ARMC Internal Medicine Residents’ anecdotal evidence was correct: there is a statistically significant increase in the number of MET calls which occurs after nursing shift change, with assessment and morning medication delivery. This new awareness will be used to implement new safety precautions for staffing and availability of the response teams.

BUYING TIME: QUALITY IMPROVEMENT PROJECT TO DECREASE PATIENT CALLS FOR MEDICATION REFILLS IN A RESIDENT RUN CONTINUITY CLINIC

Background: The internal medicine resident run continuity clinic noted a large volume of calls/faxes for medication refill requests (MRQ), and that each one demanded fifteen minutes of staff time. Objectives: Utilize the Plan-Do-Check-Act quality improvement process to decrease the amount of time devoted to MRQs by 1) Quantifying and identifying patterns among the MRQs 2) Development and implementation of an education plan for practitioners and patients, and 3) Re-assessing the impact of the educational plan by resurveying the volume of, and reasons for, MRQs. Methods: BASELINE DATA: Clinic nursing staff would log each MRQ over a 3 month period noting time between the last office visit and receipt of the MRQ, and method of request (patient walking in, call to the prescription line, call to main clinic number, pharmacy request via phone call or fax), after which an action plan would be developed and disseminated to the practitioners (POST-ACQUISITION EDUCATION). Four months later, the nurses would conduct a second survey and classify the MRQ as patient or caregiver error (POST-EDUCATION REASSESSMENT). Results: BASELINE DATA: 15.2 MRQs/day (655/43 days). Patients are typically asked to return in 12-24 weeks, but 61% of the MRQs were received within 8 weeks of a patient’s last visit indicating that physicians were not providing enough refills. 68% were routine requests, 7% prescription error, and 25% due to patients not returning as directed. Pharmacies generated 8 phone calls/messages per day, sent 171 faxes to the machine in the clinic, and 107 to a machine
on a different floor. The POST-ACQUISITION EDUCATION consisted of: 1) Instructing caregivers to provide a prescription with enough refills to get them to the next visit, plus one extra month, 2) Educating patients to bring all bottles to each visit, and 3) Pharmacies were asked to fax all MRQs to a single number instead of calling. REASSESSMENT: 2.8 MRQs/day (111/39 days) representing an 81% decrease in the volume of daily requests, with 64% due to patient error, and 10% were due to physician error. Phone calls from pharmacies dropped by 90% to 33 (~1/day). Patient calls dropped from 3/day to 1.8/day. Discussion: A simple caregiver and patient education program facilitated an 81% decrease in the volume of calls/faxes for medication refills in this resident-run continuity clinic. The 12 fewer requests/day decreased the time burden on an already limited clinical staff by 3 hours per day, and the clinical staff has stated a high level of satisfaction with the change. While faxes still require staff attention, the combination of fewer calls and transition to a fax located within our clinic reduced the time burden and number of interruptions imposed upon the staff.

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PRE-ROUNDING: A TOOL TO ACHIEVE BETTER SLEEP, SUPERVISION AND SAFETY

Background: The implementation of the 2003 duty-hour regulations by the ACGME across all specialties sparked considerable interest in strategies by which training programs could deliver excellent and safe medical care while reducing medical errors either due to resident fatigue or as a result of handoffs due to break in continuity of care. Though extensive work has been done on how the change in resident duty-hours has impacted patient safety and outcomes, limited evidence exists on strategies that optimize work schedules, decrease the risk of handoffs, improve the resident-training experience and enhance patient safety. Hypothesis: In this study, we propose that “pre-call” rounding, defined as “time spent by the covering team discussing active medical issues in an inter-disciplinary setting”, enables residents to deliver better cross coverage care, by early identification of acute medical issues and timely intervention. Specific aims: To analyze the effect of “pre-call” rounding on 1. Patient outcomes by assessing patient safety using the number of acute medical responses (AMRs = number of rapid responses (RRs) and code blues (CBs). 2. Optimizing work schedules by normalizing the number of floor calls (phone (P) and pagers (PCs)) to the floor census.  
Design and Methods: Design: Single institution, observational study design. Analyses adjusted for daily census. Setting: Cooper University Hospital, Camden, NJ Measurements: Number of floor calls (P and PCs), Number of AMRs (RRs +CBs), Average time spent pre-rounding (T), Floor census (C)  Results: The total number of AMRs was dramatically decreased, by 42%, in the pre-rounding group compared to the control group (total ± sem, Control 16 ± 0.5, pre-rounding 7 ± 1.0, t-test .016). Patient safety was assessed by normalizing the total number of adverse events to the daily floor census. A 47% decrease in the mean number of AMRs per patient per day was seen in the pre-rounding group compared to the control group (mean ± sem, Control .04 ± 0.5, Pre-rounding .02 ± .005, t-test .01). “Pre-rounding” was also associated with a significant decrease in the number of calls/patient. (33% decrease, Control 0.77 ± 0.04, Pre-rounding 0.44 ± 0.1, t-test 0.0002). Limitation: Study did not include “highest risk” ICU patients. Conclusion: Use of “pre-call” rounding improves patient outcomes by decreasing number of acute medical responses, thus enabling residents to deliver better and safer patient care, by early identification of medical issues and appropriate timely intervention. Further, our results suggest that “pre-call” rounding also helps optimize call work schedules by decreasing number of calls to floor team, in turn enhancing efficiency and decreasing resident fatigue.

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POTENTIAL CONSEQUENCES OF OVERLOOKING ABOMINAL AORTIC ANEURYSM SCREENING

Abdominal Aortic Aneurysm is a dilatation in the abdominal aorta > 3 cm that results from degeneration of the arterial wall media. AAAs occur five times more frequently in men, with increased incidence in advanced age. Additionally smoking is the strongest independent risk factor for development of AAAs, with dose effect up to five-fold increase. Aneurysms > 5.5 cm in diameter are at markedly increased annual risk of rupture. Those that rupture are associated with a 50% mortality prior to reaching a hospital, and up to an 80% in-hospital mortality for an overall mortality of 75-90%, resulting in about 14,000 deaths per year. AAA screening has been shown to significantly reduce the morbidity and mortality associated with AAA rupture. Screening is done
by ultrasound, an inexpensive non-invasive test which detects AAA with close to 100% sensitivity and specificity. The US Preventive Task Force Guidelines recommend one-time screening for AAA by abdominal ultrasound in men &gt;8805; 65 years of age who ever smoked in their lifetime. The guidelines recommend against routine screening in women and make no recommendation for males who have no smoking history. Although AAA screening has been well-documented to decrease mortality, there have been no official screening guidelines implemented in the University Hospital outpatient setting, and there are no physician reminders in the computer system. Last year, a study was conducted to measure resident compliance rates for screening. Of the 82 patients who met criteria and were eligible for screening ultrasound, three had ultrasounds ordered on or before the date of clinic visit (3.7%), and two had ultrasounds completed (2.4%). This study revealed poor AAA screening among eligible patients, which is a consistent problem found not only in academic centers, but at the national level too with screening compliance rates as low as 12.9% to 36% (Eaton et al., Lionze et al.). Based on last year’s results, a study design was implemented in the form of mini lectures to increase compliance among internal medicine residents. Three ambulatory cohorts were provided mini lectures, while two other resident cohorts remained as a control. After the intervention, of the 46 patients who met criteria for screening, 20 patients had ultrasounds ordered (43.5%). In contrast, of the 20 patients who met criteria for screening in the control arm, only two patients had ultrasounds ordered (10%). Based on these preliminary results, educational campaigns to increase awareness of AAA screening may result in increased compliance with AAA screening. Future interventions such as automated EMR reminders may increase AAA compliance even more until we reach our goal of 100% compliance.

### POSTER PRESENTATIONS

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**ATRIAL MYXOMA PRESENTING AS POSTERIOR CIRCULATION MULTI-INFARCTS: A CASE REPORT**

A 48-year old male with no known past medical history presented with intermittent right lower extremity numbness, headache, and blurring of vision for 6 months. Seven hours before admission, he developed sudden onset of severe 10/10 generalized headache with bilateral blurry vision and right thigh paresthesias and went to the emergency department where he had transient photopsia. There was no fever, loss of consciousness, seizures, or meningeal signs. On admission, he had normal cardiovascular examination, visual acuity, visual fields, cranial nerves, motor strength, light touch, pain, and vibration in all four extremities. The rest of his physical examination was normal. CT scan of the head, blood tests and CSF examination were all normal. He was admitted with new onset complicated migraine vs migrainous stroke. MRI revealed multiple acute micro-infarcts in the posterior circulation distribution and innumerable hemosiderin deposits within the supra and infratentorial brain which has the appearance of cerebral amyloid angiopathy or cerebral vasculitis. Echocardiography showed a large serpiginous mobile 6 cm mass in the left atrium suggestive of a myxoma. Our impression was multiple posterior circulation embolic strokes secondary to an atrial myxoma. He was transferred to another hospital for an open heart surgery to resect the atrial mass. Pathology confirmed the diagnosis of a 2 cm atrial myxoma with adherent thrombi. **DISCUSSION:** Cardiac myxomas are rare with an annual incidence of 0.5 per one million population. Myxomas can arise from any of the cardiac chambers but 75-88% occur in the left atrium. Left atrial myxomas may be asymptomatic or may present with constitutional, cardiovascular and/or embolic symptoms. More than 2/3 of these emboli migrate to the central nervous system. Cerebral emboli are often recurrent and their presentation ranges from progressive multi-infarct dementia to massive embolic stroke causing death. Our patient had embolization to the posterior circulation which is much less common than anterior circulation. In one retrospective analysis, pure anterior circulation involvement was found in 64% of patients, pure posterior circulation involvement in 11%, and mixed in 22%. Both thrombi attached to a myxoma, as well as tumor fragments may embolize. Cerebral emboli that involve tumor fragments can form aneurysms and metastases that can grow and mimic the clinical picture of CNS vasculitis or infective endocarditis. This aneurysm may be due to VEGF secretion and can develop several years after resection of the myxoma. The multiple hemosiderin deposits in our patient’s brain MRI which was interpreted by the radiologist as possible amyloid angiopathy or cerebral vasculitis is concerning because of the possibility of future development of cerebral aneurysms. Continuous close follow up will be needed.
RAPIDLY PROGRESSIVE ENCEPHALOPATHY IN A 73-YEAR-OLD FEMALE

A 73-year-old Haitian female presented with a three day history of headache, right shoulder pain, chest pain and difficulty sleeping. Her examination revealed normal vital signs. Lab tests were within normal limits. She was discharged home with oxycodone/acetaminophen. She returned to the ED three days later because of fever, confusion, restlessness, hallucinations, dysphagia and inability to sleep. She was tachycardic and disoriented. She had no rash, meningeal or focal neurological signs. Urine microscopy showed numerous RBCs and WBCs. Urinalysis was positive for protein, leukocyte esterase and ketones. Head CT scan showed no abnormality. A clinical diagnosis of urinary tract infection and delirium was made. She was admitted and started on IV fluids, ceftriaxone, haloperidol and Trazodone. Within 48 hours of admission, she developed difficulty swallowing, excessive salivation, incoherent speech and focal facial and upper extremity tremor-like movements. She became febrile and more disoriented with hallucinations and agitation. Vancomycin and acyclovir were added to her antibiotic regimen. Her CSF showed normal protein and glucose levels and 7 WBCs/μL with 55% lymphocytes. She was transferred to the ICU and within 24 hours, she developed respiratory failure. VEEG showed focal status epilepticus. CSF cultures and PCR were negative for bacteria, western equine encephalitis, CMV, adenovirus, influenza, HSV, enteroviruses and VZV. On hospital day 11, her serum, CSF, saliva and nuchal skin biopsy specimens were sent for rabies screening. By hospital day 12, she had developed multiple endocrine and autonomic abnormalities. She became unresponsive to painful stimuli. A positive rabies report was received on hospital day 17. Further inquiry revealed that the patient had a dog bite in her native country three months earlier. Life sustaining measures were withdrawn on hospital day 18. Autopsy revealed Negri cytoplasmic inclusions in numerous neurons within the brain. Discussion and Recommendation: Rabies is a rare and fatal preventable zoonotic disease caused by Lyssavirus. The virus is most commonly transmitted to humans from rabid animals through a bite. This case is the second rabies-related death in New Jersey in the last 40 years. Clinical features are often non-specific. Aerophobia and hydrophobia are pathognomic for rabies and occur in about 50% of patients. A clinical diagnosis of rabies can be made if there is a history of exposure to a potentially rabid animal or rabies-specific signs of hydrophobia or aerophobia are present. Once symptoms appear, diagnosis can be made by a variety of techniques. Post mortem, the standard diagnostic technique is to detect rabies virus antigen in brain tissue or identify the characteristic Negri inclusion bodies in neurons. The non-specific neurobehavioral features of rabies pose a diagnostic dilemma. We recommend that rabies should be considered in the differential diagnosis of every patient presenting with rapidly progressive encephalopathy of unclear etiology.

LEFT VENTRICULAR AND CORONARY AIR EMBOLISM COMPLICATING PERCUTANEOUS LUNG BIOPSY

Percutaneous lung biopsy is the gold standard for the tissue diagnosis of lung masses. Common risks of the procedure include pneumothorax and pulmonary hemorrhage. Air embolism is a rare but potentially fatal complication. We report a case of air embolism documented on computed tomography complicating biopsy of a lung lesion. A 51 year old male with recently diagnosed melanoma was found to have numerous pulmonary nodules on computed tomography (CT) of the chest. He was admitted following iatrogenic pneumothorax complicating percutaneous biopsy of a lingular segment of left upper lobe nodule measuring 9mm. Left chest tube was placed and the procedure terminated. Repeat biopsy was planned in view of pulmonary nodules likely representing metastases. He was placed in supine position with standard sterile prep and drape. Using CT guidance a 22 guage needle was inserted into the pulmonary nodule and a fine needle aspiration biopsy obtained. He suddenly complained of chest pressure and became diaphoretic and bradycardic with heart rate 40 beats per minute. He was started on oxygen therapy and intravenous normal saline. Electrocardiogram was unremarkable. His heart rate spontaneously returned to 90-100 beats per minute and chest pain gradually resolved. CT images showed air within the left ventricle. Gated imaging confirmed air bubbles near the left ventricular apex and within the coronary arteries. He was placed in left lateral Trendelenberg position and transferred to intensive care unit. There was a slight elevation in cardiac enzymes which trended down over
the subsequent 48 hours. Repeat chest CT the following day did not show air within the left ventricle, pulmonary arteries, aorta or coronary arteries. Percutaneous lung biopsy is considered safe and minimally invasive. Complications of the procedure include pneumothorax (most common), local bleeding, hemoptysis and injury to neurovascular structures. Air embolism is a rare complication, especially to the coronary arteries. It may manifest as fatal arrhythmias and circulatory collapse. Thus, it warrants a higher index of suspicion and immediate treatment including administration of 100% oxygen, placing the patient in head dependent left lateral Trendelenberg position.

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A RARE CASE OF INFECTIOUS CERVICAL SPONDYLITIS

Infectious spondylitis is a rare but severe disease, accounting for 2-4% of skeletal system infections. The most common causative agent is Staphylococcal aureus. Other agents include Streptococcus sp, Streptococcus pneumoniae, enterococcus, Klebsiella sp, Escherichia coli, Salmonella sp, Pseudomonas aeruginosa and non-pyogenic granulomatous infections. We report a case of infectious spondylitis involving the cervical spine. A 52 year old male with past history of chronic neck pain since adolescence, cellulitis of left leg, and melanoma s/p removal presented with progressive, sharp, severe, continuous neck pain for 1 day which radiated to both arms, right greater than left. He reported fever without chills, and denied cough, night sweats or recent travel. He also had unintentional weight loss of 40 lbs after a hospitalization for cellulitis. On examination his vitals were BP of 187/92 mm Hg, Pulse of 93/min, regular, respiratory rate of 20/min, Temperature of 98.4 degrees F. On examination, there was restricted range of motion of neck which he was unable to flex or extend his neck, soft diffuse swelling on the posterior region of the neck associated with tenderness and sensory loss of the right third, fourth and fifth fingers. Laboratory findings revealed leukocytosis with WBC 12,800/mm3, anemia with hemoglobin 10.4g/dL and hematocrit 32.2 %, sedimentation rate 114 mm/hr and C-reactive protein 146.91 mg/L. Radiographs of the cervical spine showed fusion of C3 and C4 with a possible “step down” between C4 and C5. Computed tomography of the cervical spine showed marked narrowing with irregularity at the C5-C6 disc base with misalignment of approximately 2.5 mm and marked widening of the prevertebral soft tissue, suspicious for infectious spondylitis. MRI showed small amount of fluid signal within C4-C5 and C5-C6 disc spaces and irregularity of the endplates at C5-C6 consistent with infectious spondylitis. C5 was posteriorly displaced in relation to C6. He was started empirically on vancomycin and piperacillin/tazobactam and his neck was immobilized with a Miami J Collar. Blood cultures were positive for methicillin-sensitive Staphylococcus aureus and he was switched to ceftriaxone. Infectious spondylitis usually present with back pain with or without constitutional symptoms along with neurological symptoms. Most cases develop from hematogenous spread. They may have leukocytosis, elevated ESR and CRP. Imaging studies such as MRI are most helpful in the diagnosis. Blood cultures can identify the causative agent. Biopsy and histology are also helpful in differentiating pyogenic vs granulomatous infection. Molecular diagnostic methods using broad-range 16S rDNA PCR have narrowed the diagnostic gap that existed with traditional culture-based methods, especially in the context of prior antibiotic usage or the presence of fastidious microorganisms. Management includes eradication of infection, restoration and preservation of structure and function of the spine. Appropriate antimicrobials, physical therapy and immobilization are the mainstay of treatment.

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MACROPHAGE ACTIVATION SYNDROME

INTRODUCTION: Macrophage activation syndrome/ Hemophagocytic lymphohistiocytosis (MAS/HLH) is a rare and potentially fatal disease of overactive histiocytes. It should be considered in patients presenting with SIRS with fevers unresponsive to antibiotics, pancytopenia, liver dysfunction, falling ESR and elevated ferritin. CASE: A 28-year-old female presented with a one week history of high grade fever, vomiting, headache, back pain, fatigue and bilateral leg cramps. Her vital signs included temperature- 102.4 F, pulse rate- 120 bpm and BP- 106/65 mmHg. Her physical examination was unremarkable except for facial flushing and dry mucous membranes. No rash was evident. Lab studies include- WBC 1.5, 15% bands, ANC 0.86, hemoglobin 10.8, platelets 87, without schistocytes, AST 368, ALT 203, ALP 337, INR 1.19, aPTT 33.2, D-Dimer 3580,
fibrinogen 126, fibrin degradation products 40, LDH 7335. Hence, there was pancytopenia with liver
dysfunction and coagulopathy. She was initially treated as a case of sepsis, but continued to be febrile despite
broad spectrum antibiotics. Blood, CSF, and urine cultures yielded no growth. Extensive investigations into
sources of infection were negative. CAT scan showed axillary lymphadenopathy, bibasal pleural effusions,
gallbladder, pericholecystic, periportal and perihepatic edema with hepatosplenomegaly. Since pancytopenia
persisted, a bone marrow biopsy was done which showed hypocellularity without evidence of malignancy.
Excisional biopsy of the lymph node was unrevealing. Interestingly, the ESR was normal (18 mm/hr) but ferritin
(2290) and triglycerides (320) were strikingly increased. Her ANA titer was 1:160 with negative ds-DNA and
positive Sjogren’s antibodies. This clinical scenario was highly suggestive of MAS/HLH, possibly secondary to
SLE/Sjogren’s. She was then started on dexamethasone, which rapidly led to the resolution of the fever and
improvement of the pancytopenia. DISCUSSION MAS/HLH can be a primary disease entity or secondary to an
infection, autoimmune disorder or malignancy. The diagnostic criteria for HLH include fever, splenomegaly,
cytopenia, hypofibrinogenemia, hypertriglyceridermia, hemophagocytosis in bone marrow and rash (at least five
criteria for a definite diagnosis). MAS includes falling ESR and high ferritin. 1 In 20% of cases,
hemophagocytosis is difficult to demonstrate in the first bone marrow specimen. 2 The first line treatment is
corticosteroids but cyclosporine A, etoposide, anti-TNF alpha agents and plasmapheresis can also be used.
CONCLUSION This case is presented to highlight the importance of considering a rare but potentially fatal
clinical syndrome in a patient presenting with unremitting fevers. REFERENCES 1. Kumar MK, Suresh MK,
Dalus D. Macrophage activation syndrome. Journal of The Association of Physicians of India. 2006; 54: 238-
240. 2. Gupta A, Weitzman S, Abdelhaleem M. The role of hemophagocytosis in bone marrow aspirates in the

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GLUTEUS MINIMUS TENDON RUPTURE. AN UNUSUAL PRESENTATION AFTER FLUOROQUINOLONE
USE

Fluoroquinolone use is very popular for the treatment of common infections. Their expanded use implicates an
increased incidence of their side effects. A 41 year old female bus driver presented to the hospital complaining
of one day history of right leg swelling that was gradually increasing in size. She had no background medical
history and was not taking any regular medications. A duplex venous ultrasound had demonstrated right
popliteal deep vein thrombosis (DVT). During her admission she was complaining of urinary symptoms. A
urinalysis was performed that was positive to nitrates and leukocyte esterase. She was started on
ciprofloxacin 500mg twice a day empirically. The patient received a total of three doses of ciprofloxacin before
it was changed to another class of antibiotics according to sensitivities for Klebsiella pneumoniae. The patient
was discharged home the following day with warfarin anticoagulation and cephalosporin antibiotics in good
functional status. Three weeks later the patient represented to the hospital. This time she was complaining of
sudden onset excruciating right hip pain that woke her up from her sleep. The pain was radiating into her groin
and thigh. She did not have any history of recent trauma. She was unable to bear weight on her right leg.
Marked tenderness was elicited on palpation of her right hip and she had a generalized reduced range of
motion of her right hip that was more pronounced during hip abduction. An MRI of the hip was obtained at that
time has demonstrated a high grade tear of the gluteus minimus tendon. The only mechanism that could be
implicated for her spontaneous tear of her tendon was her recent exposure to ciprofloxacin. The patient was
managed with adequate analgesia, physical therapy and advice to use crutches for ambulation. She had made
a good recovery. Fluoroquinolone induced tendon injury is a rare but known complication of this antibiotic
class. It is not commonly seen in healthy individuals after administration of fluoroquinolones but there is
increased incidence of tendon tears in patients with renal dysfunction or concomitant use of steroids. The most
common tendon involved is the Achilles tendon. Gluteus minimus tendon tear secondary to fluoroquinolone
use has not been reported in the literature. This case illustrates that fluoroquinolone use can affect any tendon
even on otherwise healthy individuals even with few doses and clinicians should be aware of this potential
complication when choosing antibiotic treatment. References: 1. Khalil Y and Zhanel GG. Fluoroquinolone-
BATH SALTS - BEWARE!

Bath salts poisoning has increased considerably in the recent years, with Poison Control getting over 6,000 calls in 2011. They have been marketed as “fake cocaine” by names like Cloud9, Bliss, Vanilla Sky, Ivory Wave or Red Dove; are very popular among adolescents and are highly lethal as well. We report a case of a 22-year-old man who was brought to the emergency room for a witnessed seizure by his room-mate, who had apparently seen him inhaling bath salts few minutes ago prior to this episode. He was found to be unresponsive and had no gag reflex, dilated fixed pupils 4 mm bilaterally, flushed diaphoretic skin, tachycardia of 110 beats/min, and had a seizure again in the emergency room. He was intubated for acute respiratory failure as well as airway protection and seizures were controlled with ativan. His laboratory workup showed no evidence of infection, negative initial urine toxicology screen, acute kidney and liver injury, and severe rhabdomyolysis. He was admitted to intensive care unit where he continued to receive supportive care and hydration. Urine cathinone screen on a later sample from day 2 was negative. His kidney and liver function were improving slowly and his rhabdomyolysis was improving as well, though he had profound weakness which improved with physical therapy. He was discharged home successfully later after 11 days. Bath salts are increasingly being abused among the young population and the effects can be fatal at times. The main chemical components are 4-fluoromethcathinone and 3,4-methylenedioxyxyprovalerone (MDPV). These agents are snorted, swallowed, smoked or injected. Bath salts, like amphetamines, cause stimulant effect by increasing the concentration of catecholamines like dopamine, serotonin and norepinephrine at the synapses. They can cause varying symptoms including headache, palpitations, nausea, hallucinations, paranoia, panic attacks, violent behaviors, heart attack, liver failure, suicide, and increased pain tolerance. They are not detected by routine urine analysis, gas chromatography-mass spectrometry or urine cathinone can be used to detect recent exposure. Treatment is usually supportive care. Perhaps with this increasing incidence of bath salts poisoning, we should look for it in young adults with drug abuse, especially when routine urine toxicology is negative, and we should include urine cathinone as well in the urine toxicology screen. References: 1. Coppola M, et al. Synthetic cathinones: Chemistry, pharmacology and toxicology of a new class of designer drugs of abuse marketed as “bath salts” or “plant food”. Toxicology Letters. 2012;211(2):144–149. 2. Prosser JM, et al. The Toxicology of Bath Salts: A Review of Synthetic Cathinones. Journal of Medical Toxicology 2011;8(1):33–42.

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REFRACTORY POST-SURGICAL PYODERMA GANGRENOsum: THE ROLE OF MULTIMODAL THERAPY IN ATTAINING DISEASE CONTROL

Objectives: (1) Recognize the diagnosis of pyoderma gangrenosum (PG), and (2) Understand the multimodal treatment options for refractory PG. Case report: A 41 year-old woman with Beckwith-Wiedemann Syndrome suffered third degree burns to her left breast from spilling heated coffee, requiring skin grafting. Recurrent graft dehiscence and infections led to elective bilateral reduction mammoplasty five years later. Following surgery she developed recurrent ulcerations at the suture margins, unresponsive to topical therapy or antibiotics. Intermittent wound cultures grew methicillin-resistant Staphylococcus aureus, and were aggressively treated. Attempted surgical closure and subsequent skin grafting were unsuccessful. Various skin biopsies revealed occasional giant cells, acute and chronic inflammation, and no vasculitis. A short course of steroids was beneficial, but her wounds promptly recurred. She was clinically diagnosed with post-surgical PG, and workup for associated inflammatory disorders was unrevealing. Over one year, she was treated with the immunomodulatory agents hydroxychloroquine, minocycline and dapsone in addition to chronic steroids, without significant improvement. Her wounds progressed, requiring eventual elective bilateral mastectomy. Approximately two months post mastectomy, she developed progressive ulcerations predominantly over her left chest wall, associated with intermittent profuse bleeding. She required multiple blood transfusions and intravenous iron for resultant anemia. She failed treatment with cyclosporine, colchicine, intravenous gamma globulin (IVIG) and mycophenolate mofetil (MMF), and required prednisone doses up to 120 mg/day with occasional intravenous steroid pulses. Surgical wound closure and negative pressure wound therapy were unsuccessful. Local steroid injections at the ulcer borders led to pathergy. She remained high-dose steroid-
dependent for two years, leading to Cushingoid habitus, steroid dysglycemia and obstructive sleep apnea. Eventually, the addition of the TNF inhibitor adalimumab to a regimen of MMF and prednisone facilitated progressive decrease in ulcer size. Hyperbaric oxygen (HBO) therapy was additionally implemented; 60 treatments of 100% oxygen at 2 Atmospheres Absolute led to further improvement. With this multimodal therapy, ulcer dimension progressively decreased from a maximum of 15 x 7.5 cm to 1.5 x 0.5 cm over one year, allowing for gradual prednisone taper to 7.5 mg daily. Discussion: PG is a rare inflammatory and ulcerative neutrophilic dermatosis, with an estimated incidence of 3 to 10 cases per million per year. It may be associated with autoimmune disorders, trauma, diabetes, immunodeficiency, tumor, or medication side effect. PG is a diagnosis of exclusion, without pathognomonic clinical or histological findings. First-line treatments include local wound care and systemic corticosteroids, along with treating the underlying disorder. Other immunomodulatory treatments include cyclosporine, TNF inhibitors, cyclophosphamide, colchicine, MMF, and IVIG. For refractory cases, surgery, negative pressure wound therapy and HBO have also shown benefit. Our report describes a case of highly refractory post-surgical PG, and illustrates the need for collaborative multimodal therapy in achieving progressive disease control.

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HYPERAMMONEMIC ENCEPHALOPATHY AFTER GASTRIC BYPASS SURGERY

Objectives: To review presentation, diagnosis, and treatment of hyperammonemic encephalopathy in a patient with a history of gastric bypass surgery. Case: 42-year-old Caucasian female with a history of obesity and gastric bypass surgery in 2007 presented with altered mental status, nausea, vomiting and diarrhea for four days. On exam she was afebrile with normal vital signs. She was somnolent yet arousable, oriented only to person, and could follow simple commands. No asterixis or other stigmata of liver failure were observed. Her abdomen was obese, non-tender, non-distended and without organomegaly. Eyes: No papilledema, PERRL. Neck: supple, no stiffness, no meningeal signs. Labs: BUN 25 mg/dL, Creatinine 2.12 mg/dL, Alk Phos 95 U/L, AST 31 U/L, ALT 18 U/L, Ammonia 192 mg/dL, Albumin 2.4 mg/dL, ESR 23 mm/hr, CK 159 units/L, INR 1.09, PTT 30 seconds. CBC: WBC 20.1 x 109/L with 1% band forms. She was admitted to ICU started on empiric antibiotics for suspected infection, as well as lactulose and rifaximine. Brain imaging, lumbar puncture and EEG were negative. She was intubated because of deteriorating mental status and hemodialysis was initiated to decrease the ammonia level. Viral hepatitis serology was negative. Iron studies and serum α1-antitrypsin level were normal. Anti-nuclear, anti-mitochondrial, and anti-smooth muscle antibodies were negative. Plasma concentrations of ornithine (28 μM), citrulline (18 μM), and arginine (44 μM) were normal, and blood and urine cultures were negative. The diagnosis of hyperammonemia post gastric bypass surgery was entertained; intravenous levocarnitine was initiated and ammonia levels progressively declined to normal with improvement in mental status. Discussion: Neurological complications of bariatric surgery occur 5-16% of the time, acutely or decades later. Encephalopathy has been described as one of these rare complications, and can be attributed to deficiencies of thiamine, urea cycle disruption and sometimes idiopathic. There have been several cases of hyperammonemia in individuals who have undergone gastric bypass surgery; some die shortly after surgery, and others have a steady decline up to four years post-op that eventually leads to coma or death. A number of these individuals were found to have a previously undiagnosed genetic mutation or an acquired urea cycle disorder due to malnourished states. Confirming urea cycle defects requires either genetic testing or specialized lab testing for enzyme deficiencies. In addition, not all patients have enzyme deficiencies or substrate accumulation on presentation. Treatment includes ammonia reducing agents such as lactulose in addition to nutrient replacement; levocarnitine has been effective given the role it plays in the urea cycle. In conclusion, urea cycle defects should be considered in gastric bypass patients who present with altered mental status.
PUERPERAL MASTITIS RAPIDLY PROGRESSING TO BREAST NECROTIZING FASCIITIS

INTRODUCTION: Necrotizing fasciitis is an uncommon infection of the skin and subcutaneous tissue. Necrotizing fasciitis of the breast is an extremely rare disease; early recognition and treatment is crucial due to the high case-fatality rate. We report here a rare case of necrotizing fasciitis of the breast secondary to puerperal mastitis in a lactating mother. CASE PRESENTATION: A 40 year old woman with no medical history delivered a healthy baby by uneventful spontaneous vaginal delivery one week prior to presentation. She was admitted for same day history of acute onset right breast pain, warmth and redness rapidly progressing to bluish discoloration. She had been breastfeeding and noted redness and pain on right breast without history of trauma. She consulted her Ob/Gyn who started her on dicloxacillin and an anti-inflammatory agent. At 1:00pm on the day of admission she experienced worsening of her symptoms and started having nausea with vomiting and diarrhea. Around 9:00pm she presented to the hospital. In the ED, she had low grade fever, hypotension and tachycardia. Physical exam revealed dark bluish discoloration of the right breast with surrounding erythema and swelling; it was exquisitely tender to palpation without crepitus. The left breast was normal. Laboratory results showed leukocytosis with left shift and a lactic acid of 4.2mmol/L. Non-contrast CT of the chest showed moderate skin thickening overlying the right breast. Prompt sepsis protocol was implemented with antibiotic coverage of clindamycin, vancomycin and piperacillin-tazobactam. Aggressive fluid resuscitation and vasopressors were started. The patient developed respiratory insufficiency and was intubated in the ICU. The initial assumption was lactation mastitis with cellulitis and sepsis, but the rapid progression and the physical findings raised the suspicion for necrotizing fasciitis. A breast surgeon was consulted and the patient was taken to the OR for urgent debridement. In the OR it became apparent there was extensive necrotizing fasciitis and complete mastectomy was performed. On the third day of admission, antibiotics were switched to intravenous penicillin G and cefazolin tailored to the breast milk and tissue cultures which grew Staphylococcus aureus, Staphylococcus epidermidis and Streptococcus pyogenes. Blood cultures were negative. The patient completed ten days of antibiotic therapy; subsequently she was taken to the OR for wound closure and discharged home. She was seen in follow up one month later and was well. DISCUSSION: Despite the rarity of necrotizing fasciitis of the breast, clinical suspicion should be high when there is fulminant tissue destruction or severe systemic signs of toxicity. Since the infection spreads along the muscle fascia, due to its relatively poor blood supply, initially the overlying tissue can appear unaffected. Aggressive surgical debridement, antibiotic therapy and critical care supportive measures can be lifesaving if implemented rapidly.

ISCHEMIC COLITIS IN A YOUNG WOMAN

A healthy 36 year old woman presented after the acute onset of severe abdominal pain in the setting of constipation for two weeks and some rectal bleeding. She reported occasional small bowel movements and one day of nausea and vomiting. She denied fevers, chills or rigors, rash, ill contacts, vaginal bleeding and similar illness in the past. She had no prior history suggestive of Inflammatory Bowel Disease. Initial evaluation including CBC, Chem7, liver function tests, abdominal X-ray, pregnancy test, lipase, amylase, LDH and lactate were all normal. Trials of enemas and laxatives were given without significant relief. CT scan demonstrated marked mural thickening of the distal transverse, descending and sigmoid colon consistent with infectious or inflammatory colitis, without signs of obstruction or free air. Her symptoms improved with hydration, antibiotics, analgesics and antiemetics. Infectious and autoimmune work up including ESR, ANCA, ASCA, TSH, stool studies for culture, ova and parasites were done. All were negative, including specific testing for Enterohemorrhagic E.coli, Salmonella, Shigella, C-diff, and Campylobacter. A Colonoscopy was performed on hospital day two, which revealed an extensive segment of inflammation, edema and necrotic membranes extending from the descending colon into the transverse colon consistent with ischemia. Biopsies confirmed the diagnosis of acute ischemic colitis. The patient had never had symptoms of heart disease, a pregnancy loss, or a family history of bleeding or clotting disorders. She also denied any use of OCPs, supplements, or tobacco. Coagulation, Factor 5 Leiden, Anti-thrombin III, Protein C and S deficiency were all tested for and all
normal. The physicians were left with the obvious question of why a healthy 36 year old woman would develop ischemic colitis, an entity generally associated with the elderly or sick. After extensive questioning, the patient admitted to taking the weight loss drug Phentermine daily for the past two years. Because she felt well and was pleased with results, she continued to take the medication far beyond the prescribed duration of 12 weeks. After a dramatic improvement, the patient was discharged home with strict instructions to avoid any weight loss pills. She remained asymptomatic during the follow up period. Based on our exhaustive work-up and her ongoing misuse of this medication, we concluded that Phentermine was the cause of her illness. Our subsequent literature search has uncovered only one prior case report of ischemic colitis attributed to this medication. Phentermine has also been associated with behavioral changes, pulmonary hypertension, and valvular heart diseases. With potential increased usage of anti-obesity drugs, better awareness and surveillance is needed among clinicians to prevent such outcomes. It seems likely that the adverse event we witnessed in our patient, despite its rarity in the literature, is more common than currently realized.

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**STRONGYLOIDIASIS AND DIFFUSE ALVEOLAR HEMORRHAGE IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS**

The presence of Strongyloides stercoralis infection in patients with Systemic Lupus Erythematosis (SLE) has been described previously. Strongyloides stercoralis hyperinfection syndrome (SHS) usually develops in patients under immunosuppressive therapy, may affect a variety of organs but the presentation with diffuse alveolar hemorrhage (DAH) is rare with only a few cases described in the literature. We present the case of a 36 year-old Hispanic female with a past medical history relevant for SLE, recently diagnosis of lupus nephritis and hypertension, currently under treatment with steroids and cyclophosphamide for worsening of lupus nephritis who developed sudden and progressive abdominal pain and respiratory distress, with the presence of bilateral crackles and severe hypoxemia. The patient underwent endotracheal intubation and mechanical ventilation and computed tomography showed the presence of bilateral pulmonary infiltrates suggestive of DAH. Bronchoalveolar lavage was done and showed the presence of filariform larvae, morphologically consistent with strongyloides stercoralis. Treatment with ivermectine was started and patient responded to treatment with improvement of clinical status. In conclusion, the development of SHS in patients with lupus, especially when receiving immunosuppressive therapy, is a severe and potentially fatal complication. Early detection and treatment may decrease mortality.

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**CHRONIC RASH OF TUBERCULOID HANSEN’S DISEASE**

Introduction: Leprosy presenting as a chronic benign skin rash without any symptoms can often be missed on initial examination and diagnostic workup, including biopsy. Biopsy specimen requires appropriate notification to the pathologist. Early diagnosis and full course of treatment are critical for preventing lifelong neuropathy and disability due to leprosy. Case presentation: A 52 year old male presented at outpatient clinic with complaints of left leg rash which he noticed two years ago. The patient became concerned recently because of increasing size of the rash. He visited three different doctors where all lab work where skin biopsies were inconclusive. The rash was not associated with any pain, tenderness, itching or prominent vessels. The patient does not recall any insect bites. Review of systems was negative. He denied past medical and surgical history and was not taking medications. Family history was non-contributory. He was born in the Philippines and lives in the US for 20 years. He denied smoking, alcohol or drug use. He denied allergies or sick contacts. He is married and lives with his family. He has traveled to Europe, Middle East and Asia many times. On physical exam, HR 78; BP 130/70; RR 14; T 98.8 F. He was not in apparent distress. General physical was benign. Heart sounds were normal with clear lungs. Abdomen was benign. Neurologically there was decreased sensation over the lesion, but no motor deficit. Skin exam showed 18x12 inch discoloration on the left leg that was raised sarcomatous lesion. Lab work was within normal limits and was negative for vasculitis and TB. Full thickness skin biopsy from the margins was sent to National Center for Hansen’s disease. The biopsy report showed findings of non caseating granulomatous infiltration in 50% of the dermis with involvement of nerves.
and was for negative acid-fast bacilli. This finding suggested tuberculoid Hansen’s disease. He was started on rifampicin and dapsone with improvement. Discussion: Hansen’s disease is caused by Mycobacterium leprae, spread by the respiratory route. It involves the skin & peripheral nerves. In the United States, 250 cases were reported in 2010. Approximately 75% of cases are immigrants from high prevalence countries including India, Brazil, Indonesia, Bangladesh and Nigeria. Tuberculoid and Lepromatous leprosy are extreme ends of the leprosy spectrum. Physical findings are hypopigmented reddish skin patches, diminished/loss of sensation in involved areas, paresthesias, enlarged tender peripheral nerves, neuropathy and ophthalmic injury. Skin biopsy obtained from the leading edge of the skin lesion confirms the presence of acid fast bacilli in a cutaneous nerve. Treatment of leprosy includes Dapsone and Rifampicin with addition of clofazimine for Lepromatous disease.

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CHRONIC ACTIVE EPSTEIN-BARR VIRUS INFECTION MIMICKING DIABETIC KETOACIDOSIS-A FATAL CASE

Introduction: Mortality in EBV infection has been well described in males with Duncan syndrome. We report a very rare fatal complication of EBV in a previously healthy female patient known as Chronic active Epstein-Barr virus infection (CAEBV) with a clinical picture mimicking diabetic ketoacidosis. Case Presentation: A twenty year old female was admitted to hospital because of lightheadedness and shortness of breath. Two weeks prior to admission she started having easy fatigability followed by polyuria and polydipsia for three days. On the day of admission the patient started having light headedness and getting progressively short of breath. Patient denied fever, sore throat, sick contacts, recent travel or loss of appetite and any significant past medical or surgical history. She was sexually active with single male partner for last six months and used safe sex practice. On admission patient had pulse 138/min, BP 195/145, RR 40/min, oxygen saturation 90% on room air and temperature 36.4C. Patient was tachypneic, lethargic, in respiratory distress, had pale conjunctiva, clear lung fields, regular tachycardia without third or fourth heart sounds and no murmurs. Her lab data showed hemoglobin 8.7g/dL, WBC 37,800/mm3 (26% neutrophils, 12%bands, 50%lymphocytes, 2% atypical lymphocytes), glucose 347mg/dL, bicarbonate 7mEq/L, anion gap 25, BUN 29mg/dL and creatinine 2.9mg/dL. Her urine was negative for pregnancy, ketones and drug screen. ABG showed pH 7.28, Pco2 17.4, Po2 60.8, oxygen saturation 86% on 35%FiO2. Her EKG was normal and portable chest x-ray showed no infiltrates. Patient was treated with IV insulin drip for suspected diabetic ketoacidosis with cautious fluid management because of possible cardiomegaly on portable chest x-ray and positive troponins (0.66ng/ml). However subsequent HbA1c value was 5.6. Within 12 hours of admission patient, however deteriorated rapidly and died of cardiorespiratory arrest secondary to multiorgan failure. Autopsy showed chronic inflammation and lymphocytic infiltrate with atypical lymphocytes in all major organs including heart, lung, liver, kidney, brain, pancreas and upper airways and positive immunohistochemical staining for EBV. Review of imaging record 18 months ago showed thickening of aryepiglottic fold and laryngeal mucosa reflecting chronic disease process. Discussion and Conclusion: This seemingly immunocompetent young female had chronic progressive course of disease due to EBV which involved all major organs - a rare but fatal disease known as chronic active EBV infection with no known treatment currently available. The disease has been defined by the presence of three features. (1) severe progressive illness that began as a primary EBV infection, or is associated with abnormal EBV-specific antibody titers (2)Histology shows evidence of major organ involvement such as lymphadenitis, meningencephalitis, or persistent hepatitis. (3) Elevated EBV DNA, RNA, or proteins are demonstrable by in situ hybridization or immunohistochemical staining of affected tissues.
CASE REPORT: TMP-SMX ASSOCIATED MULTI ORGAN FAILURE IN A PATIENT WITH URINARY TRACT INFECTION

Introduction: Trimethoprim-sulfamethoxazole (TMP-SMX) is one of the first line drugs for treatment of uncomplicated lower urinary tract infections (UTI) in women. Side effects may include nausea, vomiting, skin rash and urticaria. Life threatening reactions include Stevens-Johnson syndrome, hepatic necrosis, and aseptic meningitis. We describe a previously healthy woman who, after being treated with TMP-SMX for an uncomplicated UTI, was found to have multi organ failure attribute to TMP-SMX. Case Presentation: A 59 y/o female with past medical history of hypertension and hyperlipidemia presented after having persistent fevers despite taking TMP-SMX for 5 days for UTI. On examination she was noted to have conjunctival suffusion, mucosal ulcers on her tongue, and a morbilliform non-petechial rash on her arms and trunk. Urinalysis was suggestive of UTI. After receiving piperacillin tazobactam and intravenous steroids, she defevered. However, on day 2 she developed shortness of breath, anuria, and encephalopathy and was subsequently intubated. Her creatinine increased to 4.04 mg/dl on day 2 from 1.9 mg/dl on admission, with elevated liver enzymes and severe metabolic acidosis. Serology for Hepatitis A, B, C, cytomegalovirus, human immunodeficiency virus, herpes simplex virus, and blood cultures were negative. MRI of brain and cerebrospinal fluid studies were consistent with aseptic meningitis. A kidney biopsy showed acute tubular necrosis with focal sites of mild tubulo-interstitial nephritis and normal glomeruli. Liver enzymes normalized but she had to remain on hemodialysis for six weeks, after which her creatinine returned to baseline. After a complex hospital course, she was eventually discharged to an acute rehab facility in stable condition. Discussion: We report this case because of the rare triad of aseptic meningitis, liver necrosis and acute interstitial nephritis. There are case reports of direct acute kidney injury from TMP/SMX but few of such multi-organ failure. The onset of symptoms may occur from a few days, as in our patient, to a few months following treatment. As in most cases of drug-induced organ failure, the presumably causal link between the offending drug and organ damage in our patient was based upon timing, exclusion of other causes and biopsy results. Postulated theories for such a reaction to TMP-SMX include: type III/IV hypersensitivity reaction, direct toxicity, immune complex deposition, and induction of autoantibodies. Elevated levels of interleukin-6 have been found in patients with systemic adverse reactions to TMP-SMX. Regardless of its precise pathophysiology, withholding the medication is the uniformly effective treatment. In summary, clinicians should be vigilant of potentially fatal organ toxicities from this commonly used drug. References: 1) Trimethoprim sulfamethoxazole induced aseptic meningitis. Repplinger MD, Falk PM. American Journal of Emergency Medicine, 2011 Feb;29(2):242.e3-5. 2) IL-6 may be key mediator in trimethoprim-induced systemic adverse reaction and aseptic meningitis: Antonen JA, Saha HH, Hurme M. Clinical Nephrology 2001 Jun;55:489-90.

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INTERNAL JUGULAR AND SUBCLAVIAN VEIN THROMBOSIS AS A COMPLICATION OF IN VITRO FERTILIZATION AND OVARIAN HYPERSTIMULATION SYNDROME (OHSS)

Background: Since being introduced in the late 1970s, assisted reproductive technology (ART) has been increasingly used to help couples with infertility. Thromboembolism is a reported complication of ART especially in the setting of ovarian hyperstimulation syndrome (OHSS). We present a patient who developed internal jugular vein (IJV) and subclavian vein thrombosis after being treated with ART and in the absence of a hypercoagulable state. Case Report: A 39 year old white female underwent successful in vitro fertilization (IVF) for tubal infertility. Four weeks after IVF, the patient started to develop manifestations of moderate to severe OHSS including generalized weakness, hemoconcentration, and abdominal distention with ascites. The patient was fluid resuscitated, had a therapeutic paracentesis of 2.5 liters, and was discharged on no anticoagulation. Four weeks later, the patient complained of right sided neck pain and swelling associated with shortness of breath and fatigue. She was hemodynamically stable with a room air oxygen saturation of 96-98 percent. Doppler evaluation revealed extensive thrombosis in the right IJV and subclavian vein. There was no history of recent trauma or venous catheterization and patient reported a negative hypercoagulability workup done 3 years prior following an abortion. She was treated with intravenous heparin infusion and monitored in...
the hospital. During her hospital stay, the patient had episodes of paroxysmal atrial tachycardia associated with shortness of breath. Arterial blood gas showed respiratory alkalosis with a pH of 7.51. Lower extremity dopplers were negative for venous thrombosis and a 2-dementional echocardiogram showed no right ventricular strain. The diagnosis of pulmonary embolism was not pursued further due to the potential harmful effects of radiologic testing on the fetus. The patient was discharged home on therapeutic enoxaparin with outpatient follow-up. Discussion: OHSS is a major complication of ART which may lead to acute fluid shifts into the extravascular space resulting in hemoconcentration, hypovolemia, and ascites, as well as liver and kidney dysfunction. Although uncommon, thromboembolism has been reported in women undergoing ART mostly in the setting of OHSS. Hemoconcentration and changes in the coagulation pathway are postulated explanations for hypercoagulability in these cases. Unlike DVT occurring during natural pregnancies, thromboembolism in patients undergoing ART tends to occur early in the first trimester and favors the vessels in the upper extremities, head and neck. Screening for hypercoagulability in all patients undergoing ART is not yet recommended because it is not considered to be cost effective. Unusual presentation and lack of familiarity with this risk factor may lead to delayed or missed diagnosis which may, in turn, lead to morbidity and mortality in these otherwise healthy patients. Health care providers should keep a high index of suspicion and a low threshold for early evaluation and treatment in these patients.

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LACTOBACILLUS: THE PATHOGENIC PROBIOTIC

Introduction: Lactobacillus is recognized for its beneficial probiotic effects in the treatment of diarrheal and urinary-tract infections. Lactobacillus is an uncommon pathogen, but it has been implicated in invasive infections in high-risk populations. Risk factors include recent dental manipulation, impaired immunity, and structural heart disease. Treating an invasive Lactobacillus infection is challenging as there is a paucity of data supporting an optimal antimicrobial regimen. We report a case of subacute prosthetic valve endocarditis due to Lactobacillus complicated by splenic infarcts. Case: A 54 year-old female with a history of bioprosthetic aortic-valve replacement presented with a 3-week history of fatigue, malaise, and progressive left upper quadrant abdominal pain radiating to the left shoulder that was characterized as sharp, constant, with an intensity of 10/10, without aggravating or alleviating factors. The patient reported intermittent chills and night sweats, but denied the presence of fevers, weight loss, or associated gastrointestinal symptoms. There was no recent instrumentation including dental work and colonoscopy. On presentation, she was afebrile and physical examination was positive for a 2/6 systolic ejection murmur and left upper-quadrant abdominal tenderness without appreciable splenomegaly or peripheral stigmata of embolic phenomena. Inflammatory markers were elevated, but there was no leukocytosis. Computed-tomography revealed two wedge-shaped splenic infarcts. Three sets of blood cultures were drawn from separate peripheral sites, each growing Lactobacillus by the second hospital day. The patient underwent transesophageal-echocardiography(TEE) that showed no vegetations. Presumed Lactobacillus subacute prosthetic valve endocarditis was treated with a 6-week synergistic course of Ampicillin and Gentamicin. Three weeks after discharge repeat blood cultures remained sterile and the patient reported complete resolution of her symptoms. Discussion: Lactobacillus is a Gram-positive, immotile, non-spore forming, anaerobic rod that is part of the normal flora of the oral cavity, GI tract, and female genitourinary system. There have been 28 case reports of Lactobacillus endocarditis since 1992, the time of institution of Duke’s Criteria for endocarditis. Our patient met the definition of “possible infective endocarditis” according to Modified Duke’s Criteria. She did not have vegetations on TEE, but it was postulated that vegetations had emolized to the spleen. Several features of this case are typical of Lactobacillus endocarditis including the subacute onset, development in individuals with pre-existing structural cardiac disease (83% of patients in one case series), and predilection for embolic phenomena (42% of cases in one series). No standardized empiric treatment for Lactobacillus bacteraemia/endocarditis exists. Review of the literature reveals that Lactobacillus is almost always sensitive to Penicillin, with Gentamicin frequently added for its synergistic effect. The duration of therapy is typically 6 weeks and relapses occur frequently (39%). Conclusion: Lactobacillus endocarditis should always be suspected in high-risk individuals presenting with embolic phenomena and treated aggressively to ensure microbiologic cure.
SINUS NODE ARREST IN THE SETTING OF CARDIAC SARCOIDOSIS

Introduction: Conduction abnormalities are common in cardiac sarcoidosis. However, sinus node arrest is exceedingly rare. In this case, a 64 year-old female with history of Sarcoidosis presents with syncope and was found to be in sinus node arrest. Cardiac MRI was done and findings were suggestive of cardiac sarcoidosis. To our knowledge, only three cases of cardiac sarcoidosis claiming sinus node involvement have ever been published. This case highlights the clinical expression, methods of diagnosis, and management of this potentially fatal condition.  

Case: A 64 year-old female with a history of pulmonary sarcoidosis presented with a syncopal episode without a cardiac prodrome. Bradycardia was noted in route to the hospital and two additional episodes of syncope occurred after admission. During one of these episodes, the monitor captured 16 seconds of sinus node arrest without an escape rhythm. A temporary transvenous pacemaker was inserted on the day of admission and an echocardiogram revealed normal left ventricular systolic function. Cardiac MRI was performed which revealed delayed mid-wall enhancement in the basal and antero-septal walls consistent with an infiltrative cardiomyopathy due to sarcoidosis. A dual chamber implantable cardioverter-defibrillator was inserted on the second hospital day. The patient remained asymptomatic and began treatment with glucocorticoids as an outpatient.

Discussion: To our knowledge, this is only the fourth reported case of sinus node involvement due to cardiac sarcoidosis (CS). Sarcoidosis is a multisystem, granulomatous disease that may involve the heart in at least 25% in patients with sarcoidosis in the United States and cardiac sarcoid can account for as many as 13% to 25% of death from the disease. The left ventricular free wall and ventricular septum are the most common regions affected by granulomatous infiltration. The clinical manifestations of cardiac sarcoid are related to the location and extent of granulomatous involvement and range from asymptomatic ECG abnormalities to sudden death. Sarcoidosis can affect virtually any location of the conduction system leading to left or right bundle branch block, atrioventricular block of any degree, and rarely sinus node arrest as was the case in our patient. Complete heart block is the most common presenting conduction abnormality (25-30%), most frequently presenting as syncope although sudden death may be the initial manifestation of cardiac sarcoi. Cardiac MRI has emerged as a noninvasive means to establish a diagnosis and treatment includes glucocorticoids and implantable cardiac devices when appropriate.

Conclusion: Sarcoidosis has a predilection for cardiac tissue producing a spectrum of disease that ranges from asymptomatic infiltration to life-threatening involvement. Therefore, it is imperative that internists be familiar with the prevalence, clinical manifestations, diagnosis, treatment, and prognosis of this clinical entity.

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PPI INDUCED AIN

Proton Pump Inhibitors (PPIs) are widely used in the US for a variety of gastrointestinal disorders. Often they are started in the hospital setting and continued upon discharge. While generally well tolerated, PPIs can lead to serious complications such as acute interstitial nephritis (AIN). In this report we describe a case of PPI related AIN. A 75 year old female with a history of Type 2 DM, HTN, anemia and atrial fibrillation was admitted to the intensive care unit after being found unconscious. She was intubated for airway protection and was noted to have a creatinine of 0.7 and a sodium of 110. While the patient was on ventilatory support, pantoprazole was started for ulcer prophylaxis. She was initially treated with hypertonic saline and extubated on hospital day 2. After the diagnosis of SIADH was made, the patient was free-water restricted and given salt tablets. Once her mental status had normalized and sodium stabilized, the patient was sent to a rehabilitation center with a creatinine of 1.1. The acute kidney injury was thought to be secondary to acute tubular necrosis (ATN) from episodes of hypotension and the contrast received for CT scans. One week after discharge, the patient was readmitted with a temperature of 101° Fahrenheit and a creatinine of 1.8. There was no evidence of infection. Her fever resolved without antibiotics and she was discharged with a creatinine of 3.5. A work up of AKI was negative for glomerulonephritis but the urine sediment showed muddy-brown casts suggestive of ATN. The patient refused a kidney biopsy at this time. During the hospital stay and on discharge the patient was maintained on pantoprazole for ulcer prophylaxis. Three weeks later, she returned to the hospital for lower extremity edema and worsening renal function with a creatinine of 4.6. Her CBC now revealed peripheral eosinophilia of 21.47%. Urinalysis showed 2+ protein, 1+ blood, >182 WBC, 32 RBC, and a few eosinophils.
Spot urine protein to creatinine ratio was 2.8. With the constellation of proteinuria, eosinophiluria, pyuria, and peripheral eosinophilia, the diagnosis of AIN was made. Reviewing her medication list, the most likely culprit was pantoprazole. Therefore, pantoprazole was discontinued and she was started on prednisone. Her creatinine subsequently stabilized at 4.3 and she was discharged on a prednisone taper. At the office follow up two weeks later, her creatinine was significantly lower at 2.2 and it continued to decrease in subsequent visits. PPI used for ulcer prophylaxis is a common practice in all hospitals. A rare but serious complication of PPI use is AIN. This case demonstrates the importance of recognizing the appropriate use of PPIs (indication and duration) and awareness about one of its important complications, AIN.

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BEAUTY IS ONLY SKIN DEEP, BUT SILICONE GOES MUCH DEEPER: A CASE OF SILICONE-INDUCED GRANULOMATOUS DISEASE

Case Presentation: This is a 47 year old Hispanic woman with a history of idiopathic hypercalcemia since 2005, chronic kidney disease stage IV, who presented with a one week history of dysuria and left flank pain. Review of symptoms was otherwise unremarkable. In the emergency department, she was started on IV fluids and empirical antibiotics. Bicarbonate drip was also started for serum bicarbonate of 6 meq/L with a pH of 7.071, pCO2 21, and pO2 127. A renal ultrasound showed left pyonephrosis and an obstructed left ureteral stent, for which an emergent left percutaneous nephrostomy tube was placed. Patient has a history of hypercalcemia and recurrent nephrocalcinosis since 2005 for which she had laser treatment lithotripsy and left ureteral stent placed in 2008. She has CKD Stage 4, s/p HD for 2 weeks in September 2011. She had cord compression caused by ossification of the ligamentum flavum, s/p fusion laminectomy C2-T1 in September 2010. Biopsy of the posterior longitudinal ligament showed foreign body granulomatous reaction. She is being treated for hypercalcemia with bisphosphonates and Denosumab. Physical examination revealed tenderness in left upper, lower and suprapubic quadrant of the abdomen, and the left nephrostomy tube draining urine. Extremity exam showed firm, non-mobile, nontender subcutaneous masses over the posterior thighs and buttocks, with overlying hyperpigmented, dry skin. Labs were significant for leukocytosis with left shift, anemia, hyponatremia, hyperkalemia, anion gap metabolic acidosis, acute kidney injury, hypercalcemia, hyperphosphatemia, hypoalbuminemia, and significant pyuria. Previous laboratory evaluation for hypercalcemia showed a normal PTH, normal 1,25-dihydroxy vitamin D, but an elevated ACE level. Upon further questioning, patient stated she had silicone injections into her gluteal region and thighs for augmentation in Puerto Rico 15 years ago. Her hypercalcemia and subsequent episodes of nephrocalcinosis was attributed to silicone-induced granulomas. Discussion: Successful management of hypercalcemia usually depends upon determining its etiology. In most patients the cause is obvious from the clinical setting, and the results of serum assays of PTH, PTHrP, and vitamin D metabolites. One rare but serious cause of hypercalcemia is silicone-induced granulomatosis disease. Although injectable silicone is no longer generally available for use in humans, preparations containing silicone of varying grades and purities are injected into patients as a cheap and fast alternative to conventional plastic surgery. Initially it was thought silicone injections are inert and provoke little local and no systemic response; however, adverse reactions include chronic cellulitis, nodule formation, foreign body reaction (including granulomatous reaction), migration of silicone to different parts of the body causing disfigurement, and massive silicone embolism. Although there are many reported adverse reactions due to silicone soft tissue augmentation, there is scant literature on silicone-induced granulomas causing hypercalcemia and subsequent renal failure.
**ATLANTIC HEALTH - OVERLOOK (JEFFREY BRENSILVER, MD)**

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**EVOLUTIONARY IMMUNE RESPONSE TO CONSERVED DOMAINS PRESENT IN PARASITES AND AEROALLERGENS**

Background: The immune response based on immunoglobulin E (IgE) evolved as a defense against specific parasitic infections. In the absence of active helminthic infections, the immune system has redirected its IgE epitopes toward innocuous environmental antigens. Helminths and aeroallergens have a similar stereotypical IgE response to unique antigens that cannot be explained by chance alone. Objective: To evaluate potential homology between conserved protein domains embedded in parasitic organisms and aeroallergens. Methods: Search and retrieval systems for nucleotide and protein sequences (Entrez, BLAST, and National Center for Biotechnology Information) were searched to identify conserved domains between allergens and certain parasites. A Total Score was developed that correlated positively with homology between compared sequences. Over 2000 domains were examined. Results: We found matches with a high Total Score (> 100) that signified a strong positive correlation between sequences in allergens (n = 28) and parasites (n = 13). Multiple shared conserved domains were identified between parasites and allergens. Parasite-allergen combinations with the most significant homology (greatest Total Score) were: P. falciparum enolase and Hev b9 (Total Score, 612); S. mansoni albumin and Fel d2 (976); A. lumbricoides tropomyosin and Ani s3 (531); and Wuchereria bancrofti trypsin and Blo t3 (138). Conclusion: Homologous conserved domains exist in specific parasites and allergens, consistent with the theory that the human IgE eosinophil immune response to common allergens is a direct consequence of stimulation by parasitic organisms.

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**CONJUNCTIVAL SQUAMOUS CELL CARCINOMA HARBORING LEISHMANIA AMASTIGOTES IN A HUMAN IMMUNODEFICIENCY VI**

A 39-year-old HIV-positive Guatemalan man had decreased vision, epiphora, and pain in the right eye for 18 months. He had been continuously maintained on highly active antiretroviral therapy, azithromycin, and sulfamethoxazole/trimethoprim for 2 years. He denied fever, sweating, or flulike symptoms. Best-corrected visual acuity was 20/25 OD and 20/20 OS. The pupils were 5 mm on the right and 7 mm on the left, briskly reactive to light, and without relative afferent pupil defect. Extraocular movements were full without restriction. Intraocular pressures were 17 mm Hg in both eyes. The right upper eyelid was mildly ptotic and swollen. Funduscopic examination results were unremarkable. Ultrasound biomicroscopy and B-mode ultrasonography of the globe did not suggest extension into deeper structures or transscleral invasion. Computed tomography of the orbit revealed disease limited to preseptal soft tissue. Laboratory evaluation demonstrated a viral load of less than 48 copies/mL and a CD4 lymphocyte count of 79 cells/μL. Excisional biopsy of the mass showed moderately differentiated invasive SCC as well as intracellular microorganisms in histiocytes. Special stains for Histoplasma and Toxoplasma were negative. High-power oil immersion highlighted the Leishmania amastigotes, and CD68 staining confirmed their presence in macrophages. The Centers for Disease Control and Prevention confirmed the microorganisms as L donovani chagasi. Postoperative positron emission tomography showed increased uptake with uptake within the right lateral conjunctiva, liver, spleen, and axillary, mediastinal, mesenteric, pelvic, and cervical lymph nodes. Abdominal computed tomography revealed hepatosplenomegaly (Figure 2). A subsequent liver biopsy confirmed the diagnosis of visceral leishmaniasis. The patient was treated with intravenous liposomal amphotericin B (190 mg/d for 14 days and triweekly thereafter).
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**HYPERCALCEMIA WITH AN UNUSUAL MALIGNANT ETIOLOGY**

CASE REPORT: A 44-year-old female with a past medical history of hypertension and hypothyroidism presented with 3 days of nausea, vomiting, and fatigue. She was found to have a calcium of 15.4, white blood cells 18,400, with a borderline low PTH (13.2). Her PTHrP, 25-hydroxyl Vitamin D, chest x-ray and myeloma workup were within normal limits. She had a recent normal mammogram and no evidence of malignancy on CAT scan of abdomen/pelvis. Her peripheral blood smear had smudge cells and her bone marrow biopsy was performed. She was given intravenous normal saline, furosemide, and pamidronate and was discharged with a calcium of 13. She was readmitted one week later with similar symptoms. She was found to have a small lymph node in her left axilla, calcium of 19, worsening leukocytosis, and elevated uric acid. Her bone marrow biopsy was consistent with marrow involvement by peripheral T-Cell Lymphoma. Her peripheral blood flow cytometry revealed T-cell leukemia, favoring human T-lymphotropic virus, type I (HTLV-1) Leukemia/Lymphoma (adult T cell leukemia/lymphoma). MRI brain with contrast was negative and her HTLV-1 serology was pending. She improved again with intravenous fluids, furosemide, zoledronic acid and was discharged to get follow-up treatment at another hospital.  

DISCUSSION: Adult T cell leukemia/lymphoma (ATL) is an uncommon lymphoid neoplasm occurring in patients infected with HTLV-1. Infection with HTLV-1 is endemic in areas such as southern Japan, the Caribbean basin (eg, Jamaica and Trinidad), western Africa, etc. The risk of developing ATL following HTLV-1 infection has been estimated to be 4-5%, with a latency period of 10-30 years. ATL has several clinical variants: acute, lymphomatous, chronic, and smoldering; they
have different genomic alterations, clinic futures and prognosis. The common clinic symptoms and signs include moderate leukocytosis, bone marrow involvement, generalized lymphadenopathy, hepatosplenomegaly, hypercalcemia, and skin lesions. Also, patients may present with an elevated LDH. The diagnosis of ATL is based upon a combination of specific clinical features, the morphology and immunophenotype of the malignant cells along with confirmation of HTLV-I infection. The confirmation of HTLV-1 infection by serologic tests of anti-HTLV-1 antibodies is essential to the diagnosis of ATL. Patients with acute, lymphomatous, or chronic type ATL progress quickly without treatment. Combination chemotherapy is the main treatment option. Intrathecal chemotherapy is recommended for all patients given a 10-25% risk of central nervous system involvement. Serum calcium levels need to be frequently monitored in these patients and there is a significant risk of tumor lysis syndrome. Oral trimethoprim-sulfamethoxazole for prophylaxis of Pneumocystis jiroveci pneumonia is suggested because patients with ATL are also immunocompromised and are at risk for opportunistic infections.

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AN INTERESTING CASE OF OSMOTIC DEMYELINATION SYNDROME SECONDARY TO HIV

Osmotic demyelination syndrome (ODS), previously known as central pontine myelinosis, is a rare condition usually following rapid correction of chronic hyponatremia. We present a case of ODS secondary to AIDS. A 24 year old male diagnosed with AIDS and syphilis not on HAART was admitted with tachycardia, fever, aphasia and confusion. One month prior he was admitted for hypothermia and altered mental status with hyponatremia of 129 mEq/l which responded to intravenous fluids. He was discharged home on penicillin for treatment of syphilis. There was no history of alcohol abuse. On examination he appeared cachectic and ill. He was awake, oriented to place and time but not to person. He was confused, aphasic with impaired memory. He had a flat affect and appeared withdrawn. Pupils were equal, round, reactive to light. Extra ocular movements were intact. Motor strength was 5/5 in all four extremities. He had generalized tremors at rest worse with movement. There was decreased muscle bulk but normal tone. Sensation was normal. Romberg testing was deferred. Deep tendon reflexes were symmetrical 2+ throughout. Cerebellar exam revealed end target dysmetria and was confounded by severe tremors. Gait testing was deferred due to his condition. The remainder of his neurologic exam was negative. On admission sodium was 125 mEq/L. He was started on intravenous normal saline at 80ml/hr and after 24 hours sodium was 143 mEq/L. WBC was 2.3/mm3, hemoglobin 7.9g/dL, hematocrit 23.2%, platelets 62,000/mm3, alkaline phosphatase 531u/L, AST 220u/l, ALT 103u/L, albumin 2.8g/dL, glucose 198mg/dL, BUN 14mg/dL, creatinine 0.8mg/dL, absolute CD4 count 41/uL. Blood and urine cultures were negative. Lumbar puncture was deferred because of thrombocytopenia. Prior CT head was normal. CT of the abdomen was unremarkable. MRI of the brain revealed a focal area of restricted diffusion and increased signal within the central pons suggestive of central pontine myelinosis. On follow-up his aphasia resolved, generalized body tremors persisted. He had fluctuating confusion. He was advised HAART upon discharge. There are less than 10 reported cases of ODS secondary to HIV. Our patient had hyponatremia with underlying malnutrition. It is unlikely that the correction of the hyponatremia contributed to the ODS because his sodium level was only moderately low and the majority of cases have been reported in patients with initial sodium of 120 meq/L or less. The tremors and confusion were present prior to the MRI imaging and he had no new neurologic deficits during the hospitalization. His liver disease is most likely related to HIV. Alcoholism, malnutrition, liver disease and hypokalemia can increase the susceptibility to osmotic demyelination. We believe HIV can cause ODS, possibly as a primary effect of HIV on the brain, although the pathogenesis remains unknown.
AN INTERESTING CASE OF ISCHEMIC STROKE SECONDARY TO SEVERE IRON DEFICIENCY ANEMIA

The etiology of stroke in young persons, defined by most recent studies as individuals who are 45 years or younger, often has a broader etiology that stroke in those older than 45. Common causes of stroke in young persons include: spontaneous intracerebral hemorrhage, cardio embolic phenomenon, hypercoagulable states, cervicocephalic arterial dissection. Rarer causes include cerebral vasculitis Moya-Moya disease, premature atherosclerotic vascular disease, cocaine use and migraine. We report a rare case of ischemic stroke in a young woman secondary to severe iron deficiency anemia. A 34 year old female smoker with history of menorrhagia presented with left upper extremity numbness and weakness of the left hand with loss of grip, associated with a severe global headache which resolved after a few hours. There was no facial asymmetry, change in speech, weakness or numbness of other extremities, visual changes, and stool or urine incontinence. She complained of progressive lightheadedness and dizziness for 3 months. Three years ago she took oral contraceptive pills for only 3 months with improvement in the menorrhagia. She denied family history of stroke or neurologic disease. On examination conjunctiva were pale. She was awake, alert and oriented with normal speech and language. Cranial nerves II to XII were intact. She had decreased light touch over the left upper extremity with a left pronator drift. Motor strength was 5/5 in both upper and lower extremities in all muscle groups. The deep tendon reflexes were +2 throughout. She had normal muscle bulk and tone, no dysmetria and normal gait. Laboratory evaluation revealed hemoglobin of 4.1g/dl and iron studies were consistent with iron deficiency. She received 4 units of packed red blood cells, after which the patient's functional deficits improved. Computed tomography of the head revealed asymmetric diminished attenuation extending from the right corona radiate into the right upper parietal convexity. Brain MRI revealed an acute right parietal lobe infarct. MRA of the head was normal. Doppler ultrasound of the carotids was unremarkable. Transthoracic echocardiogram was negative. There are few reported cases of stroke secondary to iron deficiency anemia. Three physiological mechanisms resulting from iron deficiency anemia that could explain this include a hypercoagulable state, secondary thrombosis and low flow oxygen state to the brain with resultant hypoxia. Majority of the reported cases are in young children and are secondary to thrombosis. Our patient took oral contraceptives for 3 months, and she was a smoker. However there was no evidence of thromboembolism on neuroimaging evaluation. Therefore, it is likely that the iron deficiency anemia caused hypoxia induced tissue injury secondary to low oxygen flow to the brain in our patient. These findings suggest that iron deficiency anemia should be considered as a risk factor for ischemic stroke.

EXACERBATION OF A RARE NEUROMUSCULAR DISORDER SECONDARY TO THE USE OF MACROLIDE ANTIBIOTIC

Kearns-Sayre syndrome is a rare mitochondrial myopathy first described in 1958. It is characterized by ophthalmoparesis and pigmentary retinopathy before age twenty, frequently associated with cardiac conduction abnormalities, proximal myopathy, and symptoms of endocrine dysfunction. Our patient was diagnosed after developing ptosis at the age of 12, a typical presentation of the syndrome. A 23 year old male was hospitalized for new onset paresthesias and weakness in both lower extremities. His only other medical history included multiple eye surgeries and placement of a permanent pacemaker. Although cachectic in appearance, he had been highly functioning, independent of his activities of daily living, and held two jobs. Four days prior, he developed cough and congestion and was diagnosed by chest x-ray with lobar pneumonia. He was treated with azithromycin and a cough suppressant containing codeine. Examination demonstrated bilateral ophthalmoplegia and 4/5 muscle strength in both lower extremities. Sensory exam was intact. Patellar and Achilles reflexes were 1+. Azithromycin was discontinued and he was placed on ceftriaxone. The next day his vital capacity dropped from 1.95 liters to 1.43 liters and his negative inspiratory force changed from -22 to -10 cm of water pressure, prompting transfer to intensive care. There was concern for acute inflammatory demyelinating neuropathy, or Guillain-Barre Syndrome, as a cause for his weakness and
lower extremity paresthesias. However, the patient maintained deep tendon reflexes over the next twenty-four hours, his sensorimotor function improved over several days, and his vital capacity had improved to 2.5 liters with a negative inspiratory force of -20. Thus, his sensorimotor type of neuropathy was deemed an exacerbation of his neuromuscular syndrome. He was able to ambulate with assistance and was discharged ten days later to a rehab facility with a vital capacity of 3.5 liters and a negative inspiratory force of -52. Macrolides are commonly used in the treatment of community acquired pneumonia. There are previously documented reports of exacerbations of myasthenia gravis secondary to macrolide use, most significantly with the antibiotic telithromycin. Macrolides contribute to exacerbations by inhibiting the presynaptic release of acetylcholine at nicotinic acetylcholine receptors. This case demonstrates that macrolides may also contribute to exacerbations of other neuromuscular diseases, including Kearns-Sayre syndrome, and caution should be used when prescribing macrolides to these patients.

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ADRENERGIC OVERDRIVE: A “NOT-SO-SYMPATHETIC” MASS WITH SYMPATHETIC POWERS

Like pheochromocytomas, extra-adrenal pheochromocytomas (EAPs) produce a classic triad of headaches, palpitations, and diaphoresis in the setting of existing hypertension. EAPs typically occur subdiaphragmatically but may arise in any part of the paraganglionic system. The most common site of occurrence is the superior para-aortic region between the diaphragm and the inferior renal pole. Because of their malignant potential in up to 40% of cases, they should be monitored. A 60-year-old female with past medical history of obesity, hypertension, diabetes, and hepatitis C on interferon therapy developed abdominal pain. Abdominal ultrasound revealed a hypoechoic mass on the pancreas. Computed tomography showed a lesion on the anterior pancreatic head. This was confirmed by magnetic resonance imaging as a 5.0 x 3.5 x 3.1 cm heterogeneous hemorrhagic mass displacing the inferior vena cava and compressing the left renal vein. An ultrasound-guided biopsy was inconclusive. She underwent exploratory laparotomy with retroperitoneal lymph node dissection, repair of vena cava, and cholecystectomy. Initial attempts at removing the mass were accompanied by severe hypertensive crises with systolic blood pressures exceeding 200 mmHg. After intraoperative stabilization, the mass was removed without consequence. Histology confirmed EAP with negative margins. Genetic testing for hereditary tumors was unremarkable. Postsurgical catecholamine testing was negative. Prior to surgery she denied headaches or palpitations but admitted being forty pounds lighter. She attributed her periodic diaphoresis to her interferon treatments and denied any other symptoms. She acknowledged long standing hypertension for 10-12 years under reasonable control with carvedilol 25mg twice a day and losartan 50mg daily. Although the history and physical exam findings may be helpful, there is no classic presentation for pheochromocytomas or EAPs. Patients can have classic hyperadrenergic attacks that overstimulate the sympathetic nervous system, consisting of headaches, palpitations, and diaphoresis, or can have non-specific symptoms but rarely are asymptomatic. In our patient’s case, it is likely that her catecholamine-producing tumor contributed to her thinner body habitus by speeding up her metabolism. Once the diagnosis is made, localization and surgical resection is necessary. While CT scanning is the imaging modality of choice, the clinical diagnosis is typically confirmed by 24 hour measurement of urinary fractionated catecholamines and metanephrines, which are intra-tumoral metabolites of catecholamine-producing tumors. This is the most reliable detection method for identifying catecholamine-secreting tumors, with a sensitivity and specificity of nearly 98 percent. Because of poor overall accuracy in testing for pheochromocytoma, measurement of plasma catecholamines no longer has a role. Lifelong follow-up with annual urinary catecholamine and metanephrine measurement is crucial, as EAPs are more likely to recur and metastasize than their adrenal counterparts.
UNMASKING PRE-EXISTANT CENTRAL DIABETES INSIPIDUS IN A PATIENT WITH NEUROSARCOIDOSIS AFTER GASTRIC BYPASS SURGERY: A RARE PRESENTATION

Central Diabetes Insipidus (CDI) is the result of decrease or absence of anti-diuretic hormone (ADH) due to diseases or conditions affecting the hypothalamic-neurohypophyseal system. It is characterized by excessive urination and thirst. Known causes include germinoma/craniopharyngioma, Langerhans cell Histiocytosis, local inflammatory, autoimmune or vascular diseases, trauma resulting from surgery or accident, sarcoidosis, metastases and cerebral or cranial malformations. Abrupt CDI occurs usually after a cranial event causing hypothalamic or pituitary damage such as trauma or surgery. We report a rare presentation of CDI that became apparent after gastric bypass surgery in a patient with neurosarcoidosis. A 39 year old female with past history of diabetes mellitus type 2 and neurosarcoi dosis treated with prednisone and methotrexate underwent Roux-en-Y gastric bypass surgery for morbid obesity. Immediately after surgery, she developed severe polyuria with average measured urine output of more than 800ml/hour. Intravenous fluid (IVF) was started. In retrospect she had severe thirst for the past year, drinking more than 10 liters of water per day. Her baseline sodium was persistently between 140-145 mmol/L in the past two years. Post-operative serum sodium was 159mmol/L, serum osmolality 327 mOsm/Kg, and urine osmolality 50 mOsm/Kg. Urine output was between 800-1000ml/hr of maximally diluted urine with specific gravity < 1.005. Dextrose 5% with free water fluid was started at a rate matching her hourly urine output plus maintenance. Vasopressin was started at 10 units every 12 hours. Neuro-imaging revealed empty sella turcica. Appropriate correction of her sodium was achieved by close monitoring of her urine output, blood pressure, serial serum sodium and osmolality, and titrating vasopressin accordingly. CDI is a rare complication of sarcoi diosis infiltration of the hypothalamic-pituitary region. She had undiagnosed symptoms for a year and was able to compensate for her polyuria by excessive water drinking. This case illustrates a very rare presentation of pre-existing undiagnosed (CDI) which was revealed after gastric bypass surgery since she was unable to keep up with her oral drinking. Despite the rarity of this case we emphasize the importance of pre-surgical screening with full history and physical examination, and close monitoring of urine output and electrolytes post-surgery.

SADDLE PULMONARY EMBOLISM AFTER PROLONGED IMMOBILIZATION DUE TO DOXYLAMINE OVERDOSE

A 69 year old male with history of hypercholesterolemia, depression and three previous suicide attempts was found in a motel room after ingestion of 96 pills of doxylamine. A suicide note was also found. On admission, he had altered mental status and was unable to respond or follow commands. Blood pressure 163 / 114, pulse 133 bpm; respirations 20/min, temperature 97.5 F and pulse oximetry 97% on room air. One episode of possible coffee grounds emesis was noted. Urine toxicology, urinalysis, complete blood count, metabolic profile, cardiac enzymes, liver function tests, coagulation profile and salicylate levels were normal. Blood alcohol level was 20 mg/dl. Electrocardiogram revealed sinus tachycardia with prolonged QRS at 108 milliseconds and Q waves in lead III, unchanged from a prior EKG. Well’s criteria score was 1.5 (estimated 1-3% risk of pulmonary embolism) for the tachycardia and Padua score was 0. Chest radiograph and computed tomography of the head without contrast were unremarkable. He was placed in sequential compression boots for deep venous thrombosis prophylaxis because gastrointestinal bleed could not be ruled out. He required two point restraints because of combativeness. No further vomiting was noted. The initial emesis was felt to be from the toxic ingestion with no evidence of bleeding. 72 hours later, he developed profound hypoxia with oxygen saturation of 70% on 4 liters of oxygen via nasal cannula. He was placed on a non-rebreather mask with 10 L of oxygen. Emergent CT angiogram of the chest showed a saddle pulmonary embolus extending across the pulmonary artery bifurcation with extensive thrombus in bilateral upper and lower lobes with right ventricular strain (McConnell sign) visible on echocardiogram. Lower extremity duplex ultrasound was negative. Cardiothoracic surgery was called for possible therapeutic thrombolysis or thrombectomy which the patient declined. Psychiatry found he was not competent to make medical decisions since he was still suicidal. The patient met criteria for involuntary commitment.
after stabilization of his medical problems. Tachycardia and the lack of pharmacologic anticoagulation prophylaxis may have been the only risk factors for the development of saddle pulmonary embolism in this patient. However, the role of pneumatic compression devices is not clear in DVT/PE prevention because of the variation in compression settings and attributes affecting both efficacy and compliance. Further studies may be warranted to identify which compression device settings are most effective for prevention and optimum compliance especially in the setting of suspected bleeding.

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RECURRENT VENOUS THROMBOSIS IN A POST 9-11 FIRST RESPONDER

First responders from 9/11 continue to suffer physical and emotional consequences of this tragedy. We present a patient with a hypercoagulable mutation and multiple venous thromboses diagnosed after 9/11. A 56-year-old ex-firefighter with past history of recurrent deep venous thrombosis and coronary artery bypass grafting on warfarin, clopidogrel and aspirin presented with a two-day history of retrosternal pain radiating to the left shoulder and non-tender swelling at the base of his neck, exacerbated by coughing. Duplex ultrasound of the left upper extremity showed a new non-occlusive thrombus in the left basilic vein. INR was 3.9. His history is complicated by asbestos and smoke exposure during the 9-11-2001 World Trade Center collapse. Throughout the years he had numerous venous thrombosis and pulmonary emboli requiring vena cava filter and stents placed in the distal inferior vena cava to both common iliac veins. A heterozygous G20210A mutation was identified. He also has diabetes mellitus, thyroid carcinoma s/p radioactive iodine, Crohn disease resulting in small bowel resection, and myocardial infarction status post CABG x 3 vessels. He was switched to warfarin to maintain INR of 3.5-4.5 and continued on clopidogrel and aspirin therapy for anticoagulation at discharge. There was no acute coronary syndrome during this admission. Prothrombin G20210A is a common inherited thrombophilia disorder. Carriers are at increased risk for deep vein thrombosis. Our patient never had any thrombotic events prior to 9/11. Recently the federal government has extended coverage to more than 50 different types of cancer under the James Zadroga 9/11 Health and Compensation Act after seeing an increasing trend in certain cancers for 9/11 first responders. We propose that inhalation injury predisposes to expression of these mutations, thus resulting in thrombosis and resistance to anticoagulation. Further research is needed to determine the best therapy for refractory and recurrent venous thrombosis despite current management and to establish a relationship between inhalation injury post 9-11 and thrombosis.

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A COMMON ORGANISM AT AN UNCOMMON SITE: MULTIPLE HEPATIC ABScesses CAUSED BY Streptococcus intermedius

Streptococcus intermedius is a part of Streptococcus anginosus group, subgroup of viridans streptococci. It is a Gram-positive, catalase-negative cocci. This facultative anaerobe is found in normal oral flora (especially dental plaque), nasopharynx and gastrointestinal tract with the ability to cause abscess and systemic infection. A 21 year old male with history of treated dental infection one year prior presented with a three week history of fever and chills and one week of nausea, vomiting, diarrhea and abdominal pain. On physical examination he was febrile. The abdomen was soft and non tender without organomegaly. There were no cardiac murmurs. Laboratory studies showed alkaline phosphatase 318 U/L, alanine aminotransferase 50 U/L, WBC 14.7x1000/μL, ESR 115mm/hr, CRP 155 mg/dL. Computed tomography (CT) of the abdomen demonstrated two liver abscesses, one of which was 6.3 x 5.6 x 5 cm size with thickening of a long segment of ileum spreading towards the terminal ileum. Empiric treatment with ciprofloxacin and metronidazole was started. On the next day he underwent successful radiology-guided drainage of the abscess with removal of 150ml purulent fluid followed by drainage catheter placement. Fever persisted for 3 additional days. On day 5 the anaerobic culture from abscess fluid was positive for Streptococcus intermedius. A transthoracic echocardiogram was negative for endocarditis. After 5 day course of intravenous ciprofloxacin, metronidazole and 3 days of intravenous vancomycin he was discharged on intravenous ertapenem and the drainage catheter was removed several weeks later. Subsequent CT of the abdomen showed resolution of both abscess cavities and thickening of small bowel. Predisposing factors to Streptococcus intermedius infection
include biliary tract pathology, immunodeficiency states, diabetes, alcohol consumption, and mucosal disturbances. Our patient did not have any of these factors. Though dental intervention might have been the source, its successful treatment one year prior decreases the likelihood of any association. A review of the literature from 2005-2011 showed Streptococcus intermedius as the most common species to cause hepatic abscess with or without defined cause. The propensity of this commensal pathogen to cause abscesses should prompt further investigation to rule out other infections such as brain abscess and rarely endocarditis. Most of the Streptococcus intermedius species are sensitive to penicillin, cephalosporins and vancomycin with few species identified resistant against erythromycin and clindamycin. Remission is achieved with drainage of the abscess and appropriate antibiotic therapy.

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A BENIGN SOFT TISSUE MASS FOUND TO BE A MALIGNANT FIBROUS HISTIOCYTOMA

Soft tissue sarcomas compose less than 1% of total cancer diagnoses in the United States. Patients often complain of a mass or lump that grows over a short time span ranging from weeks to months. We describe a case that illustrates the importance of evaluating all “benign” soft tissue masses. A 67 year old female was evaluated by her primary care physician for a painless soft tissue mass in her left thigh. The mass had been slowly evolving for several weeks. There was no trauma to the area. Physical examination revealed two superficial masses on the proximal left thigh with no regional lymphadenopathy. The remainder of the exam was unremarkable. An ultrasound of the left posterior thigh demonstrated two hypo echoic soft tissue masses in the posterior mid left thigh. Magnetic resonance imaging of the left femur demonstrated the 2 lesions to be located posteriorly and laterally to the mid bicep femoris muscle with the proximal lesion measured at 1.9 x 1.8 x 1.6cm and the distal lesion 2.7 x 2.6 x 2.8cm. The lesions were thought to originate from the fascia without involvement of the bone or adjacent structures. Both tumors were resected and the pathology revealed spindle cell neoplastic growth of a pleomorphic sarcoma consistent with malignant fibrous histiocytoma (MFH) stage II A (T1A N0 M0, grade 2). The lesion extended beyond the excision margins. Computed tomography of the chest was unremarkable. She was referred for wide margin type excision and radiation therapy with possible adjuvant chemotherapy as per NCCN guidelines. Clinicians must exercise caution when evaluating a soft tissue mass that may appear benign. MFH represents the most common form of sarcomas accounting for 28% of soft tissue tumors and 2% of bone tumors. MFH typically presents in patients from 50 to 70 years of age with a slight male predominance. The mass can arise in any part of the body but is frequently found in the lower extremity, especially the thigh. Other common locations include the upper extremity and retroperitoneum. Since a benign soft tissue mass is more likely to be encountered in the outpatient setting and can be dismissed as a harmless entity, this vignette emphasizes the importance of an initial thorough evaluation and further investigation by a specialist.

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CORONARY CAMERAL FISTULAE

Coronary artery fistulae include coronary arteries opening directly into cardiac chambers after bypassing the myocardium (coronary cameral fistula), directly opening into a coronary vein (coronary arteriovenous fistula) or a coronary artery opening into pulmonary trunk (coronary – pulmonary artery fistula). Coronary artery fistulae are thought to arise from persistence of sinusoidal connections during early embryonic development. Rarely, a coronary cameral fistula can also be iatrogenic, developing after cardiac catheterization or pacemaker placement, caused by tip of the catheter puncturing through a vessel wall into a cardiac chamber. A 75 year old female with a history of dyslipidemia and current tobacco abuse presented with recurrent left sided pressure-like chest pain. Her cardiac enzymes were negative and pain resolved with Nitroglycerine. EKG showed nonspecific ST-T changes. She was started on heparin protocol, aspirin, metoprolol and statin. Coronary angiography showed significant coronary cameral fistula of the left anterior descending artery with drainage limited to left ventricle (confirmed by no step-up in oxygen saturation on right heart catheterization), diffuse coronary ectasia (bordering on aneurysmal) of left anterior descending, right coronary artery and circumflex arteries, and no flow-limiting coronary artery disease. She had remained asymptomatic throughout her life despite having huge ectasias, but gradually developed diastolic dysfunction and impaired relaxation.
causing decreased micro vascular filling leading to micro vascular ischemia. She was discharged home on beta blocker to reduce myocardial oxygen demand and medical management for risk factor modification. Coronary artery fistulae are rare, seen in less than 1% of patients undergoing coronary angiography. Cardiac catheterization with angiography is the gold standard for diagnosis of these fistulae. Most fistulae are small and asymptomatic and often detected incidentally. Larger fistulae can lead to myocardial ischemia by coronary steal phenomenon especially with increased myocardial oxygen demands. Eventually the vessel attempts to compensate by progressive enlargement of the ostium and feeding artery leading to diffuse ectasia which can result in frank aneurysmal formation, intimal rupture, medial degeneration, calcifications, myocardial infarction, infective endocarditis, mural thrombi or high output congestive heart failure. If symptomatic, closure of the fistula is recommended either by transcatheter embolization or surgical closure of the fistular opening into the cardiac chamber.

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POOP FOR THOUGHT: FIRST FECAL TRANSPLANT IN SOUTHERN NEW JERSEY

Clostridium difficile (C. diff) derives its name from the Greek origin “kloster” meaning spindle and “difficile” which is Latin for difficult. These anaerobic spore-forming Gram-positive rods are notorious for causing severe diarrhea followed by pseudomembranous colitis and, in rare cases, toxic megacolon in patients whose gut flora is washed out after antibiotic treatment. This is a case report of severe refractory C. diff colitis treated with fecal transplant. A 76 year old female with history of self-treated intermittent chronic diarrhea for 2 years was admitted with frequent non-bloody diarrhea associated with fever. Her diarrhea began 4 months ago after multiple antibiotics for treatment of septic shock due to urinary tract infection. She was diagnosed with pseudomembranous colitis and positive stools for C. diff which was successfully treated with metronidazole, vancomycin, probiotic and cholestyramine. Computed tomography was negative for toxic megacolon. She was readmitted with severe persistent C. diff diarrhea which was now refractory to treatment with metronidazole, vancomycin, rifaximin, fidaxomicin and even nitazoxanide. Fecal transplantation was considered to be the next best treatment option. Due to time constraints, stool studies were not performed on donor stools from her daughter. She received successful fecal transplant via colonoscopy. Subsequently, she was discharged home on maintenance probiotic therapy with follow-up phone interviews demonstrating gradual improvement in her diarrhea. Her symptoms completely resolved 7 weeks after the fecal transplant. Repeat stool testing for C. diff have been negative. C. diff is transmitted by fecal-oral route. Spores remain viable in hospital and nursing home environments for a long time. Antibiotics damage normal human flora and remove protective bacterial species leading to Clostridium difficile diarrhea. Most commonly implicated antibiotics include fluoroquinolones, cephalosporins, and beta-lactamase inhibitors. Metronidazole, vancomycin and nitazoxanide are available for treatment of C. diff with variable success rates. Recently the FDA approved fidaxomicin is being used which was not effective in our patient. Fecal transplant or fecal bacteriotherapy was first introduced in 1958 as an attempt to restore normal colonic flora, eradicate C. diff and improve patient’s quality of life. Successful resolution of C. diff infections have been reported with fecal transplant, which is a relatively inexpensive and simple technique. This case demonstrates the persistent nature of C. diff and its refractoriness to various antibiotic treatment options. Fecal transplant is the novel technique suitable for such cases.

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HYPONATREMIA DUE TO POLY-PHARMACY

Hyponatremia is the most common electrolyte abnormality encountered in clinical practice. The most common presentation is a hypo-osmolar state. This group can further be classified depending upon volume status as hyper, hypo, or euovolemic states. Patients in the euovolemic group primarily develop hypotremia secondary to inadequate anti-diuretic hormone (ADH) suppression. Hypotremia can be multi-factorial, including side-effects of medications. We report a case of hypotremia secondary to poly-pharmacy. A 79-year-old female with history of hypertension, arthritis and gastroesophageal reflux disease presented with intractable nausea and vomiting. One week prior to admission her antihypertensive medications were adjusted because of severe headaches. She had also been taking both selective and non-selective nonsteroidal anti-inflammatory medications and opiates for her arthritis pain. In addition, she also took...
olmesartan/amlodipine/hydrochlorothiazide, clonidine, omeprazole and butalbital/aspirin/caffeine. On admission she was afebrile with a blood pressure 103/50, pulse 74 bpm, respiratory rate 18/min, and pulse oximetry 98% on room air. Physical exam was unremarkable. Chest x-ray and computed tomography of the abdomen showed no acute pathology. Laboratory studies showed severe hyponatremia 107 mmol/L, potassium: 3.4 mmol/L, serum chloride 69 mmol/L and serum uric acid level of 1.4 mg/dL. BUN and creatinine were 7 mg/dL and 0.6 mg/dL respectively. Random urine sodium was 57 mmol/L, random urine creatinine was 45.6 mg/dL and random urine chloride was 87mmol/L Serum and urine osmolality were 217 mosm/kg and 394 mosm/kg respectively. She received one liter bolus of normal saline while in ER which worsened serum sodium to105 mmol/L. The following day urine osmolality dropped to 61 mosm/kg. Her laboratory findings were consistent with SIADH. Decreased solute intake and volume depletion were also considered as possible causes. Nausea itself is the most potent stimulator of ADH. The use of thiazides, anti-inflammatory, opiates and caffeine all could produce or worsen hyponatremia and were discontinued. Slow correction of serum sodium was achieved over a period of 5 days with fluid restriction, increased oral solute intake, use of intravenous normal saline and minimal use of hypertonic saline (500 ml). She was kept off all anti hypertensives and analgesics and her outpatient serum sodium was maintained at 133 mmol/L. Age-related changes in pharmacokinetics and pharmacodynamics may alter course of intended therapy and lead to serious consequences from avoidable side effects. Various criteria have been established by expert panels in order to prevent inappropriate medication prescribing in older adults, e.g. Beers criteria. Our patient was receiving multiple medications. Volume depletion, potentiation of diuresis as well as development and worsening or SIADH are all possible due to combined or singular effects of these medications. Careful monitoring and measurement of electrolyte levels early in the course of new therapy in elderly patients should be considered.

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PERSISTENT LEFT SUPERIOR VENA CAVA IN A PATIENT WITH INTERRUPTED IVC AND NO CONGENITAL HEART DISEASE

Persistent Left Superior Vena Cava (PLSVC) is an uncommon congenital anomaly, with an incidence of 0.3% in the absence of associated congenital heart disease. It is often diagnosed incidentally, during cannulation of the central veins for cardiac monitoring or pacemaker placement. The incidence increases ten-fold when associated with other anomalies such as atrial or ventricular septal defects. As a result, careful noninvasive screening for PLSVC with a venogram or CTA may prevent unexpected incidents during procedures involving central venous access in at-risk populations. We report a case of PLSVC diagnosed incidentally during permanent pacemaker (PPM) placement in a patient with known congenital abdominal anomalies. Additionally our patient had a PLSVC in association with interruption of the inferior vena cava (IVC) with azygous continuation. A 67 year old female with a history of paroxysmal atrial fibrillation and partial situs inversus was admitted to the hospital for asymptomatic bradyarrhythmia and near syncope. She was normally controlled with olmesartan/amlodipine/hydrochlorothiazide, clonidine, omeprazole and butalbital/aspirin/caffeine. On admission, she was afebrile with a blood pressure 103/50, pulse 74 bpm, respiratory rate 18/min, and pulse oximetry 98% on room air. Physical exam was unremarkable. Chest x-ray and computed tomography of the abdomen showed no acute pathology. Laboratory studies showed severe hyponatremia 107 mmol/L, potassium: 3.4 mmol/L, serum chloride 69 mmol/L and serum uric acid level of 1.4 mg/dL. BUN and creatinine were 7 mg/dL and 0.6 mg/dL respectively. Random urine sodium was 57 mmol/L, random urine creatinine was 45.6 mg/dL and random urine chloride was 87 mmol/L. Serum and urine osmolality were 217 mosm/kg and 394 mosm/kg respectively. She received one liter bolus of normal saline while in ER which worsened serum sodium to105 mmol/L. The following day urine osmolality dropped to 61 mosm/kg. Her laboratory findings were consistent with SIADH. Decreased solute intake and volume depletion were also considered as possible causes. Nausea itself is the most potent stimulator of ADH. The use of thiazides, anti-inflammatory, opiates and caffeine all could produce or worsen hyponatremia and were discontinued. Slow correction of serum sodium was achieved over a period of 5 days with fluid restriction, increased oral solute intake, use of intravenous normal saline and minimal use of hypertonic saline (500 ml). She was kept off all anti hypertensives and analgesics and her outpatient serum sodium was maintained at 133 mmol/L. Age-related changes in pharmacokinetics and pharmacodynamics may alter course of intended therapy and lead to serious consequences from avoidable side effects. Various criteria have been established by expert panels in order to prevent inappropriate medication prescribing in older adults, e.g. Beers criteria. Our patient was receiving multiple medications. Volume depletion, potentiation of diuresis as well as development and worsening or SIADH are all possible due to combined or singular effects of these medications. Careful monitoring and measurement of electrolyte levels early in the course of new therapy in elderly patients should be considered.

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A RARE CASE OF NON-BACTERIAL THROMBOTIC ENDOCARDITIS IN MULTIPLE MYELOMA

Non-bacterial thrombotic endocarditis (NBTE), formerly known as marantic endocarditis, is a well-known complication of advanced malignancy. The word “marantic” was derived from the Greek word marantikos, meaning “wasting away” since it was usually seen in terminal cancer. NBTE is a serious and potentially under diagnosed manifestation in thrombophilic states such as cancer. Hypercoagulable states are often seen in patients with malignancy but only rarely reported in multiple myeloma associated with NBTE. We report a case of NBTE in a patient with multiple myeloma. A 72-year-old woman with history of diabetes mellitus, hypertension, dyslipidemia and multiple myeloma diagnosed in 2008 status post chemotherapy completed in 2011 presented with recurrent episodes of transient ischemic attacks (TIAs). Prior carotid ultrasound and echocardiography were unremarkable. She is a life-long non-smoker and denied illicit drug use. On examination, there were no carotid bruits. She had a grade II systolic murmur over the lower left sternal border. Transthoracic echocardiography showed severe mitral annular calcification but vegetation could not be excluded. Transesophageal echocardiogram showed a 1.5 cm irregular, non-mobile, cystic mass on the posterior mitral valve leaflet suggestive of vegetation, although not very typical. Cardiac catheterization did not show significant coronary artery disease. She did not have any other signs/symptoms suggestive of bacterial endocarditis. She was diagnosed with non-bacterial thrombotic endocarditis and started on heparin followed by warfarin. She has not had any subsequent TIAs. NBTE vegetations are friable eosinophilic masses formed by degenerating platelets and fibrin. They are typically small, less than 1cm in size, broad-based, and irregular in shape. There is a higher prevalence of left sided valvular involvement, particularly on the mitral valve. Due to its small size compared to infective endocarditis, NBTE can easily go undetected on transthoracic echocardiography and should be suspected in individuals with recurrent cerebrovascular accidents, TIAs or other thromboembolic events and underlying malignancy. If initial transthoracic echocardiography does not show evidence of vegetation, transesophageal echocardiography should be considered to evaluate for small (< 3 mm) vegetations, particularly in the absence of any cardinal signs of bacterial endocarditis. As per 2008 ACCP Guidelines, patients with NBTE and systemic or pulmonary emboli should be anticoagulated if there are no contraindications.

A TOXIC CASE OF THYROIDITIS

Thyrotoxicosis (thyroid storm) is a rare but severe complication of hyperthyroidism, often occurring when a thyrotoxic patient becomes physically stressed. Symptoms often include elevated temperature, arrhythmias/tachycardia, vomiting, diarrhea, coma and even death. Thyroid storm requires prompt treatment and hospitalization. A 41 year-old male with history of untreated hyperthyroidism and seizures presented to the emergency department with abdominal pain, nausea, vomiting, diarrhea and shortness of breath. On physical examination he had right upper quadrant tenderness. TSH was less than 0.01 uIU/mL. T3 was greater than 20 ng/dL and T4 was 9.14 ng/dL. AST was elevated at 2264 U/L and ALT was also elevated at 1687 U/L. Patient received CT scan of the abdomen with contrast. That evening he became unresponsive, had PEA cardiac arrest and was resuscitated and intubated. He was started on propranolol, propylthiouracil, iodine and hydrocortisone. Eventually he was discharged home on methimazole. He had difficulties adhering to his medication regimen and was being considered for thyroidectomy. This patient presented with classic symptoms of hyperthyroidism. His state progressed to thyroid storm after the administration of contrast dye containing iodine. Iodine-induced thyrotoxicosis was first described when iodine supplementation was introduced to areas of iodine deficiency. In the United States, iodine-induced thyrotoxicosis is usually iatrogenic, after iodine-containing contrast is given. Often the patient has underlying Graves' disease. This case shows that recognition of hyperthyroidism is very important because administration of iodinated contrast for diagnostic tests may tip a patient with this condition into a thyrotoxic state.
**RHABDOMYOLYSIS CAUSED BY TOTAL BODY POTASSIUM DEPLETION FROM LAXATIVE ABUSE**

Rhabdomyolysis is characterized by myonecrosis with release of intracellular muscle components into the circulation. Symptoms include malaise, fever, tachycardia, nausea and vomiting, and abdominal pain. Associated electrolyte abnormalities include hyperkalemia, hyperphosphatemia and hypocalcemia. We present a case of rhabdomyolysis with acute kidney injury caused by severe hypokalemia from laxative abuse. A 64-year-old female with history of hypertension, gout, osteoarthritis and bipolar disorder was admitted with increased confusion, lethargy and drowsiness. Family members reported generalized weakness and myalgias for two days, with frequent falls and multiple bruises. History revealed intentional weight loss of 50 pounds in the past year with strict diet and chronic use of laxatives. Other medications were colchicine, olmesartan, furosemide, oxycodone-acetaminophen, cyclobenzaprine, clonazepam and pregabalin and recently prn ibuprofen. On admission she appeared volume-depleted with BP 80 / 49, pulse oximetry 95% on room air and pulse 96 bpm. Her BMI was 37, creatinine 3.0 mg/dL (baseline 1.4 mg/dL 8 months prior) BUN 34 mg/dL, albumin 3.6 g/dL, potassium 2.8 mEq, sodium 135 mEq and creatine kinase 17000 units/L with CK-MB 84.05 ng/mL. Urine myoglobin was 3035 ng/ml, random urine sodium 55.0 and urine creatinine was 49.6. Chest X-ray, computed tomography of the head and kidney ultrasound were unremarkable. She received appropriate hydration and potassium replacement and was discharged 8 days later with normal serum potassium, creatine kinase of 555 units/L and creatinine 1.2 mg/dL. The mechanism of hypokalemic rhabdomyolysis is not completely understood. During exercise there is release of potassium from skeletal muscle cells that mediates an increase in muscle perfusion by producing vasodilatation. Severe hypokalemia with serum potassium less than 2.5 mEq/L may produce vasoconstriction of the skeletal muscle vasculature, producing ischemic injury and muscle breakdown that may promote the development of rhabdomyolysis. In hypokalemic rhabdomyolysis, the serum potassium level may underestimate or mask the underlying total body potassium depletion because of release of potassium from intracellular stores due to myonecrosis. Our patient had a history of chronic laxative abuse which aggravated the potassium loss compounded by decreased oral intake secondary to a vigorous self-induced weight loss program. Her treatment was mainly supportive with potassium replacement therapy.

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**A GOODPASTURE"S ALPHABET ANTIBODY SOUP**

Anti Glomerular Basement Membrane (GBM) disease is a rare antibody-mediated disorder that occurs in fewer than one in a million population and results in acute or rapidly progressive glomerulonephritis. Goodpasture’s Syndrome is anti-GBM disease with alveolar hemorrhage. Here we describe an unusual case of Goodpasture’s Syndrome that presented with anti-myeloperoxidase (MPO), anti-proteinase 3 (PR3) and perinuclear antineutrophil cytoplasmic antibody (P-ANCA) positivity. A 56-year-old female with a history of chronic kidney disease stage III, bilateral retinal detachment with blindness, sensorineural deafness, hypothyroidism and intellectual developmental disorder, was admitted for evaluation of acute kidney injury and anemia. She had intermittent spontaneous epistaxis for 3 months prior. She had used substantial doses of Ibuprofen for headaches recently. She had mild fatigue but otherwise had no overt gastrointestinal or genitourinary bleeding, uremic or vasculitic symptoms. Physical exam disclosed mild dehydration but no skin, cardiac, pulmonary or gastrointestinal abnormalities. She had serum creatinine of 6.5 mg/dL, mild non anion gap metabolic acidosis and normocytic anemia with hemoglobin of 6.1 g/dL. Urine analysis showed hematuria, proteinuria and granular casts. There were no red cell casts or acanthocyturia. Fractional excretion of sodium was greater than 2%. Renal ultrasound showed nephrosclerosis without hydronephrosis. She received intravenous fluids, sodium citrate and packed red cell transfusions. Stool was negative for occult blood. HIV, hepatitis B and C, and cryoglobulins were negative. Serum electrophoresis and complement levels were normal. ANCA and anti-GBM antibodies were pending. She was discharged with serum creatinine of 3.7 mg/dL. Three days later, she was readmitted due to hemoptysis, fever and acute kidney injury with serum creatinine of 3.5 mg/dL. Chest film showed diffuse opacities and coarse infiltrates throughout the right lung and base of the left lung. She became increasingly dyspneic and hypoxic requiring intubation. Bronchoscopy revealed alveolar hemorrhage.
Previously pending serologies came back positive for anti-GBM, P-ANCA, anti-PR3 and anti-MPO antibodies. She received intravenous pulse steroids, oral cyclophosphamide, daily plasmapheresis and antibiotics. She developed oliguric renal failure and required continuous renal replacement therapy. Oral prednisone and cyclophosphamide were continued. ANCA and anti-GBM antibodies were cleared within 2 weeks. She could not be weaned from the ventilator. Her family opted terminal weaning after which she expired. Up to 40% of anti-GBM disease are positive for ANCA that is typically anti-MPO. Treatment involves immunosuppressive regimen for 2 to 3 months along with initial plasmapheresis until antibodies are cleared. Dual anti-PR3 and anti-MPO positivity with anti-GBM positivity is extremely rare. These patients have an extreme course and are prone to recur after remission. They require plasmapheresis with aggressive immunosuppression and maintenance therapy for 24 months as in ANCA vasculitis.

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DARK SIDE OF COLCHICINE

Colchicine was the first FDA approved drug for treatment of acute gout, familial Mediterranean fever and prophylaxis against gout flares. Currently, it is a second line drug because of its narrow therapeutic index and toxicity on high doses. We report a case of colchicine toxicity. A 48 year old male with history of gout presented with new onset lethargy. On arrival, he complained of abdominal pain and vomiting after taking 200 tabs of ibuprofen (unknown dose) approximately 8 hours prior in an attempt to commit suicide. On examination he was poorly responsive with blood pressure 116/88 mmHg, pulse 94/min, oxygen saturation 92% on 2L nasal cannula oxygen with diffuse abdominal tenderness on deep palpation. Creatinine was 2mg/dL, total bilirubin 1.5mg/dL, aspartate transaminase 141U/L, alanine transaminase 83U/L, WBC 7.5/µL with 17% bands, platelets 171,000/µL, prothrombin time 12.8, INR 1.2, pH of 7.31, bicarbonate 25mmol/L, calcium 9.5mg/dL, salicylate levels of 0mg/dL, acetaminophen level of 10µg/mL. Ten hours later he confessed to consuming an unknown amount of colchicine 0.6 mg tablets. He was no longer a candidate for activated charcoal administration. He developed acute oxygen desaturation requiring mechanical intubation. He had respiratory acidosis with both anion gap and non anion gap metabolic acidosis. Within 24 hours of admission, he developed multiorgan failure including anuric acute renal failure, NSTEMI and cardiogenic shock. He was started on vasopressors, bicarbonate drip, dextrose drip and CVVHD. Blood glucose fluctuated between 20-500mg/dl, creatinine was 4.0mg/dL, pH 7.03, bicarbonate 9, calcium 5.4mg/dL, platelets 29,000, INR 2.2 after 8 U FFP, lactate of 13mmol/L. Within 48 hours of admission, he succumbed to cardiac arrest despite resuscitative measures. Colchicine inhibits neutrophil activity leading to anti-inflammatory action. It also inhibits mitosis in rapidly dividing cells, thus affecting bone marrow. Gastrointestinal (GI) symptoms occur due to enterohepatic circulation and rapid GI epithelial turnover. Cardiogenic shock is caused by inhibiting cardiac contractility. Kidneys demonstrate acute tubular necrosis for which dialysis is only a supportive measure. Overdose with nonsteroidal anti-inflammatory agents or colchicine is notorious for causing multiorgan failure as seen in our patient. Colchicine toxicity can be extremely lethal due to its irreversible action. Only supportive treatment and resuscitative measures are available to date for colchicine toxicity. Colchicine specific Fab fragments have been used for lethal overdose but are not commercially available due to high cost and rare cases of colchicine poisoning. Therefore, caution should be practiced with prescribing these medications due to fatal side effects.

CAPITAL HEALTH (SABA A. HASAN, MD)

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AIRWAY COMPRESSION AS A COMPLICATION OF GASTRIC BAND SURGERY

Introduction: Laparoscopic gastric banding is the second most common weight loss surgery, after gastric bypass. The most significant problem associated with gastric banding has been alteration in the size of the stomach pouch which is isolated above the band. However, respiratory distress secondary to severe dilatation of the esophagus and compression of the airway is not a widely recognized complication of bariatric surgery. Case: A 69 year old Caucasian female, with a history of gastric lap band surgery 2 years prior to admission,
presented to the ED with shortness of breath for three days. She was recently discharged from another hospital a week prior due to pneumonia. Upon admission, she had no abdominal pain, nausea, vomiting, or dysphagia; however, she did have shortness of breath, diaphoresis, and stridor. In the ED, CT chest showed no pulmonary embolus or pneumonia. The CT did show a 3.3 X 4.5 cm dilation of the esophagus proximal to the site of the gastric band, extending superiorly to the level of the thyroid gland. A naso-gastric tube was placed to decompress the esophagus, and IV steroid were administered but no significant relief occurred. She was transferred to the facility where her original surgery was performed, and the bariatric surgery team deflated the gastric band, resulting in complete relief of symptoms. An upper endoscopy after band deflation showed mild gastritis. Discussion: Bariatric surgery is associated with a wide variety of complication, even when the intended weight loss is successful. Since the surgery 2 years ago, our patient lost 84 pounds, but had multiple hospital admissions, most recently for pneumonia, which in retrospect was suspicious for aspiration. Teaching Point: Generalists in internal medicine and emergency medicine must maintain a high index of suspicion for complications of bariatric surgery, as patients frequently do not present to their bariatric surgery team, particularly when symptoms appear late after successful surgery.

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VIDEO-ASSISTED THORACOSCOPIC LUNG BIOPSY IN INTERSTITIAL LUNG DISEASE AND ACUTE RESPIRATORY FAILURE: DOES RISK OUTWEIGH THE BENEFITS?

INTRODUCTION: Video-assisted thoracoscopic (VAT) lung biopsy is frequently sought in patients with interstitial lung disease to identify the underlying etiology and guide appropriate treatment. The procedure has a high diagnostic yield, and relatively low morbidity and mortality due to its minimally invasive nature. However, few studies have addressed issues surrounding patient selection in acute cases of respiratory failure. We present a case highlighting the importance of candidate selection for VAT lung biopsy in order to improve mortality. CASE DESCRIPTION: A 73-year-old male with chronic obstructive pulmonary disease, chronic tobacco use, peripheral vascular disease, chronic kidney disease stage III, and systolic heart failure presented with gradually worsening shortness of breath over a two week period along with yellowish productive cough. He worked in a factory as a press operator before retiring 4 years ago. Imaging studies revealed diffuse interstitial thickening and fibrosis with the presence of bilateral infiltrates. The patient was admitted with a clinical diagnosis of interstitial lung disease with superimposed pneumonia and he was ordered intermittent bilevel positive airway pressure (BiPAP), broad-spectrum antibiotics and corticosteroids. He remained unresponsive to aggressive treatment and a VAT lung biopsy was requested in order to determine etiology. After an uneventful procedure, the patient was transferred back to the intensive care unit intubated and in the first 24 hours post-operative period became hypotensive and more hypoxic requiring vasopressor support. Chest x-ray revealed worsening bilateral infiltrates and a PaO2/FiO2 ratio of 36 suggesting worsening of acute respiratory distress syndrome. Pressure support and positive end expiratory pressure were maximized. Arterial blood gasses revealed worsening hypercapnia and severe respiratory acidosis. The biopsy report of the lung specimen revealed acute lung injury pattern with organizing pneumonia. Despite aggressive vasopressor support and broad-spectrum antibiotic coverage the patient progressed to multi-organ failure, and expired in the next 24 hours. DISCUSSION: In patients with underlying pneumonia and interstitial lung fibrosis, lung biopsy procedures may exacerbate the inflammatory process, worsen the lung injury and lead to multi-organ failure and death. Careful selection of patients in acute respiratory failure is crucial in improving mortality associated with VAT lung biopsy.

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MAN, MIND AND MADNESS!

Introduction: Incidence of syphilis is once again increasing throughout the world. The disease is known as the great mimicker because of its wide range of clinical presentations. Symptomatic neurosyphilis can develop at any stage of the disease. Herein reported is a case of a 51-year-old man who initially presented with symptoms of mania mimicking schizophrenia, but was subsequently diagnosed with general paresis of the insane. Case: A 51 year old African American man presented with acute psychosis to the emergency department. His
medical history was significant only for long standing mental illness diagnosed as paranoid schizophrenia. Family history was unremarkable. During examination he was withdrawn answering only in monosyllables. Vital signs were stable. He was found to have Argyll Robertson pupils which responded to accommodation but not to light. He also had an abnormal Romberg’s test. He scored a 23/30 on the MMSE scale suggestive of moderate cognitive impairment. Imaging studies which included MRI of the brain and spine were negative. Initial laboratory studies were remarkable for his RPR being positive in 1:8 dilution and FTA-ABS was reactive. His trepanoma pallidum AB particle agglutination test was also reactive. A lumbar puncture revealed CSF that was reactive to VDRL in a 1:1 ratio. HIV testing was negative. The patient was admitted and treated with IV penicillin. Discussion: The patient described here presented with multitude of psychiatric signs and symptoms. Patients with neurosyphilis can also present with many different physical or neurologic symptoms that lead to admission or follow-up at a medical or neurology unit. What was interesting about this case was that the patient showed exclusively psychiatric manifestations, leading to direct admission to a psychiatric unit rather than a medical or neurology unit with psychiatric consultation. Clinicians—including Internists and neurologists, and especially psychiatrists—need to have a high index of suspicion for neurosyphilis, which may have an exclusively psychiatric presentation and because of the increasing incidence of syphilis in recent years.

Teaching points: Neurosyphilis must be considered in the differential diagnosis of psychiatric conditions, even in the absence of medical or other neurological findings.

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BUBBLE IN THE BRAIN- NOT TO BE TAKEN LIGHTLY!

Introduction: Air embolism is a well-published complication arising from central venous catheter use. Literature and case studies provide information regarding clinical sequelae. This case report describes the neurological complications likely caused by a cerebral air embolism related to central venous catheter removal. Case: We report a 48 year old Caucasian man who was admitted due to swelling of his left leg. Venous duplex of the left leg showed presence of a large DVT and the CT of his chest showed multiple sub segmental PE. The patient was started on heparin drip and later bridged with warfarin therapy. His work up for hypercoagulable state was negative. The patient was being discharged. While he was sitting in a reclining position in his chair, during the process of removal of his central venous catheter from the right side of the neck, he rapidly became confused, diaphoretic, and his mental status declined. He also had a seizure activity with incontinence of bladder. He received 2 doses of 1 mg IV lorazepam. After sometime the patient regained consciousness. Patient remained in postictal phase for some time. Later on examination he did not show signs of any cranial nerve involvement nor was there any weakness. EEG was unremarkable. CT angiography of the brain showed presence of air in the cavernous sinus and along the pterygoid plexus. The patient was then transferred to ICU for overnight observation. He gradually improved and at the time of discharge, the patient did not have any neurological deficit. Discussion: Venous air embolism is an infrequent complication of invasive diagnostic and therapeutic maneuvers. The cardiovascular, pulmonary, and central nervous systems may all be affected, with severity ranging from no symptoms to immediate cardiovascular collapse. Therapeutic interventions include mechanical measures, such as positioning, withdrawal of air from the right atrium, and measures aimed at reducing bubble size and treatment with hyperbaric oxygen. Teaching points: There is an abundance of literature describing best practice, complications, and treatment of venous air embolism associated with central line catheter use. Utilization of central venous catheters is increasing. With increased utilization comes the responsibility to improve commonplace knowledge and ensure that practice guidelines and protocols are dependable and consistent.
RICHTER’S TRANSFORMATION – A RARE PRESENTATION WITH INVOLVEMENT OF OPTIC NERVE

Introduction: Richter’s transformation denotes the development of high-grade non-Hodgkin lymphoma, prolymphocytic leukemia, Hodgkin disease, or acute leukemia in patients with chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma. Involvement of the optic nerve as a presenting symptom is rare. Case: A 61 year old Hispanic man presented with decrease in vision in the left eye for the past 5 days. There was no history of any weakness, any significant headache or vomiting. His past medical history was significant only for hypertension. Family history was unremarkable. On examination his vital signs were stable. He was found to have generalized lymphadenopathy. His visual acuity was 20/30 in the right eye and 20/60 in the left eye with papilledema. Speech and language, along with cranial nerves, motor and sensory exams were normal. Laboratory evaluation found mild anemia with 13% reactive lymphocytes. The CT scan of his chest, abdomen and pelvis revealed multiple lymph nodes. MRI of the brain showed diffuse enhancement of the left optic nerve, with slight enlargement suggestive of optic neuritis. Also there was presence of subcortical white matter FLAIR hyperintense foci in the cerebral hemispheres bilaterally as well as FLAIR hyperintensity within the pons and cerebellar peduncles. Lumbar puncture showed lymphocytosis and increased protein. CSF fluid showed many poorly preserved atypical lymphoid cells suspicious for lymphoproliferative disorder. Finally an excisional lymph node biopsy in neck revealed large B-cell lymphoma with immunoblastic features, arising in a background of small lymphocytic lymphoma (SLL). The lymph node architecture was effaced. Mitotic figures were seen. Immunostains showed the small lymphoid cells to be B cells. The larger lymphoid cells were B suggestive of Richter (Large B-cell) transformation of SLL. The patient was transferred to a tertiary center for bone marrow and cytogenetic evaluation and for chemotherapy. Discussion: Richter’s transformation or syndrome denotes the development of aggressive lymphoma that can arise in patients with small lymphocytic lymphoma (SLL). It frequently arises in lymph nodes or bone marrow and rarely presents with extra nodal involvement. Presenting features typically include a rapid clinical deterioration. Involvement of the optic nerve with decrease in vision is a rare presentation such that this case expands the clinical spectrum of organ involvement in Richter’s syndrome.

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RIGHT ATRIAL THROMBUS ASSOCIATED WITH INDWELLING DIALYSIS CATHETERS

Learning Objective: Right atrial thrombus (RAT) associated with indwelling dialysis catheters may be successfully managed conservatively. Background: Hemodialysis catheters (HCs) were developed for short term use in hemodialysis patients with ESRD as the arterio-venous (AV) fistula takes months to mature. However, what was intended as a temporary measure has become more long-term as many ESRD patients have difficult anatomy for AV fistulae. Infection and thrombosis are challenging complications since though catheter removal and/or thrombectomy may be ideal, this strategy must be weighed against the need for continued dialysis access. We report 2 patients who presented with bacteremia and RAT secondary to indwelling dialysis catheters who were successfully managed with conservative strategies. Case series presentation: Case 1: A 43 year old African American male with ESRD secondary to interstitial nephritis and diabetic nephropathy presented with fever, chills, and generalized malaise. Blood cultures were positive for Staphylococcus epidermidis and Streptococcus agalactiae. The source of bacteremia was identified as the right internal jugular dialysis catheter placed 5 months earlier. Transthoracic echocardiogram showed a large mass within the right atrium. The CT chest confirmed a low density mass within the right atrium adjacent to the catheter tip measuring 2.5 x 1.0 x 2.0 cm. Intravenous heparin was bridged to warfarin, the catheter was exchanged, and repeat transthoracic echocardiogram two months later showed no mass in the right atrium. Antibiotics were continued for 6 weeks. Case 2: A 73 year old African American female with ESRD secondary to diabetic and hypertensive nephropathy presented with rapid atrial fibrillation and sepsis. Blood cultures grew Staphylococcus epidermidis and Staphylococcus capitis. Transthoracic echocardiogram showed dilated right and left atria with a large mobile echogenic density in the right atrium measuring 1.9 x 1.6 cm. Transesophageal echocardiogram showed a multi-lobed mass within the right atrium attached to the inferior
vena cava and the right atrial wall with a catheter in the right atrium. The patient refused surgical thrombectomy and the catheter was not exchanged due to concern for thromboembolism. Intravenous heparin was bridged to warfarin, and repeat transthoracic echocardiogram two months later showed no right atrial mass and patient now has a functional AV fistula used for HD. Antibiotics were continued for 6 weeks.

Discussion: The optimal management of dialysis catheter-associated RAT is unclear. Thrombectomy has been reported to be associated with a lower mortality compared with conservative management with anticoagulation and antibiotics. However, some patients may refuse or are poor candidates for surgery. All ESRD patients require stable dialysis access which complicates the decision process. Literature has advocated conservative approach to clots less than 2 cm, and thrombectomy for larger clots, however our experience suggests larger clots may be successfully managed conservatively.

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PERIPHERAL NERVE INVOLVEMENT IN GIANT CELL ARTERITIS

Background: The diagnosis of Giant cell arteritis (GCA) is based on clinical signs and symptoms and confirmed by serological tests (elevated ESR, CRP, Platelets) and positive temporal artery biopsy. However, GCA may affect multiple arterial beds. We treated a patient with biopsy proved GCA in whom sural nerve biopsy also showed evidence of arteritis. Case: An 83yr-old man presented with 3-month history of unremitting temporal headaches and blurred vision. On examination he exhibited temporal scalp tenderness. Temporal artery biopsy confirmed the diagnosis of Giant cell arteritis. Steroid therapy was initiated, however due to progressive bilateral lower extremity weakness and sensory loss plus persistently elevated inflammatory markers, a sural nerve biopsy was performed. Histological exam showed focal perivascular lymphocytic infiltrates and focal epineural blood vessel scarring consistent with arteritis. Discussion: Peripheral nerve involvement may occur associated with other signs and symptoms in GCA. Arteritis may injure peripheral nerves diffusely or individually, particularly the peroneal, tibial, and sural nerves. Also, the brachial plexus is particularly vulnerable. Clinicians must be aware of the possibility of systemic manifestations of GCA, and recognize symptoms of peripheral neuropathy as part of the spectrum of disease involvement. In individuals with suspected GCA, the symptomatic peripheral nerve may be considered as an additional site for biopsy.

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RELAPSING THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP) AND HEMOLYTIC UREMIC SYNDROME (HUS) WITH RETINAL DETACHMENT

Background: Acute thrombotic thrombocytopenic purpura (TTP) is characterized by widespread deposition of microvascular occlusive thrombi of platelets and fibrin without mediation by an inflammatory process. Patients may experience a wide variety of complications of this illness, such that clinicians must be vigilant for manifestations in almost all organ systems. Case: A 31-year-old African American male with PMH of HTN presented to ER with nausea and vomiting for 2 days. He denied fever, abdominal pain, diarrhea, and headache. On exam, he did not appear acutely ill. Vitals were normal except BP 135/97. The remainder of the physical examination was normal except ecchymoses to left arm, right arm, right leg and right flank. Pertinent lab data on admission included platelet count 16 x 10^9/L, moderate schistocytes on blood smear, BUN 76 mg/dl, creatinine 7.07 mg/dl and AST 100 U/L, indirect bilirubin 4.9 mg/dl, and LDH 7768 U/L. Plasmapharesis was started in the morning, and during the ICU stay, he developed low-grade fever, but no mental status changes. Kidney function did not improve initially, but the patient refused hemodialysis. He never became fluid overloaded and after 4 rounds of plasmapharesis, the renal function did improve. On day 12 of hospitalization, the patient complained of blurry vision in the left eye. Central serous retinal detachment was found on ophthalmological examination. No specific therapy was advised, and symptoms subsided in 4 days. There was an early relapse of the TTP on day 14 leading to the addition of rituximab therapy. He was discharged with stable platelet count and low LDH level, currently the patient is followed by hematology with no further relapses. The diagnosis of TTP/HUS was further confirmed weeks later by a less than 2% ADAMSTS-13 activity level. Discussion: This case of TTP/HUS presented with thrombocytopenia, microangiopathic hemolytic anemia, renal failure, evolving fever during treatment, but no neurological changes. He experienced
a rare but recognized complication of retinal detachment which is seen more commonly in relapsing cases associated with hypertension. Plasmapharesis was successful in avoiding dialysis, however multiple organ systems may be involved and requires that clinicians caring for these patients maintain a high index of suspicion with any new symptoms the patient may experience.

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A CLOT BY ANY OTHER NAME IS STILL A CLOT, UNLESS YOU THINK IT'S A STONE

Deep vein thrombosis, divided into distal and proximal vein thrombosis, and acute pulmonary embolism are two manifestations of the same disorder, venous thromboembolism (VTE). Compression ultrasonography is the test of choice when VTE is suspected. However, one drawback of this test is that it does not effectively evaluate the iliac veins. Thus, physicians must consider using contrast venogram in a high risk patient regardless of the results of doppler ultrasonography. Case: A 38 year old Caucasian male presents to the ED on October 29, 2011 due to pressure-like pain in the left inguinal region. The pain was exacerbated by movement, and relieved with rest. Because of a history of prior right sided DVT and PE in 2006 after an ankle fracture, a venous doppler ultrasound was performed, but was negative. An abdominal CT identified a 1.3 mm calcification in the left kidney. In addition, a urinalysis suggested a urinary infection, and he was discharged on pain medications and ciprofloxacin for possible urinary tract infection. The pain was attributed to the renal calculus seen on CT. The next morning, the patient’s thigh had swollen considerably with severe pain, diaphoresis, shortness of breath, palpitations, and lightheadedness. He returned to the ED, where a left lower extremity venogram revealed extensive left iliac vein DVT. The patient received thrombolysis treatment, Angiojet thrombectomy, stent placement in the iliac vein, and an IVC filter. In addition, a chest CT was negative for PE. The venogram revealed that the patient had May-Thurner syndrome, an anatomical cause for VTE in iliac vein. When the iliac vein is compressed between the vertebral column and the iliac artery, it can produce stasis of blood at the site of compression leading to development of clot. The first episode of VTE in 2006 was attributed to immobility as the cause of DVT, which in retrospect may have been one precipitating factor in the setting of the unrecognized anatomical variant. The second episode of VTE was initially misdiagnosed as a renal calculus, but the patient rapidly worsened, and hence, it was not until the venogram was performed that the actual cause of VTE was identified. Teaching Points: 1. Physicians must remember to consider a venogram when there is suspected VTE in a high risk patient. 2. Incidentally found renal calculi are frequently not the cause of patient’s symptoms, especially when the clinical syndrome is atypical for renal colic.

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SERRATED POLYPYSIS SYNDROME: WHAT’S A SERRATED POLYP??

Introduction: Colorectal cancer ranks third in both incidence and cause of cancer death in both men and women. Traditionally colorectal adenoma is considered the precursor lesion to colorectal cancer with two main pathways of carcinogenesis, namely that of chromosomal instability, which gives rise to Familial Adenomatous Polyposis, and that of microsatellite instability, which is thought to give rise to Lynch Syndrome. Over the past decade, some “serrated” polyps have been recognized as premalignant lesions and as markers for synchronous and metachronous colorectal neoplasia. This has given rise to another pathway in which the “serrated poly” is the precursor lesion for colorectal cancer. It is associated with an increased risk of colorectal cancer by 25 to 40%, and an increased risk for developing interval colorectal cancer between otherwise normal screening procedures. Case Report: A 66 year old Caucasian female underwent a routine colonoscopy. She had no other prior colonoscopies and had no symptoms such as bleeding, weight loss, change in bowel habits or abdominal pain. Patient was noted to have mild anemia on her recent lab work, with Hg of 11.7gm/dL and MCV of 90.5 fl. Her past medical history included successfully treated Grade I Astrocytoma (well differentiated) 20 years ago, Obstructive Sleep Apnea, Hypertension, Transient Ischemic Attack, and Dyslipedemia. She reported occasional alcohol use, no tobacco or drug use, and her family history was negative for colon cancer, polyps, or endometrial cancer. The colonoscopy demonstrated 30 polyps varying in size from 0.5 cm to 2-3 cm throughout the right colon, transverse colon and sigmoid colon. Biopsies
revealed "sessile serrated" adenomas, characterized by prominent histological serrations, distorted and dilated bases of crypts, with aberrant branching. No dysplasia was noted. The results met criteria for diagnosing the Serrated Polyposis Syndrome (SPS), and the patient elected for surveillance colonoscopy at one year instead of surgical intervention. Discussion: A histological description of serrated polyps as an increasingly common result on pathology reports, and internists must be ready to interpret these findings for their patients and refer for appropriate surveillance and therapy. Individually, these polyps are recognized to be pre-malignant, and the SPS may confer a high enough risk to refer for surgery. Ongoing studies are underway to characterize the biology and clinical significance of this new type of colon polyp. Teaching Point: 1. Recognize new developments in Colorectal Cancer Screening in relation to SPS.

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PREVALENCE AND SIGNIFICANCE OF VITAMIN D DEFICIENCY IN AN URBAN RESIDENTS-CONTINUITY CLINIC

Background: Vitamin D deficiency is now recognized as a pandemic. The main causative factor of low levels of Vitamin D is lack of sun exposure. At risk populations have been identified: females (pregnant and postmenopausal), African Americans, extremes of age, high BMI, malabsorption syndromes, patients on anti-seizure medications and chronic use of glucocorticoids. Vitamin D deficiency causes muscle weakness, skeletal demineralization and rickets in children. It will precipitate and exacerbate osteopenia, osteoporosis and increase risk of fractures in the elderly. Vitamin D deficiency increases the risk of metabolic, neoplastic and immune disorders such as Type 1 diabetes mellitus and multiple sclerosis. It is also associated with increased risk of atherosclerosis, hypertension and infectious diseases. The goal of our project is to identify the prevalence and characteristics of patients with Vitamin D deficiency in our community and to determine if there is an association between age and BMI with Vitamin D concentrations. Methods: We performed a retrospective chart review of three hundred four (N=304) patients (primarily uninsured and underinsured) whose vitamin D levels were checked in our resident’s continuity clinic between August 2011- August 2012. The information was entered into an Excel spreadsheet for analysis. Patients were classified into different groups (gender, age, race, BMI and season). For our study purpose, vitamin D levels were defined as deficiency <20ng/ml, insufficiency 21-30ng/ml, or normal (>30ng/ml). Results: Our study found that overall 37.5% of patients had Vitamin D deficiency and 39.8% had insufficiency. African Americans (79%) and Hispanics (84%) had the highest prevalence of vitamin D deficiency and insufficiency with the mean values of 22.7 ± 9.61 ng/ ml and 24.1 ± 9.83 ng/ml respectively. Female gender was found to have a higher prevalence of both vitamin D deficiency and insufficiency (77%) than male gender (23%) with a mean value of 24.6 ± 9.46 ng/ml. We found a negative correlation between Vitamin D levels and BMI (p <0.046) and a positive correlation between age and Vitamin D levels (p <0.001). The overall prevalence was lower in summer compared to other seasons, but statistically this was not significant. Discussion: Low Vitamin D levels are highly prevalent in the population that we serve. A descriptive analysis reveals that the population at highest risk includes people who are African American, Hispanic, and female. The correlation with age suggests that even the young adult population is at risk. This is important, since it provides information on possible ‘at risk’ populations and can help residents identify groups for screening.

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EXPECTED AND UNEXPECTED SEQUELAE OF METFORMIN OVERDOSE

Background: One of the adverse effects of metformin is lactic acidosis which might occur at a therapeutic dose but more commonly in the case of an overdose. The incidence is estimated to be 2 to 10 per 1000 patients receiving metformin per year. Metformin inhibits hepatic gluconeogenesis from lactate resulting in additional lactate and substrate for lactate production. Literature shows several cases of metformin overdose leading to fatal or nearly fatal lactic acidosis/anion gap metabolic acidosis. Other metabolic effects of metformin may appear in the setting of overdose that are less familiar. Case: An 18-year old obese, diabetic female ingested about 57g of metformin in a suicide attempt. This patient experienced severe metabolic derangement within
the first 9 hours of presentation from nearly normal laboratory values (pH 7.42, serum creatinine 0.72 mg/dl, serum bicarbonate 21 mmol/L) and stable vital signs to a severe, nearly fatal anion gap metabolic acidosis (pH 7.01, Bicarbonate 7 mg/dl, anion gap 35) plus severe lactic acidosis (lactic acid 15.6 mmol/L) associated with acute renal insufficiency (serum creatinine 1.69 mg/dl). The patient required emergency hemodialysis to survive despite aggressive intravenous fluids and sodium bicarbonate administration. Although the patient was NPO because of nausea and vomiting, and despite her metformin overdose, her blood sugar unexpectedly kept rising from a value of 235 mg/dl on presentation to a high of 579 mg/dl requiring insulin drip therapy.

Discussion: Metformin is one of the commonly used antihyperglycemic medications. Although metformin is known not to cause hypoglycemia at a therapeutic dose, in the case of an overdose, clinicians may choose to guard against hypoglycemia. However, our case, along with other reports in the literature, suggests that metformin overdose may be associated with a paradoxical hyperglycemia that may need insulin therapy. In metformin overdose, clinicians must expect severe lactic acidosis and prepare for hemodialysis as soon as possible for optimal outcome. Finally, it is important to consider metformin overdose in a case of unexplained lactic acidosis.

DREXEL - ST. PETER’S (NAYAN K. KOTHARI, MD)

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NEEDLE LICKERS ABSCCESS -- BY EIKENELLA CORRODENS

Introduction: Infections at insulin injection sites are very rare. Occasionally an infection does occur and can lead to an abscess. The most common organism is Staphylococcus aureus, however, other rare organisms do exist. We report a case of injection site abscess due to Eikenella corrodens. Case: A 43-year-old woman with diabetes presented with a 3-week history of fever with temperature of 101 oF, and an erythematous lump measuring 5 cm on her abdomen at the insulin injection site. The lump was fluctuant and tender to touch. She was diagnosed with abdominal wall abscess and underwent I&D. She was initially treated with Clindamycin and Augmentin. The swab culture showed positive Eikenella corrodens, and sensitive to Augmentin. With 2 weeks oral Augmentin treatment, she was cured. Interestingly, Eikenella corrodens is a normal oral flora bacterium, and does not exist on skin. How did an oral bacterium cause abdominal wall infection? She injects Humulin with a syringe and needle twice daily and never reuses them. However, after questioning, she reported that she often licked the needle before injection, which we considered the cause. Discussion: Eikenella corrodens, a fastidious, slow-growing, gram-negative, facultative anaerobic bacillus, may be encountered in wounds exposed to human saliva, especially human bites. Dozens of cases were reported of Eikenella corrodens infection in intravenous drug abusers, because of licking the needles as a “cleaning step” when sharing them. There has been a case reported on this topic titled “Needle licker’s osteomyelitis” (1). There are nearly 26 million diabetic patients in U.S. and 6 million patients are on insulin injection (2). Even though insulin injection related infections are rare, given the large population, this remains an issue that need to be properly addressed. There have only been case reports on infections related with insulin injection with uncommon bacteria. The prevalence of this issue remains to be studied, and patients education regards to sterile techniques should be emphasized. Reference: (1), Swisher LA, Roberts JR, Glynn MJ. Needle licker’s osteomyelitis. Am J Emerg Med. 1994 May;12(3):343-6. (2), 2011 National Diabetes Fact Sheet

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DUAL CHALLENGE: DIAGNOSIS AND TREATMENT OF COLON CANCER IN PREGNANCY

Introduction: Incidence of cancers during pregnancy ranges from 0.07-0.1%. Cancers of breast, cervix, ovary, lymphoma and melanoma are the most frequently seen neoplasms in pregnancy. Colorectal cancer (CRC) during pregnancy is very rare, constituting about 0.002% of all pregnancies. Case: This is a 38 years old Brazilian lady, G2P0001 at 21 weeks of gestation, presented to emergency room with severe abdominal pain for a day associated with nausea and vomiting. Prior to this, she had one episode of tarry stool which was attributed to internal hemorrhoid, and a 5 kg weight loss during the first trimester. She was diagnosed with UTI and was subsequently discharged home after intravenous hydration, pain control and ampicillin. She returned
a day later with persistent lower abdominal pain. MRI without gadolinium revealed prominent wall thickening in the sigmoid colon. A diagnosis of diverticulitis was then made and she was again discharged home on oral metronidazole therapy. At 28 weeks of gestation, she underwent an outpatient sigmoidoscopy, due to persistent moderate-to-severe abdominal pain and bloody stools. A large tumor was found in the sigmoid colon with biopsy revealing of adenocarcinoma. CEA was 72.9 ng/ml. Abdominal ultrasound did not reveal any evidence of liver metastasis. Patient underwent scheduled vaginal delivery at 32nd week of gestation. Subsequent colonoscopy confirmed adenocarcinoma and ruled out metachronous tumors. CAT scan staging revealed local invasion to the mesentery, and retroperitoneal lymphadenopathy. Robotic surgical resection and staging further confirmed the local involvement of the small intestine but no obvious liver metastases. Pathology revealed stage 3C, poorly differentiated mucinous adenocarcinoma of the sigmoid colon. Patient is currently receiving her third cycle of FOXFOL chemotherapy. Discussion: Of all reported CRC cases during pregnancy (less than 300 total cases in last 30 years), majority were in their advanced stage (stage 3 and above), with poor prognosis and a high 5 year mortality rate. This is mainly due to the signs and symptoms of CRC in pregnancy being non-specific, and often similar to the normal physiological changes that occur during pregnancy. Due to paucity of cases, no generally accepted guidelines are available for diagnosing and managing CRC during pregnancy. Reference: Saif MW. Management of colorectal cancer in pregnancy: a multimodality approach. Clinical Colorectal Cancer 2005; 5: 247-256. Longo SA., Moore RC., Canzoneri BJ., and Robichaux A. Gastrointestinal conditions during pregnancy. Clin Colon Rectal Surg 2010; 23:80-89

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A COLD CONFUSED EDEMATICOUS ELDERLY WOMAN

Introduction: Myxedema coma (MC), the extreme manifestation of hypothyroidism, is an uncommon but potentially lethal condition. Patients with hypothyroidism may exhibit a number of physiologic alterations to compensate for the lack of thyroid hormone. If these homeostatic mechanisms are overwhelmed by factors such as infection, medication, the patient may decompensate into MC. I am presenting a case of MC resulting from multiple drugs and alcohol interaction. Case Description: A 54 year old lady with a history of gout, hypothyroidism and alcoholism was found by her family unresponsive on the couch. She had been well up until 6 hours prior to admission. Her medications included indomethacin(I), colchicine(C), synthroid and benzodiazepine. She had history of alcohol abuse according to the family. Vitals on admission: respiratory rate: 8/min, blood pressure: 81/65mmHg, temperature: 96.9 F, pulse rate: 70/min. Pertinent physical examination revealed she had periorbital edema and non pitting edema of lower extremities. Laboratory studies show:

| WBC-12.1 | HB9.4 | Platelet126 |
| Creatinine6.96/Blood urea nitrogen 51 | K7.6 | Chloride101 |
| Bicarbonate18 | Anion gap 16 | PH 7.13/PCO2 58 |
| Creatinine kinase >50.000 | Lactic acid 13 | INR0.97 |
| TSH 151, Free T4 <0.07, T3 0.2 | AST 68,ALT 98 | Opiates positive in the urine |

Patient was admitted to ICU where she was intubated for respiratory distress and was started on Continuous Veno-Venous Dialysis. Diagnosis of MC was made based on TSH. There were no signs of infection. The next day patient was started on intravenous steroid and synthroid. She clinically improved and successfully extubated within 7 days. Discussion: MC in the elderly, although uncommon, is frequently overlooked and has a high mortality rate. Signs and symptoms are varied and are often insidious(3).The known precipitating causes of MC were ruled out in this patient, which left her medications the combination of (I) and (C), opiates, alcohol and non compliance with synthroid as the probable cause. Conclusion: Due to the widespread use of multiple medications, clinicians should be reminded of the rare, yet life-threatening, occurrence of MC when treating patients, especially with combination therapies(1). Prompt recognition and emergency medical treatment are essential for a successful outcome. Prevention requires screening of elderly patients at risk for hypothyroidism and assuring compliance with thyroid hormone replacement therapy(2). References: 1.Church CO , Myxedema Coma  Associated with Combination Aripiprazole and Sertraline Therapy The Annals of Pharmacotherapy December 1, 2009: 43: 2113-2116 2. Olsen CG Myxedema coma in the elderly- J Am Board Fam Pract - 01-SEP-1995; 8(5); 376-83 3. Rodríguez I, et al. Factors associated with mortality of patients with myxedema coma: prospective study in 11 cases treated in a single institution. J Endocrinol 2004: 180;347
NEUROLEPTIC MALIGNANT SYNDROME IN A 60 YEAR OLD MALE WITH SCHIZOAFFECTIVE DISORDER

Neuroleptic malignant syndrome in a 60 year old male with Schizoaffective disorder S.Vulava MD;N.Srivastava MD Saint Peter’s University Hospital, New Brunswick, NJ. Introduction: Neuroleptic malignant syndrome (NMS) is an uncommon complication of therapy with major tranquilizers. Manifestations include pallor, diaphoresis, blood pressure fluctuation, tachycardia, and tachypneic hypoventilation, which may necessitate respirator support. Death often occurs, but full recovery can result with prompt recognition and proper management. Manifestations of NMS are attributed to dopamine receptor blockade in the striatum, increasing thermogenesis, and in the hypothalamus, impairing heat dissipation. Case: A 60 year old male patient with a history of hypertension, diabetes, hyperlipidemia, schizoaffective disorder was brought in by the EMS from an assisted living facility due to weakness and lethargy for 4 days. He presented with altered mental status, muscular rigidity, lethargy and mumbling speech. He had decreased urinary output, productive cough with yellowish sputum. Upon admission he was found to have a temp of 103.6. He had an elevated white count and a CPK of 1435 with an anion gap of 20. Prior to admission he was on treatment with Risperidone and Seroquel. He was begun treatment with empiric antibiotics. Despite this he continued to have elevated body temperature. A diagnosis of neuroleptic malignant syndrome was made and he was begun on treatment with Bromocriptine. Dantrolene sodium was initially given to control his muscular rigidity. Over the course of his stay in the hospital he began showing improvement in his sensorium, rigidity and was finally discharged to a psychiatric facility. Discussion: Neuroleptic malignant syndrome is a rare but potentially fatal reaction associated with neuroleptic drugs. It occurs in about 0.2% of patients treated with neuroleptics. Risk factors include previous episodes, dehydration, agitation, and the rate and route of neuroleptic administration. Standardized criteria for the diagnosis of NMS have been developed and emphasize the classic findings of hyperthermia, muscle rigidity, mental status changes, and autonomic dysfunction. The syndrome lasts 7 to 10 days in uncomplicated cases receiving oral neuroleptics. Treatment consists of early recognition, discontinuation of triggering drugs, management of fluid balance, temperature reduction, and monitoring for complications. Use of dopamine agonists or dantrolene or both should be considered and may be indicated in more severe, prolonged, or refractory cases. Electroconvulsive therapy has been used successfully in some cases and is particularly useful in the post-NMS patient. Neuroleptics may be safely reintroduced in the management of the majority of patients recovered from an NMS episode, although a significant risk of recurrence does exist, dependent in part on time elapsed since recovery and dose or potency of neuroleptics used. Data drawn from clinical observations supports the primary role of an acute reduction in brain dopamine activity in the development of NMS.

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REFRACTORY THROMBOTIC THROMBOCYTOPENIC PURPURA

INTRODUCTION Thrombotic Thrombocytopenic Purpura (TTP) is a rare thrombotic microangiopathic hemolytic anemia (MAHA) with a survival rate of 10% without plasmapheresis.1 Refractory TTP is failure to achieve complete remission (platelet count >150,000) after seven daily plasma exchanges.2 CASE A healthy 26 year old Hispanic male presented with headache, fever, altered mental status and diarrhea. Physical examination showed hyperthermia (102 F), generalized petechiae and obtundation without focal neurologic deficits. He had thrombocytopenia (59 x 103/mm3) and MAHA (Hgb of 5.8 g/dL, abundant schistocytes, elevated LDH: 4647 u/L, low haptoglobin, elevated reticulocytes, negative Coombs’ test). Creatinine was 0.9 mg/dL with minimal urine output. He was diagnosed with TTP, and started immediately on daily plasmapheresis (1 plasma volume exchange/day). His clinical course deteriorated throughout the first five sessions. Platelet count dropped further to 10 x 103/mm3. Urine output decreased and neurologic findings worsened with the development of hemiparesis. He continued to be febrile, even with empiric broad spectrum antibiotics.

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antibiotics. Blood, CSF, stool and urine cultures were negative. Bone marrow biopsy was normal. Plasmapheresis was then increased to 1.5 plasma volume exchanges/day, and methylprednisolone 125mg IV twice daily was started. The platelet count began to increase after the seventh exchange, and normalized (180,000 x 103/mm3) after the eleventh. A total of thirteen sessions were given. The patient had complete neurologic recovery. ADAMTS 13 (A Disintegrin and Metalloproteinase with Thrombospondin-like motif) activity was severely low at 3% (normal: >67%), confirming the diagnosis of TTP. Upon discharge, his platelets were 310 x 103/mm3, hemoglobin 10.9 g/dL, LDH 584 u/L. DISCUSSION The classic pentad (MAHA, thrombocytopenia, neurologic symptoms, renal insufficiency, fever) in TTP occurs in only 5% of cases. Plasmapheresis should be initiated within 24 hours, to replace ADAMTS13 and to remove its autoantibodies. An increased platelet count is expected after the second treatment, and complete remission in one week.1 There are no evidence-based therapeutic recommendations for refractory TTP, but increasing the volume or frequency of plasma exchange have been tried.2 Severe ADAMTS13 deficiency defines a subgroup of patients who benefit from the addition of corticosteroid therapy.1 CONCLUSION The clinical diagnosis of TTP is critical for the prompt initiation of plasmapheresis. There should be a high index of suspicion for TTP in patients with MAHA and thrombocytopenia, since the condition is fatal without treatment. Refractoriness to therapy may be encountered. But if the clinical basis for diagnosis is strong, escalation of treatment is required. REFERENCES 1 George, J. How I Treat Patients with TTP: 2010. Blood Journal. 2010. 116(20) : 4060-4069 2 Yarranton, H. An Update on the Pathogenesis and Management of Acquired TTP. Curr Opin Neurol. 2003. 16:367-373

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MEMBRANOUS GLOMERULOPATHY WITH ANCA ASSOCIATED NECROTIZING AND CRESCENTIC GLOMERULONEPHRITIS

INTRODUCTION: MN , the most common cause of nephrotic syndrome in Caucasian adults is often idiopathic(1). The natural history is variable with approximately one third of patients progressing to end stage renal disease within 10 years. In contrast pauci- immune NCGN is an aggressive disease with one year mortality rate of up to 80% in the absence of immunosuppressive therapy. CASE: A 57 year old female with history of hypertension and coronary artery disease underwent cholecystectomy in june 2011 and developed post op hypotension due to blood loss which resulted in acute kidney injury with a creatinine of 2.03. She was treated with hydration and blood transfusions. Her creatinine improved to 1.4, but she developed significant proteinuria of 3 grams which worsened gradually. She also had worsening bilateral pedal edema and underwent renal biopsy one year later. Her workup was negative for ANA, anti DNA antibody, C3,C4, hepatitis B surface antigen, HCV,HIV ,ANCA, anti GBM antibody and for monoclonal protein. Renal biopsy showed stage 2 to3 membranous glomerulopathy and the additional finding of a single glomerulus with segmental fibrinoid necrosis suggesting superimposed focal necrotizing glomerulonephritis of the pauci immune type. Treatment for the ANCA portion of biopsy was held as proteinurea decreased to 1.9 grams following biopsy, creatinine remained stable and without hematuria. The blood pressure was well controlled by valsartan. DISCUSSION: The occurrence of ANCA associated NCGN and primary MN in the same patient is rare with only 10 reported cases (5). They include seven men and three women with a mean age of 61 years. In one patient MN preceeded the development of ANCA associated NCGN(7). In the remaining nine patients they were diagnosed simultaneously on biopsy. Evidence of systemic vasculitis was present in five out of the ten patients. Among ten reported cases induction therapy consisted of prednisone and cyclosporine, prednisone and azathoprine and prednisone alone. After a mean follow up of 38 months four patients had complete renal recovery and three had partial recovery with decreased creatinine and one had stable renal function and two progressed to end stage renal disease. Conclusion: The dual diagnosis of MN and ANCA associated NCGN should be considered in patients who present with rapidly progressive glomerulonephritis and nephrotic syndrome. The finding of MN is associated with greater degree of proteinuria and found to have a negative impact on the already poor prognosis of this condition. References: 1. Nasr SH et al:Membranous glomerulopathy with ANCA associated Necrotizing and Crescentic glomerulonephritis. J Am Soc nephrol4: 299-308,2009 2. Gaber LW, Wall BM, Cooke CR: Coexistence of ANCA associated glomerulonephritis and membranous glomerulopathy, clinical nephrol 52: 253-255,1999
A CASE OF GASTROCNEMIUS PYOMYOSITIS

Introduction: Pyomyositis is primary infection of skeletal muscle with abscess formation, seen in immuno-compromised individuals. Staphylococcus aureus is the usual culprit. MRI is most sensitive non-invasive diagnostic technique, while muscle histology with culture is most accurate and gold standard for diagnosis.

Case Report: 37 year-old man from Mexico presented to ER with complaints of pain and swelling in left leg for 3 days following muscle pull with strenuous activity. He had past medical history of poorly controlled diabetes mellitus, urinary tract infection and oral candidiasis. He was hospitalized 1 year ago with endocarditis with right renal vein thrombosis with multiple renal and pulmonary septic emboli and uveitis. On examination he was found to have erythematous, swollen and tender left leg with multiple small ulcerations over the shin and a swollen left knee joint. He also had oral and genital candidiasis with poor oral hygiene and dentation. Left leg Doppler study ruled out deep venous thrombosis. Subsequently, he underwent knee arthrocentesis for suspected septic arthritis. Synovial fluid was cloudy with few cells on microscopy and culture showed no growth. Considering initial differential diagnosis of Baker cyst v/s cellulitis, he underwent leg x-ray that revealed soft tissue swelling in calf region prompting MRI. MRI revealed left leg gastrocnemius enlargement with edema of soleus muscle. Incision and drainage showed bloody purulent discharge. Culture grew Staphylococcus aureus. During the course of hospital stay, he had four sets of blood cultures that showed no growth.

Discussion: Pyomyositis is usually seen in tropical areas but there has been increasing number of cases reported in US over past few decades. The diagnosis is often difficult as most of the patients are immuno-compromised and pose a wide differential. Early diagnosis, initiation of appropriate antibiotic therapy and drainage of the pus lead to favorable outcomes.


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MEGA AORTA SYNDROME AS A CAUSE OF BRADYCARDIA?

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Introduction: Mega aorta syndrome (MAS) defined by aortic diameter of > 6cm, is a slow progressive disease state that presents itself over a number of years [1]. Etiology can be from infectious, autoimmune, to Marfan’s syndrome. Case: This is an 89 y/o female with a history of idiopathic pulmonary fibrosis, thoracic aortic aneurysm found incidentally on CT chest in 2009, who came with three months of intermittent and progressive episodes of lightheadedness. In the first episode, she was sitting upright in a chair when she felt lightheaded as if she was about to faint. In three months, the frequency of the episodes increased from once a month to 3-4 times a week. Each episode had the same presentation and duration, except for the increase in frequency which prompted the patient to seek medical attention. While she was at her primary care doctor’s office, she became bradycardic (40’s) and hypotensive (SBP 70’s) and was brought to the emergency department. There was no change in her medication, no loss of consciousness or loss of bowel or bladder function during the episodes. In the ED she had normal vital signs, but then suddenly had another episode of dizziness and vitals signs showed a heart rate of 40’s and a SBP of 60’s. The patient’s cardiac monitor showed that she was in sinus bradycardia. The patient was admitted to the intensive care unit for symptomatic bradycardia. A retrospective review of the patients serial CT scans of the chest showed that over the last year, her aneurysm has been growing: • Ascending aorta: 5.2-2/2009 5.5-4/2011 5.5-11/2011 5.5 / 1-12 6.5 9/28/2012 • Descending aorta: 3.4-2/2009 not reported not reported not reported 4.5 9/28/2012 Discussion: The patient, never having symptoms of dizziness prior to her current presentation, as well as evidence of an expanding aneurysm with no change in medication recently, it is likely that the expanding aneurysm as well as concurrent beta blockade contributed to her episodes of symptomatic bradycardia. Conclusion: MAS patients are symptomatic prior to major catastrophic presentation [1]. The maximum wall tension for thoracic aorta is 6 cm and beyond that risk for rupture or dissection is high [1,2]. It is likely that this patient’s episode of dizziness

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VISUOPERCEPTUAL AND CALCULATION DEFICITS IN A 73 YEAR-OLD

Introduction Posterior Cortical Atrophy (PCA) is a rare form of presenile dementia characterized by cortical visual dysfunction and preserved memory, insight and judgment until later in the course of the disease. The most common pathologic cause is Alzheimer’s disease (AD) and is considered an atypical variant of the disease. Other functions like writing, calculation and recognition of common objects are usually affected. Imaging studies will suggest more pronounced atrophy in the occipital lobes compared to other areas of the brain. Case A 73 year-old male retired banker is seen in the office because of progressive visual impairment and calculation deficits. He first noticed difficulty with calculation and inability to play the piano as before. As time went by he noticed increasing difficulty in reading the newspaper, locating items and getting dressed, as he was unable to locate the sleeves of his shirts. He was seen by an ophthalmologist who could not find any ocular abnormalities. No change in personality was noted, although because of these problems he lost interest in reading, stopped driving and developed anhedonia with a subsequent depressed mood. He found frustration when simple things like bathing and shaving would take him hours to perform. Evaluation by neurologist then was recommended. On physical examination patient was alert, comfortable and cooperative. Neurological examination disclosed a slow speech and lack of orientation to time. Showed impaired short-term memory and dysnomia. Transcortical sensory aphasia was present as well as poor calculation and visuospatial construction. Funduscopic examination, cranial nerves and visual acuity were normal. MRI of the brain showed diffuse atrophy, especially in the occipito-temporal areas. Patient was then diagnosed with PCA and was started on donepezil trial with some improvement. His condition deteriorated over the course of a few years rendering him incapable to perform his daily activities and permanently disabled. Discussion Being a rare form of AD, PCA may be difficult to recognize and usually misdiagnosed. It poses a challenge for current researchers and patients, since no cure exists for this condition. Early diagnosis of the condition may provide the opportunity to maintain some quality of life since this patients often benefit from physical and occupational therapy and medications, like cholinesterase inhibitors and antidepressants, which provide some improvement in daily functioning. This disorder remains a challenge to both physicians and patients but ongoing studies about neuropathology and behavior of the disease may provide future treatments. References D. F. Tangwai, MDCM; N.R. Graff-Radford, MBBCh. Clinical, genetic, and neuropathologic characteristics of posterior cortical atrophy. Neurology. 2004;63:1168-1174.

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THE SEARCH FOR ANCHOVY PASTE

Introduction: Amebic liver abscesses common worldwide, are rare in the United States, mostly in the western and southern states and in Hispanic populations. A high index of suspicion can lead to a prompt diagnosis and avoid unnecessary diagnostic tests. Case: A 41-year-old male from Sierra Leone presented to the emergency room because of an episode of sharp right upper quadrant abdominal pain radiating to right shoulder. He had been experiencing abdominal pain and fever intermittently for two weeks. He had immigrated to the U.S. 18 months prior to admission. Upon physical examination, the patient was comfortable in bed. He was afebrile. His abdomen was soft; right upper quadrant tenderness was appreciated but no masses or organomegaly. CBC showed hemoglobin 10.6 g/dL, hematocrit 31.5%, WBC 16.5 10 3/mm3 and platelet count was 522 10 3/mm3. He had neutrophilia but no eosinophilia. Liver transaminases, serum alkaline phosphatase and total bilirubin levels were within normal limits. An ultrasound of the right upper quadrant revealed a solitary liver abscess in the right hepatic lobe. He was admitted and underwent an IR-guided needle aspiration of the abscess. Moderate polymorphonuclear lymphocytes but no organisms were seen in the aspirate. Subsequent testing of the aspirate was positive for E. Histolytica IgG. He was started on a course of oral metronidazole 500 mg three times a day and discharged home. Discussion: Amebic liver abscesses are rare in the United States
and occur almost exclusively in immigrants. This patient had the classic signs and symptoms for an amebic liver abscess, presenting with fever and abdominal pain. The right shoulder pain correlated with the location of the abscess. Abscesses may contain a large necrotic center resembling anchovy paste. Diagnosis is based on detection of one or more space-occupying lesions in the liver by ultrasound or CT and a positive serologic test for antibodies to E. histolytica antigens. Conclusion: In diverse populations, it is important to consider amebiasis as a differential diagnosis for a liver abscess. Congly SE, et al. Amoebic liver abscess in USA: a population-based study of incidence, temporal trends and mortality. Liver Int. 2011; 31: 1191-8.

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A CASE OF INDETERMINATE COLITIS

Introduction In 10% of cases of Inflammatory Bowel Disease (IBD) presenting with colitis, no distinction can be made between Crohn’s disease (CD) and Ulcerative Colitis (UC). This population is diagnosed with indeterminate colitis with manifestations of both CD and UC. Case Description A 42 year old Caucasian lady presented to the emergency department with a 2 week history of progressively worsening bloody diarrhea, tenesmus, severe, diffuse, crampy abdominal pain, generalized fatigue, anorexia, weight loss, subjective fevers, and night sweats. She reported having similar symptoms 19 years ago, with concurrent oral ulcers, conjunctival redness, and joint aches. She was diagnosed with Crohn’s disease, treated with 6 mercaptopurine, sulfasalazine, and prednisone and went into remission for 19 years. On hospitalization, physical examination showed hyperactive bowel sounds, generalized abdominal tenderness to moderate palpation, and moderately severe rectal tenderness. Laboratory findings include leukocytosis, iron deficiency anemia, ASCA+/pANCA-. CT scan of the abdomen showed edema in the transverse and descending colon consistent with colitis, colonoscopy revealed pancolitis without skipped areas, and colonic biopsy was suggestive of ulcerative colitis. Despite having past and current symptoms suggestive of Crohn’s disease, her subsequent development of worsening bloody diarrhea and her radiographic findings were indicative of features of UC superimposing on CD, suggesting the diagnosis of Indeterminate Colitis. During hospitalization, treatment was initiated with a course of Mescalamine, 6mercaptopurine, Solumedrol, Percocet for pain control, Iron, Vitamin B12, Folic acid, and she was kept on a lactose-free diet. However, approximately two weeks after commencement of therapy, her symptoms of bloody diarrhea, tenesmus, and crampy abdominal pain persisted. Having failed initial therapy, she was started on intravenous Infliximab. With biologic therapy, her symptoms resolved and she went into remission. Discussion This case illustrates an Inflammatory bowel disease previously thought to be entirely Crohn’s disease, but subsequently exhibiting additional features indicative of Ulcerative Colitis – suggesting that she is one of the 10% of IBD cases with Indeterminate Colitis. Studies have shown that in patients with IBD, serologic markers of ASCA+/pANCA- correlated with CD in 8 of 10 patients with Indeterminate Colitis. There are still no widely accepted histologic criteria or findings for the diagnosis of Indeterminate Colitis; it remains a diagnosis of exclusion. Medical therapy for Indeterminate Colitis is the same as that for UC and CD. References: 1. E.Telakis, E.Tsironi. Indeterminate Colitis – definition, diagnosis, characteristics, and management. Annals of gastroenterology 2008; 21 (3): 173-179 2. Joossens S, Reinisch W, Vermeire S, Sendid B, Poulain D, Peeters M, Geboes K, Bossuyt X, Vandewalle P, Oberhuber G, Vogelsang H, Rutgeerts P, Colombel JF. The Value of serologic markers in indeterminate colitis: a prospective follow-up study. Gastroenterology. 2002 May;122(5):1242-7
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DRUG INDUCED MENINGITIS IN PREGNANCY

TITLE: Drug induced meningitis in pregnancy. Authors: Dr. Ajit Indavarapu MD, Saint Peter’s University Hospital /Drexel University Hospital. Introduction: Meningitis can be caused by infectious or non infectious agents. Drug induced meningitis is one of the under recognized causes of aseptic meningitis. Non steroidal anti inflammatory agents, antibiotics, anti neoplastic drugs and IV immunoglobulins are the frequent causes of aseptic meningitis(1). This is a case of drug induced meningitis caused by antibiotics in pregnancy. Case: A 30 yr old female G2P1 in 25th week of pregnancy presented to the emergency department with fever, headache, nausea and vomiting of two days duration. Three days before the ER visit she was given nitrofurantoin for suspected UTI and amoxicillin for suspected sinusitis. She had a constant headache, fever up to 101F associated with chills and rigors, nausea with vomiting and photophobia. On examination she had neck stiffness with decreased range of movements. Kerning”s sign was negative. Lymphocyte count was 13.2*10^5/L(80% neutrophils), CT Head was negative. Nitrofurantoin and amoxicillin were discontinued on admission and was empirically started on vancomycin, ceftriaxone and ampicillin(for listeria). Lumbar puncture showed a WBC count of 114 cells/cumm with 74% polymorphs, glucose and protein were within normal range and Gram stain was negative. Blood and urine cultures were negative. All the bacterial, viral and fungal causes of meningitis were negative. Vancomycin, ceftriaxone and ampicillin were discontinued on the second day of admission based on CSF analysis results. She improved rapidly over a period of two days and was discharged home on the third day. Based on CSF analysis consistent with meningitis, the temporal relationship between ingestion of a drug and onset of clinical symptoms, exclusion of other causes of meningitis and improvement of symptoms after discontinuation of the drugs, a diagnosis of drug induced meningitis was made. Discussion: Drug induced meningitis can mimic infective meningitis. This case is an indication that drug induced meningitis should be included in differential diagnosis of aseptic meningitis(2). It is a diagnosis of exclusion and patients who are suspected of drug induced meningitis can be treated by third generation cephalosporins, which are known to cause meninges only exceptionally(3) and they would be active against the most frequent organisms in a healthy individual until the appropriate CSF studies are available. The diagnosis of drug induced meningitis can be made depending on the relation of onset of symptoms with the drug use, quick resolution of symptoms after discontinuation of the drug(2). References: 1. Chaudhry HJ, Drug-induced aseptic meningitis. Diagnosis leads to quick resolution. Postgrad Med. 1991;90:65-70. 2. Shahien R, Amoxicillin-induced aseptic meningoencephalitis. Int J Gen Med. 2010;3:157-62. 3. Watson JDG, Aseptic meningitis associated with high dose intravenous immunoglobulin therapy. J Neurol Neurosurg Psychiatry.1991;54:275-276.

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UNUSUAL CAUSE OF PERITONITIS IN A YOUNG PERITONEAL DIALYSIS PATIENT

Introduction Gram positive cocci (Staphylococcus) are the most common cause of peritonitis in patients undergoing peritoneal dialysis, whereas Gram negative and anaerobes comprise a relatively smaller percentage. We present a case report describing a 21 year old patient on Continuous Cyclic Peritoneal Dialysis (CCPD) with peritonitis due to Neisseria Sicca. The world literature on Neisseria Sicca peritonitis comprises of 3 reported cases in Europe and 3 in the United States[1]. Case Report A 21 year old Hispanic male diagnosed recently with ESRD from unclear cause, was ultimately started on CCPD. Further, the patient had two hospital admissions for abdominal pain, once due to intussusception and at another occasion due to Clostridium difficile infection. 8 months after dialysis initiation, the patient presented again with nausea, vomiting and abdominal pain for 1 day. A cloudy peritoneal fluid was aspirated for gram stain and culture. The patient was started empirically on intraperitoneal (IP) Vancomycin. Peritoneal fluid analysis showed a WBC count of 15900/ml and 96% polymorphonucleocytes. The initial gram stain was reported as both gram positive and gram negative cocci and IP Imipenem/Cilastatin was added to the antibiotic regimen. However, the patient developed seizures and Imipenem/Cilastatin was changed to IP Amikacin. Subsequently, the gram stain result was reinterpreted as gram negative cocci and peritoneal fluid cultures grew N.Sicca only. Vancomycin was stopped and IP Amikacin was continued for a total of 7 days. The patient became asymptomatic on Day 2 of
Amikacin treatment and repeat peritoneal cultures were negative. Discussion N. Sicca rarely causes invasive diseases and is considered a commensal organism in the oropharynx[1]. The first documented case of N. Sicca peritonitis was reported in 1994[2]. Initially laboratory had misinterpreted the N. Sicca gram stain as both gram positive and gram negative. A similar problem was described by Novak et al in their case report. Hence, the significance of our case report lies in enhancing the evidence that a variably staining gram stain of N. Sicca could be misread initially as gram-positive cocci by the technologist (especially if gram-positive cocci are suspected), and this could delay adequate treatment. This case report also shows that once identified accurately, N. Sicca peritonitis can be successfully treated with IP Amikacin without removal of PD catheter and preventing premature failure of CCPD. References 1. Novak DJ, Bashir K et al: Neisseria Sicca/Subflava: Continuous Ambulatory Peritoneal Dialysis-Assocaiated Peritonitis. LABMED.38:363-364, 2007 2. Alicia M. Neu, Barbara Case et al: Neisseria sicca peritonitis in a patient maintained on chronic peritoneal dialysis. Pediatr Nephrol. 8:601-602, 1994

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**ALCOHOL-INDUCED FULMINANT HEPATITIS IN A NON-ALCOHOLIC PATIENT**

Introduction: Frequent causes of acute hepatic injury, include drug intoxications such as acetaminophen, viral hepatitis, mushroom poisoning, autoimmune hepatitis or shock liver. We report a case of a previously healthy young man who presented with extremely high hepatic enzymes after heavy alcohol consumption. Case: This is a 35 year old male without any significant medical history who presented with severe dehydration secondary to non-bloody diarrhea and non-bloody, non-bilious emesis three days prior to admission. He also complained of a diffuse, non-radiating abdominal pain. One day prior, the patient consumed large amounts of alcohol. He is not alcohol dependant but he occasionally binge drinks. He did not consume any herbas, mushrooms, or any other medications at home except for acetaminophen occasionally. On presentation his vital signs were normal; he had dry mucus membranes and mild abdominal tenderness on physical examination. Initial laboratory assessment revealed extremely high liver enzymes with the ALT being >10000U/L and the AST > 7500U/L; his total bilirubin was 4.1, Albumin was 2.6 and INR was 1.32. At that time creatinine was 6.18 and BUN was 53. Imaging studies of the abdomen showed fatty liver changes and perinephric inflammatory fluid bilaterally. His Maddrey’s discriminant function score at the time of presentation was 18, MELD score was 32 and the possibility of liver transplantation and transfer to a specialized transplant center were considered. It was decided to follow the neurologic status of the patient and the liver enzymes in the ICU and treat empirically with volume resuscitation. Patient never showed any signs or symptoms of hepatic encephalopathy. His liver enzymes decreased to 44 and 509 for AST and ALT respectively within the next eight days. His creatinine reached a peak of 11.72 secondary to acute tubular necrosis and decreased to 6.99 on day of discharge. The work up for this fulminant hepatitis was negative for autoimmune diseases and any of the common viral causes of hepatic failure; acetaminophen levels were less than 10 and alcohol was not detected. Less common infectious causes like leptospirosis were also considered but were negative. Discussion: Acute alcoholism is not a well established cause of acute liver injury especially without a coexisting liver disease. The most interesting fact about this particular case is the remarkable discrepancy between the patient’s clinical picture with his laboratory findings and his immediate response to volume resuscitation. References Patton, H., M. Misel, and R.G. Gish, Acute liver failure in adults: an evidence-based management protocol for clinicians. Gastroenterol Hepatol (N Y), 2012. 8:161-212

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**NUMBERS CAN BE DECEIVING: MASSIVE PROTEINURIA AT 22 WEEKS OF GESTATION**

Introduction: Pre-eclampsia is hypertension and proteinuria of 300mg/24 hours after 20 weeks of pregnancy. However, an underlying renal disorder should be considered in the presence of proteinuria in the nephrotic range of more than 3.5grams/24 hours. Hence, a kidney biopsy is indicated. Case: Patient is a 41 year old G2P1 African American female with no known medical history at 22 weeks of pregnancy presenting with the chief complaint of worsening leg edema and facial swelling for 1 week. Vital signs blood pressure 150/90, current weight of 111kg, pre-pregnancy weight 102kg. She had bilateral leg edema, the rest of the physical
findings were normal. Laboratory tests showed normal CBC, glucose, BUN, creatinine and bicarbonate. Albumin was low at 2.5. Urinalysis showed hematuria and proteinuria. 24-hour urine revealed significant proteinuria of 31 grams/24 hours. Sample was calculated to be adequate based on her pre-pregnancy weight. LDL and TGC were 208 and 205, respectively. Rapid HIV test, ANA, anti-dsDNA, C3, C4, anti-HCV, HBsAg were negative. An ultrasound-guided biopsy was performed to rule out a primary renal disease. Biopsy showed endotheliosis, mesangiolysis and glomerular basement membrane duplication suggestive of pre-eclampsia. Patient subsequently managed with bed rest, 2 grams sodium diet, labetalol and diltiazem as anti-hypertensive drugs, which well-controlled her blood pressure. A neonatal ICU consult was done in the event pre-term termination of pregnancy was inevitable. CBC, BUN/Crea, AST/ALT were closely monitored for possible development of HELLP and acute renal failure. Discussion: Severe pre-eclampsia is sometimes the cause of nephrotic syndrome in pregnancy. An underlying renal disease causing nephrotic syndrome during pregnancy is rare. If there is suspicion of de novo renal disease based on history, physical, presence of massive proteinuria, as in this case, a renal biopsy is indicated.1 Proteinuria, particularly above 900mg/mmol (9grams/24hours) is a proven poor prognostic indicator for fetal and maternal outcomes, such as higher blood pressure, preterm delivery, abruptio placenta, HELLP and eclampsia.2 Proteinuria and hypertension is expected to resolve 6 weeks post-partum.2 References: Chan P, Brown M, Simpson J, Davis G. Proteinuria in pre-eclampsia: how much matters? BJOG: an International Journal of Obstetrics and Gynaecology. 2005; 112: 280-285 2. Thangaratinam S, Coomarasamy A, Fidelma O'Mahony, Sharp S, Zamora J, Khan K, Ismail K. Estimation of proteinuria as a predictor of complications of pre-eclampsia: a systemic review. BMC Med; 2009; 7: 10

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POSTPARTUM CONJUGATED HYPERBILIRUBINEMIA, A DIAGNOSTIC CHALLENGE IN SICKLE CELL DISEASE PATIENTS.

Introduction: This is a case of conjugated hyperbilirubinemia in a postpartum lady with sickle cell disease. Her causative factors for this were sickle cell crisis secondary to dehydration, postsurgical cholestasis or antibiotics induced cholestatic jaundice. Case Report: This is a 29 year old African American female who developed new onset jaundice 2 days after cesarean section delivery at 39th week of gestation. After delivery, she developed yellow eyes, intermittent fever (102.4 F), chills, nausea and episodes of watery, non-bloody, bilious vomiting after eating meals, chest pain which was worse on deep breathing and right upper quadrant abdominal pain. She was diagnosed with sickle cell disease and had multiple blood transfusions. She has never smoked, does not drink alcohol and never used any drugs. Her medications are prenatal vitamins and folic acid. On examination she had scleral icterus and decreased breath sounds and dullness to percussion on right lower zone. She was treated with ceftriaxone and azithromycin for pneumonia. Her total bilirubin was 29 with direct bilirubin of 18.9, AST 107, ALT 71, and Alkaline Phosphatase 262. Her abdominal ultrasound showed gallbladder sludge and multiple gallstones in gall bladder, common bile duct was 2mm and no pericholecystic fluid. She was managed with intravenous hydration and oxygen. Her total bilirubin dropped to 13.1 and Alkaline Phosphatase to 187 on discharge. Discussion: Sickle cell disease patient are at increased risk of developing intrahepatic cholestasis due to intrasinusoidal sickling which may lead to ischemic injury to hepatocytes. This patient may have developed intrahepatic sickling after delivery which explains her elevation of alkaline phosphatase. In patients without liver disease, hyperbilirunemia are exclusively unconjugated and only uncommonly exceed levels of 4.5mg/dl. Unlike unconjugated bilirubin, conjugated bilirubin does not bind significantly to neural tissue and does not lead to kernicterus or other forms of toxicity. References: 1.Omata M, Johnson CS, Tong M, Tatter D. Pathological spectrum of liver diseases in sickle cell disease. Digestive Diseases and Sciences journal 1986; 31:247 2.Brunetta DM, Silva-Pinto AC, do Carmo Favarin de Macedo M, Bassi SC, Piccolo Feliciano JV, Ribeiro FB, Prado Bde P Jr, De Santis GC, de Lucena Angulo I, Covas DT. Intrahepatic cholestasis in sickle cell disease: a case report. Hindawi Publishing Corporation Volume 2011: 975731, 3 pages doi:10.1155/2011/975731
Introduction
Leukemoid reaction is a nonneoplastic process characterized by leukocytosis exceeding 50,000/mm³. It usually represents a reaction to systemic inflammation or infection with significant increase in early neutrophil precursors. However, it can be a paraneoplastic manifestation of underlying malignant tumors. Case report An 81 year old female presented to ER with complaints of fatigue for 6 weeks and 20 lbs weight loss. At the presentation her vital signs were stable and physical examination was unremarkable. Initial blood counts revealed leukocytosis of 50,200 with 83% of neutrophils, hemoglobin of 8.6 and normal platelet count. Peripheral smear did not reveal any premature forms. Bone marrow biopsy showed hypercellularity with granulocytic hyperplasia, flowcytometry showed polyclonal expansion of B-cells and T-cells with normal CD4 to CD8 ratio with no increase in CD34+ cells, consistent with leukemoid reaction. Serum AFP, CEA and CE19-9 were within normal limits. Contrast enhanced CT scan of abdomen and thorax revealed a mass in left lobe of liver, multiple bilateral pulmonary nodules, a mass lesion in the right lobe of thyroid, 2 masses in the midpole of right kidney and a posterior wall rectal mass. It was difficult to determine the primary source. Ultrasound guided biopsy of the hepatic mass showed malignant cells with poorly differentiated carcinoma. Immunohistochemical marker studies were performed and the tumor cells were positive for pankeratin, CK7 and p63; and negative for HMB-45, CD20, TTF-1, CEA, chromogranin, ER, PR, and Her2-neu. Diagnosis of poorly differentiated squamous cell carcinoma (SCC) was made. Patient opted not to pursue further aggressive work up. 3 months after the initial diagnosis she succumbed to the malignancy. Discussion Cancer of unknown primary site (CUP) accounts for 4 to 5 percent of all invasive cancers. SCC comprises approximately 5 percent of CUPs. Paraneoplastic leukemoid reaction can be caused by increased production of growth factors or cytokines by the neoplastic cells. Granulocyte-macrophage colony-stimulating factor (GM-CSF), granulocyte colony-stimulating factor (G-CSF), interleukin 3 (IL-3) and interleukin 6 (IL-6) have been implicated so far. It can also result from mechanical stimulation of the bone marrow secondary to bone metastasis. Paraneoplastic leukemoid reaction has been reported mainly in relation with lung, gastrointestinal, genitourinary and head and neck cancers. This case, to our knowledge, is the first case of leukemoid reaction in a patient with SCC with unknown primary source in the English literature. Due to limited literature, it is not possible to establish any cause-effect relationship or association between leukemoid reaction and SCC of unknown primary. References 1. Greco FA, Hainsworth JD. Introduction: Unknown primary cancer. Semin Oncol 2009; 36:6. 2. Hocking W, Goodman J, Golde D. Granulocytosis associated with tumor cell production of colony-stimulating activity. Blood. 1983;61:600–603.

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MASSIVE SUBCUTANEOUS EMPHYSEMA FROM ENDOTRACHEAL INTUBATION

Introduction: Tracheobronchial rupture is a rare complication of endotracheal intubation usually presenting as a tear in the posterior membranous wall of the trachea leading to pneumothorax, pneumomediastinum, pneumoperitoneum, or subcutaneous emphysema. Older women with short stature are at an increased risk. Management requires surgical intervention if severe but otherwise can be managed conservatively. Case: An 88-year-old female with COPD was found at home in respiratory distress requiring rapid intubation and transport to the emergency room by paramedics. Initial chest radiograph in the ER was significant for endotracheal tube projecting over right main stem bronchus with no other pathology. ET-tube was subsequently pulled back 1cm. Shortly thereafter, massive swelling and crepitus was noted over her anterior chest, shoulders, neck, and face. Follow-up radiograph revealed extensive subcutaneous emphysema and pneumomediastinum without pneumothorax. CT of the thorax showed a deformity in the distal membranous trachea which was suggestive of a tracheal laceration. Bronchoscopy revealed a laceration in the distal membranous trachea, which correlated with the CT findings. The tear was managed conservatively with broad-spectrum antibiotics, low tidal volume ventilation, and low PEEP. The ET-tube was repositioned below the tear into the right main-stem bronchus to allow for mechanical ventilation. Her condition improved and she was later extubated. Conclusion: Intubation causing tracheobronchial rupture is rare with a reported incidence...
of 0.005%. [1] Risk factors include COPD, chronic use of steroids, advanced age, and female sex all of which were present in our patient. Other factors include forced intubation attempts, introducers protruding past the tip of the ET tube, or over-inflation of the ET-cuff. Diagnosis is suspected when subcutaneous emphysema is evident on physical exam. This finding is protective as it alerts the provider of an underlying issue. Chest radiograph will likely show no pneumothorax but may reveal pneumomediastinum and subcutaneous emphysema. CT may show a defect in posterior trachea. Definitive diagnosis is made through bronchoscopic visualization. [2,3] All patients should receive broad-spectrum antibiotics. If severe, lacerations are managed surgically. Conservative management is considered if subcutaneous emphysema does not worsen with ventilation, or if the ET tube can be placed distal to the laceration. Ventilation management should target the underlying pathology rather than the laceration however low tidal volumes and low PEEP should be used to avoid further injury. Double-lumen endotracheal tubes are used in patients with distal lacerations. References: 1. Borasio P, Ardissone F, Chiampo G. Postintubation tracheal rupture. A report on ten cases. Eur J Cardiothorac Surg 1997; 12: 98100. 2. Conti M, Marie P, Marquette CH, et al. Management of postintubation tracheobronchial ruptures. CHEST 2006;130: 412-8. 3. Jougon J, Ballester M, Choukroun E, Dubrez J, Reboul G, Velly JF. Conservative treatment for postintubation tracheobronchial rupture. Ann Thorac Surg 2000; 69: 21620.

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CORONARY SUBCLAVIAN STEAL SYNDROME: CASE REPORT

Introduction: Coronary subclavian steal syndrome (CSSS) with retrograde blood flow in the left internal mammary-coronary bypass graft is a rare complication but a potential risk after cardiac surgery. Here we present a case of CSSS. Case: A 73-year-old man with history of hypertension, type 2 diabetes, hyperlipidemia, coronary arterial disease status post coronary bypass graft (CABG) 5 years ago presented with unstable angina. He underwent cardiac catheterization which showed 80% stenosis in the proximal portion of the left subclavian artery with the retrograde blood flow in the left internal mammary coronary bypass. This could have been the cause of insufficiency in coronary blood flow and ischemia of the myocardial muscle. Diagnosis of CSSS was made. He finally underwent implantation of a subclavian stent which resolved his condition. The patient remained free of any symptoms 2 years after this procedure. Discussion: CSSS is defined as a reversal of flow in patients who have undergone prior CABG utilizing the internal mammary artery (IMA), leading to myocardial ischemia and recurrent angina. This is typically caused by proximal subclavian artery stenosis which may become progressive after CABG and cause significant impairment of forward flow across lesion resulting in reversal of flow in IMA and therefore becoming symptomatic(1). The incidence of a symptomatic CSSS is low as 0.44%(2). The dominant symptoms are angina pectoris, arm ischemia and vertebrobasilar insufficiency. Proximal aortic arch and direct subclavian artery arteriography are the gold standard for diagnosing SCCC. Alternative diagnostic procedures include doppler, ultrasound, CT, or MRI. Treatment of choice is endovascular intervention including balloon dilation and stent placement. Further treatment option is surgical repair which may include carotid-subclavian bypass, aortic-subclavian bypass, axillary-axillary bypass, or transposition of the subclavian artery. Conclusion: Identification of a significant subclavian artery stenosis prior to CABG can prevent CSSS. Those patients with a high-grade subclavian artery stenosis should be treated (percutaneously or surgically) prior to CABG(3). Reference: 1. Takach T, Reul G, Cooley D. Myocardial thievery: the coronary-subclavian steal syndrome. Ann Thora Surg.2006; 81(1): 386–392. 2. Rossum AC, Steel SR, Hartshorne MF. Evaluation of coronary subclavian steal syndrome using sestamibi imaging and duplex scanning with observed vertebral subclavian steal. Clinical Cardiology. 2000;23(3):226–229. 3. Hwang HY, Kim JH, Lee W, Park JH, Kim KB. Left subclavian artery stenosis in coronary artery bypass: prevalence and revascularization strategies. Ann Thorac Surg. 2010;89(4):1146.
HEPATIC INFARCTION IN ANTIPHOSPHOLIPID AND HELLP SYNDROME

Hepatic Infarction in Antiphospholipid and HELLP Syndrome L. Centino MD, A. Bukhari MD Saint Peter’s University Hospital/Drexel University College of Medicine, New Brunswick, NJ INTRODUCTION Hepatic infarction is considered a rare complication of antiphospholipid syndrome (APS) during pregnancy. However, when these patients present with HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets) Syndrome and right upper quadrant (RUQ) pain, hepatic infarction becomes the primary diagnosis. CASE A 29-year-old 16 weeks pregnant (G2P0) Indian female with primary APS, positive anticardiolipin antibody and lupus anticoagulant presented with three days of epigastric and RUQ pain. She was on Lovenox 40 mg once daily (OD) since her 7th week of pregnancy for prophylaxis against thrombosis. Physical examination found her to be afebrile, HR 115/minute and BP 180/90 mmHg. Abdominal examination revealed epigastric and RUQ tenderness. Laboratory findings were consistent with HELLP Syndrome – hemoglobin 11.0 g/dL, platelet 44000/mm3, LDH 2606 U/L and AST/ALT 1389/2095 U/L. Peripheral smear revealed schistocytes. Abdominal MRI showed multiple wedge-shaped areas with high T2 signal intensity in the liver consistent with infarction. Fetal sonogram revealed absence of heart tones consistent with fetal demise. Patient underwent dilation and evacuation. Her therapy included magnesium sulfate, prednisone, plasmapheresis, Lovenox 80 mg OD and low dose aspirin OD. Patient was then discharged home on same dose of Lovenox and Aspirin. DISCUSSION The dual blood supply to the liver makes hepatic infarction a rare entity, however it can occur as a complication of APS. The correlation between APS and hepatic infarction is not fully elucidated but thought to be due to the hypercoagulable state resulting in thrombosis of the vascular bed (1). Nearly one-third of pregnant women have hepatic infarction with APS complicated with HELLP syndrome or pre-eclampsia. (2). The thrombogenic state associated with HELLP may further contribute to hepatic injury. The diagnosis of hepatic infarction should be sought after in these individuals with RUQ pain using either doppler ultrasound, CT or MRI. Recommendations include termination of pregnancy and treatment with aspirin and anticoagulation. Anticoagulation with heparin was associated with hemorrhagic conversion or liver rupture occurs in about 1/40,000-250,000 cases (2,3). CONCLUSION Pregnant women with underlying APS, HELLP and RUQ tenderness should have an evaluation for hepatic infarction. Although anticoagulation is relatively safe, signs for hemorrhagic conversion should be monitored closely. REFERENCES 1. Uthman, I., et.al. The Abdominal Manifestations of the Antiphospholipid Syndrome. Rheumatology. 2007;46:1641 – 1647. 2. Pauzner, R., et.al. Hepatic Infarctions During Pregnancy are Associated with the Antiphospholipid Syndrome and in addition with Complete or Incomplete HELLP syndrome. J Thromb Haemost. 2003;1:1758 – 63. 3. Poo, J., et.al. Hepatic Hematoma and Hepatic Rupture in Pregnancy. Annals of Hepatology. 2006;5(3): 224 – 226.

A CASE OF ANTEROGRAD E AMNESIA IN A MIDDLE AGE FEMALE

Introduction Transient Global Amnesia(TGA) is a rare self limiting condition(1). The incidence is reported to be 0.005% to 0.010% per year. The incidence of TGA increases in those over 50 years of age. Precipitating factors include strenuous physical activity, sexual intercourse, severe pain, and psychological stress. Case description A 55 year old Caucasian female was brought to the hospital by her husband because of sudden change in her mental status. Earlier in the morning the patient and her husband had sexual intercourse. Shortly after this she developed intense frontal headache radiating to the back. She had experienced similar headaches in the past, all occurring shortly after sexual intercourse and resolving spontaneously with time. This time the headache was associated with difficulty incorporating new memories and retaining new information. There were no associated visual auras, nausea, vomiting nor did she have any past history of migraines. She described having no numbness, weakness, slurred speech, fevers, chills or changes in bowel or urinary function or recent trauma to her head. Her medical history was significant for breast cancer status post breast conserving therapy. On physical examination she was resting calm in bed in no distress but kept asking repeatedly what was happening and why she was brought to the hospital. She was oriented to place, had full self awareness but was disoriented to time. Remote memory was intact but short term memory was noted to be affected. Her speech was fluent without dysarthria or aphasia. There was no apraxia or nuchal
rigidity. Cranial nerve examination revealed no abnormalities. No papilledema was noted. Motor and sensory functions, coordination and gait were all intact. The rest of her physical examination was otherwise unremarkable. CT scan of the head revealed no abnormalities. A lumbar puncture performed did not show any evidence of xanthochromia. Laboratory findings were otherwise unremarkable. Subsequent magnetic resonance imaging and MR angiography were also unremarkable. Over the course of 24-hour she returned back to her baseline normal neurological state and there were no recurrences. She was diagnosed with Transient Global Amnesia and was subsequently send home. Discussion Differentiating TGA from conditions that may lead to a change in baseline mental function is crucial. The hallmark of Transient global amnesia is anterograde amnesia, without alteration in level of consciousness. Cognitive functions are usually intact and remote memory is not disrupted. Focal neurological deficits and seizure activity are absent. In comparison to patients with delirium, these patients have no inattention. Clinicians should always rule out life threatening conditions like subarachnoid hemorrhage and stroke before making the diagnosis of TGA. 1.Quinette P, et al. What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. Brain 2006; 129:1640.

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PSEUDOMYXOMA PERITONEI

Pseudomyxoma peritonei (PMP) is an uncommon clinical syndrome characterized by the slow and progressive accumulation of peritoneal implants and mucinous ascites. Overtime, accumulation of mucin in the peritoneal cavity results in massive symptomatic distension and associated mechanical and functional gastrointestinal obstruction. In almost all cases, these cells are derived from a ruptured appendiceal neoplasm. CASE REPORT: - 36 year old female from Mexico with a past medical history of Diabetes mellitus and dyslipidemia presented to the Emergency department with recurrent ventral hernia- previously repaired in 2007 which was associated with poorly localized abdominal pain of 2 months duration and gradually got worse for past 1 week. Abdominal pain was associated with nausea, vomiting and increased girth of the abdomen. She also reported a 40 lb weight loss in the past 2 years. She had regular menstrual cycles and LMP was 2 weeks prior to admission. She is a mother of three children and last child birth was ten years ago. No history of ovarian or colon malignancy in the family. Vital signs- on admission- BP-108/65 mmHg, Temp-97.3F, PR-71/minute, O2 Saturation- 97%, Wt-104.1 kg, BMI- 36.8. Physical exam-revealed abdominal distension with ascites and ventral hernia. Findings on CT- Abdomen were suggestive of a tubular fluid-filled complex mass in the right lower quadrant. A normal appendix was not seen which could represent a mucocele of the appendix with pseudomyxoma peritonei. Despite considerable ascites, fluid could not be aspirated from the abdominal cavity. On diagnostic laparoscopy the entire peritoneal surface both visceral and parietal peritoneum was studded with several cystic looking, fluid filled lesions of varying sizes, some as small as a few millimeters and as big as 2-3 cm in size. Frozen section and histopathology examination confirmed the diagnosis of pseudomyxoma peritonei. The patient was referred for cytoreduction and intraperitoneal chemotherapy. DISCUSSION: - PMP remains a disease that follows “an unremitting but prolonged clinical course.” Despite a much improved understanding about the biology of this condition, the impact of therapy is still incompletely understood. Even though complete cytoreduction is associated with prolonged overall survival, recurrence of disease is common and multiple operations are frequently required. Patients may enjoy periods of remission, free of symptoms, but long term disease free survival is distinctly uncommon. Reference- Long term survival following treatment of pseudomyxoma peritonei- an analysis of surgical therapy. Miner TJ, Shia J, Jaques DP, Klimstra DS, Brennan MF, Coit DG, et al. Annals of Surgery: Volume 241, Number 2, February 2005 Pages 300-308.
REPORT OF A CASE OF OSTEO RADIONECROSIS OF MAXILLA AND MANDIBLE PRESENTING AS OSTEO MYELITIS TEN YEARS AFTER RADIOTHERAPY FOR FOLLICULAR LYMPHOMA OF THE BASE OF TONGUE

Background. Osteonecrosis following radiotherapy for head and neck cancers is occasionally encountered; it uncommonly occurs in maxilla as compared with mandible. Report of the Case. A 91 year-old gentleman presented with pain in the lower jaw of two days’ duration. He had undergone dental extraction in the left lower jaw 2 months previously. In 2002, he was diagnosed with follicular-center lymphoma that was confirmed to be limited to the base of the tongue (BoT) on imaging and other workup. He received 4 cycles of chemotherapy based on rituximab, cyclophosphamide, vincristine and prednisone (R-CHOP). Consolidative radiation to a dose of 50.4 Gray in 28 fractions was delivered to BoT, and a dose of 45 gray in 25 fractions was delivered to the lower neck and supraclavicular lymph nodes bilaterally. Examination revealed an induration in the submental region of the neck without definite lymphadenopathy or swelling of the neck. A yellowish purulent ooze from the left lower alveolar margin was observed. CT scan of the neck revealed an erosive radiolucency involving the mandible, with an adjacent soft-tissue swelling, consistent with osteomyelitis, and a large osteolytic lesion in the left maxilla. Biopsy of the maxillary osteolytic lesion, and debridement of the mandibular osteomyelitic lesion was performed. Histopathologic examination confirmed osteomyelitis of the jaw. Empty Haversian spaces with sclerosis and lack of marrow tissue were documented, which represented osteonecrosis that could be attributed to the previous radiotherapy. He was started on IV antibiotics for osteomyelitis, and was referred for the hyperbaric oxygen (HBO) therapy. Discussion. Osteoradionecrosis (ORN) is the necrosis of the osseous tissue following exposure to radiation that manifests weeks or months to several years following the exposure, and is characterized by hypocellular, hypovascular, and hypoxic bone tissue [1]. Osteonecrosis of the jaw is particularly common; it is more common in the mandible than maxilla probably due to a relatively less rich blood supply. It is commonly triggered by the dental extraction, when it manifests as osteomyelitis. Any osteomyelitis of the mandible following dental extraction in a patient previously exposed for a head-and-neck malignancy is to be presumed to be due to osteoradionecrosis unless proved otherwise. The role of HBO in the management is debated [2]. Most patients with ORN have received the radiation for squamous cell carcinoma of the head and neck, whereas the author’s case received it for follicular lymphoma. The fact that the osteoradionecrosis occurred 10 years after the radiation therapy itself makes the case unique. References 1. Marx RE. Osteoradionecrosis: a new concept of its pathophysiology. J Oral Maxillofac Surg 1983;41:283-8. 2. Mendenhall WM. Mandibular osteoradionecrosis. J Clin Oncol;22:4867-8.

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A CASE OF PULMONARY ANGIOSARCOMA WITH INITIAL PRESENTATION RESEMBLING PULMONARY EMBOLISM

Background. Angiosarcoma accounts for 1 to 2% of all soft-tissue tumors. Initial presentation may mimic pulmonary embolism. Report of the Case. A 75 year-old gentleman presented to us with shortness of breath of 3 to 4 months’ duration. He had elevated prostate-specific antigen (PSA), which was investigated with a biopsy, which confirmed prostate cancer. As a part of symptom-guided metastatic work-up CT scan of the chest was performed that revealed multiple lung masses, along with a partially encasing soft-tissue mass invading the main pulmonary artery with extension and near-complete occlusion of the left pulmonary artery, giving the appearance of pulmonary embolism. However, biopsy from one of the lung masses revealed high-grade sarcoma. Pulmonary artery biopsy confirmed the tumor cells to be positive for TLE1, CD99, and Calponin and negative for desmin, SMA, WT1, cytokeratin (E1/AE3), HMB-45, S-100, CD-34, and negative for BCL2, EMA, TTF-1. He received 3D conformal palliative EBRT to the main pulmonary artery primary lesion to the left MPA to a dose of 45 Gray in 18 fractions. Stable MPA mass at the end of the treatment was documented. One year after the initial presentation of PA angiosarcoma he was in performance status 3 with ongoing survival as this report was being written. Discussion. Angiosarcoma accounts for 1 to 2% of all soft-tissue tumors. Pulmonary angiosarcoma is a very rare cancer [1]. Only about 21 cases have been reported in the English language literature so far. Presenting symptom is generally hemoptysis. Because of its origin, it is
occasionally mistaken and initially misdiagnosed as pulmonary embolism, in which case the angiosarcoma presents with shortness of breath. The common radiographic presentation may be multiple peripheral pulmonary nodules or a solitary mass [2]. In the classical form of angiosarcoma, vasoformative features like anastomosing vascular channels lined by malignant endothelium are seen on histopathology. The hallmark of epithelioid variant is solid-sheeted growth pattern with large epithelioid or spindled tumor cells with abundant eosinophilic cytoplasm, large vesicular highly-pleomorphic nuclei and prominent nucleoli. Intracytoplasmic luminas with extravasated red blood cells can usually be seen. Since the disease often presents in advanced stage, the outlook of treatment is palliative, with radiation being the most rewarding modality. Doxorubicin, dacarbazine, vincristine and cyclophosphamide are among the drugs which have been shown to efficacy in pulmonary angiosarcoma. Survival ranges from 1 to 9 months from the diagnosis. References: 1. Primary pulmonary epithelioid angiosarcoma presenting as a solitary pulmonary nodule on image. Yang CF, Chen TW, Tseng GC, Chiang IP. Pathol Int 2012; 62:424-8. 2. Eichner R, Schwendy S, Liebl F, Huber A, Langer R. Two cases of primary pulmonary angiosarcoma as a rare cause of lung haemorrhage. Pathology 2011;43:386–9.

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CHRONIC EXPOSURE TO 1,1-DIFLUOROETHANE AND SEVERE PNEUMONITIS, SOMETHING TO CONSIDER

Introduction: 1,1-Difluoroethane is a refrigerant and the principal component of some computer cleaners; it has been shown to cause central nervous system depression, fatal arrhythmias and sudden death on acute exposure. Chronic Exposure to the component has only been described on rats. Case: This is a 31 year old Mexican female with chronic exposure to 1,1-Difluoroethane at work. She presented to the emergency room twice over 6 weeks with complaints of cough, and wheezing; She was treated with albuterol, azithromycin, and guaifenesin for “bronchitis” and sent home. Worsening of symptoms made her return to the Emergency room for a third time, she presented with shortness of breath at rest, severe cough and orthopnea. On physical examination she looked uncomfortable, with difficulty breathing, she could not speak full sentences without getting dyspneic. On lung auscultation there was diminished air entry, wheezes and crackles bilaterally. Intravenous Steroids were started, as well as wide range coverage of Intravenous antibiotics and antifungal, she also required Bipap. CT scan showed Patchy bilateral upper lobe parenchymal consolidation and ground glass opacities. Blood cultures were negative throughout hospitalization; HIV, ANA, RF, ANCA, pneumococcal and legionella antigens were also negative. As she responded partially to the administered treatment, intravenous steroids were stopped, suspecting an “atypical infection” was the cause of the symptoms and steroids were not needed. Symptoms worsened very fast and steroids had to be restarted. A decision to stop antibiotics and antifungal was made as cultures were negative and no other signs of infection were present. Discussion: 1,1-Difluoroethane is hydrocarbon from the organofluorine family used in refrigerants and canned air; Exposure to this fume has been shown to produce deathly consequences, there are few cases that report fatal arrhythmias, sudden death, and angioedema, Chronic exposure to the component has not been studied in humans; nevertheless in rats it has shown lung irritation, pulmonary edema and ARDS. Conclusion: The lack of any other findings that could explain these symptoms after an intensive workup, with the known exposure to the fume clearly indicates that 1,1-Difluoroethane acted as an irritant; the administration of steroids plus the removal of the stimulus resolved the symptoms. References: 1.- Avella J, Wilson JC, Lehrer M Fatal cardiac arrhythmia after repeated exposure to 1,1-difluoroethane (DFE). Am J Forensic Med Pathol. 2006 Mar;27(1):58-60 2.- Avella J, Kunaparaju N, Kumar S, Lehrer M, Zito SW, Barletta M. Uptake and distribution of the abused inhalant 1,1-difluoroethane in the rat. J Anal Toxicol. 2010 Sep;34(7):381-8.
STEROIDS AND LYMPHOMAS

Intro: In 1832 Thomas Hodgkin published a paper on lymphatic disease. Subsequently, many researchers, clinicians and scientists have described various lymphomas their manifestations, diagnostic and treatment modalities.

Case: A 72-year-old lady from Peru was admitted with anterior neck pain and swelling for 10 days. She had similar symptoms two weeks ago for which extensive workup was done including lymph node and bone marrow biopsy, both of which failed to reveal the etiology. Even flow cytometry failed to give a diagnosis. She had similar but less intense symptoms three months ago in Peru where she got penicillin and steroids for 1 week. During this hospitalization our suspicion for Lymphoma was high and a repeat abdominal and thoracic imaging and lymph node biopsy were done, which revealed a Diffuse Large B Cell Lymphoma (also called Burritt’s like Lymphoma).

Discussion: Steroid treatment can hinder the diagnosis of CNS Lymphomas. This can cause delay in diagnosis and treatment, increasing morbidity and mortality. This probably holds true for peripheral lymphomas too. Conclusion: Steroids can alter the architectural and molecular appearance of lymphomas, both central and peripheral. This can delay diagnosis and affect patient care. Given the fact that steroids are a part of many treatment modalities used to treat certain lymphomas.


THE DANGERS OF TREATING NEUROCYSTICERCOSIS IN CIRRHOTIC PATIENTS

INTRODUCTION: Neurocysticercosis, a parasitic infection of the nervous system, is a leading cause of epilepsy in the developing world. Treatment with Albendazole is not without side effects, and can potentially be fatal. CASE A 62-year-old female with type 2 Diabetes presented with seizures, hemiparesis and confusion while she was in Guatemala. MRI of the brain showed multiple ring-enhancing lesions, and she was diagnosed with Neurocysticercosis. She was started on a 30-day course of Albendazole 200mg twice daily. Phenytoin 100mg thrice daily was given for seizure prophylaxis. She was also concurrently found to have pancytopenia and liver cirrhosis with portal hypertension. Hepatitis panel and HIV testing were negative. Two weeks after treatment was started, she was evaluated in the US for a cutaneous reaction to Phenytoin, which resolved upon replacement of the drug with Leviteracetam. Repeat MRI of the brain showed an increase in the size and number of the lesions. Albendazole was then increased to 400mg twice daily, and the course was extended for two more weeks. The patient was discharged, but returned two weeks later with fatigue. Laboratory studies showed profound worsening of the pancytopenia and further deterioration of liver function. Bone marrow biopsy revealed Aplastic Anemia. She developed Neutropenic Sepsis with E. coli and P. aeruginosa bacteremia, eventually leading to multi-organ failure and death.

Number of Days on Albendazole

<table>
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<tr>
<th>Day 0</th>
<th>Day 14</th>
<th>Day 20</th>
<th>Day 36 (Plus Phenytoin)</th>
<th>(Plus Leviteracetam)</th>
<th>WBC</th>
<th>4.6</th>
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<td>1</td>
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<td>9.7</td>
<td>9.1 7.2 6.7 Platelets</td>
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DISCUSSION Limited literature exists regarding the treatment of Neurocysticercosis in cirrhotic patients. Treatment can be dangerous as observed unfortunately in this case. Severe pancytopenia and death have been reported as a result of Albendazole therapy(1,2) and case reports suggest an increased toxicity of Phenytoin in cirrhotic patients(1,2). Some anti-seizure medications, including Phenytoin and Leviteracetam, are also associated with bone marrow suppression(3). The subsequent pancytopenia can be mistaken easily to be a result of the underlying cirrhosis, leading to a delayed diagnosis of drug-induced bone marrow suppression.

CONCLUSION The antiparasitic and anti-seizure treatment of Neurocysticercosis in cirrhotic patients poses a significant challenge. Given its potential harms and limited proven benefits, Albendazole should be avoided in patients with cirrhosis. Further studies are needed to develop definite treatment guidelines for this subgroup of patients.

BODY PAIN THAT LED TO VISUAL LOSS – GCA A MASTER MASQUERADER

Introduction Giant cell arteritis (GCA) is a well known masquerader. Early diagnosis and treatment are the key to preventing devastating visual loss. GCA may present with systemic symptoms, important being polymyalgia rheumatica (PMR) which if missed, like in our patient will result in irreversible visual loss. Case 84 year old lady from Germany presented to the Emergency Department (ED) with sudden onset, complete, painless loss of vision in the right eye when she woke up that morning. She had been ill for 4 weeks with fever, myalgia, anorexia, malaise and significant weight loss of 20 pounds for which she went to the primary doctor who prescribed ibuprofen. Significant other symptoms included inability to chew from jaw claudication and scalp tenderness. On examination, there was only perception of light in the right eye, with 6/6 vision in the left eye. A relative afferent pupillary defect was noted in the right eye with pallor of right optic disc, consistent with anterior ischemic optic neuropathy (AION). Investigations revealed an ESR of 104mm/hr, CRP of 130mg/dl, platelet of 554,000/microL and alkaline phosphatase of 154U/l. A biopsy of the right temporal artery showed focal acute and chronic inflammation with giant cells consistent with temporal arteritis. She received intravenous steroids immediately. The following day she reported improvement in appetite, jaw pain and malaise. There was, however no improvement in her vision and she was discharged home on oral prednisone. Discussion Early recognition of the systemic symptoms of GCA could potentially prevent blindness. PMR has been observed in 40-60% of patients with GCA. Simple tests like ESR and CRP should be ordered in elderly patients who present with polymyalgia, anorexia, malaise and weight loss. Untreated patients with one eye involvement carry a risk of 20-50% for the second eye. Familiarity with atypical manifestations like fever of unknown origin, cough, tongue pain, transient ischemic attack, scalp necrosis and chronic ear pain facilitates early diagnosis. Ocular symptoms include ophthalmoplegia, diplopia, ptosis, miosis, choroidal ischemia, AION. AION accounts for 80% of the visual loss. Conclusion Permanent vision loss is almost never the first symptom of GCA and is rarely reversible with treatment of the disease. The many disguises of this condition may challenge the diagnostic skills of any experienced physician. References 1) Cheng CK, Lee CC, Huang KH, Wu TE, Peng PH. Giant cell (Temporal) arteritis with anterior ischemic optic neuropathy: a biopsy-proven case in Taiwan. J Formos Med Assoc. 2010;109(7):550-54 2) Paraskevas KI, Boumpas DT, Vrentzos GE, Mikhailidis DP. Oral and ocular/orbital manifestations of temporal arteritis: a disease with deceptive clinical symptoms and devastating consequences. Clin Rheumatol. 2007;26(7):1044-8

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OLOSA HUNT SYNDROME – STILL A DIAGNOSIS OF EXCLUSON

Introduction Tolosa Hunt Syndrome (THS) was defined by International Headache Society (IHS) as episodic orbital pain associated with paralysis of one or more of the third, fourth and/or sixth cranial nerves which usually resolves spontaneously but tends to relapse and remit. In 2004, the IHS re-defined the diagnostic criteria of THS. Granuloma as demonstrated by MRI or biopsy is now also required for diagnosis. Case 60 y/o male complained of left eye pain for 6 days. Over the next few days the pain gradually worsened and was associated with drooping of left eyelid and diplopia. There was no history of migraine, fever, weight loss, joint pain, rash or trauma. On examination, there was evidence of third nerve palsy with complete left eye ptosis, hypotropism, exotropism and horizontal diplopia of left eye. Acuity was 20/40 in right eye, and < 20/100 in left eye. He could not adduct his left eye. All other extraocular movements were intact. Tonometry and fundoscopic examinations were normal. He had no visual field defects and there was no evidence of any other focal neurological deficit. Hematological and cerebrospinal analysis was normal. All imaging including MRI with and without contrast, MRA and MRV of brain were normal. After excluding all other possible causes (Cavernous sinus thrombosis, primary or metastatic intracranial tumor, lymphoma, aneurysm, carotid dissection, infection, vasculitis, basal meningitis) of painful third nerve palsy, a working diagnosis of THS was made. The patient was given a trial of steroids and pain resolved completely within 24 hours. He was
discharged home on 60mg/day of prednisone and on subsequent follow up in 7 weeks there was marked improvement in paresis with 80% of eye opening and moderate improvement in diplopia. Discussion THS was first described by Tolosa in 1954 in a patient with unilateral recurrent painful ophthalmoplegia with involvement of cranial nerves III, IV, and VI. It was then essentially a clinical diagnosis of exclusion. However the IHS Classification of 2004, described it as an entity that occurs rarely, has unknown etiopathogenesis, and manifests clinically by unilateral orbital pain associated with simple or multiple oculomotor paralyses and final diagnosis requires imaging or biopsy evidence. Several recent studies have described various cases of THS without MRI evidence of granuloma (1,2). Conclusion The diagnostic criteria of THS should once again be clinical with imaging playing only a supportive role. Otherwise cases like ours with characteristic clinical features of THS and normal MRI will continue to remain in no man’s land. References 1. La Mantia L et al. Tolosa–Hunt syndrome: critical literature review based on IHS 2004 criteria. Cephalalgia. 2006; 26: 772-81 2. Kirbas D et al. Idiopathic Tolosa-Hunt syndrome: four additional cases. Ideggyogy Sz. 2008; 61: 250-4

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MIXED MEBRANOUS PROLIFERATIVE GLOMERULONEPHRITIS IN HIV & HCV

INTRODUCTION: Membranous Proliferative Glomerulonephritis (MPGN) is caused by proliferation and deposition of immune mediated substances in the glomerular mesangium and basement membrane. These deposits are visualized on electron microscopy as being subendothelial or subepithelial. The disease is classified into 3 types, Type I, II, or III and based on location of deposits, and the type of immune mediated reaction associated with it; whether it be complement activation of the classical or alternative pathway. CASE: A 55-year-old African American male with known HIV, HCV, HTN and CHF was evaluated for hematuria with continuous increase in creatinine and proteinuria (up to 1 gram/day) and a GFR between 30 and 59 (Stage 3 Chronic Kidney Disease). He tested positive in serolog’s for cryoglobulins, ANA, double-stranded DNA, ASO titers and a low C3 complement. Renal biopsy showed complex renal disease involving diffuse mesangial proliferative glomerulonephritis with immune complex type as well (cryoglobulin and HIV-associated). Immunofluorescence that stains 2+ for IgA and trace for IgM, IgG, C3, C1q, kappa and lambda.

DISCUSSION: Renal disease in HIV infected patients was first described in 1984 as a focal segmental glomerulonephritis and termed HIV Associated Nephropathy (HIVAN). Having HIV and an HCV co-infection accelerates the path for HIV disease, leading to an accelerated progression in renal pathology. Time of diagnosis and disease progression poorly correlate with CD4 counts. Type III MPGN consists of two variants; Burkholder variant (MPGN type I with subepithelial deposits) and Strife and Anders variant (complex intramembranous and subendothelial deposits, with marked basement membrane irregularities). Patients present with mixed nephrotic and nephritic syndromes and hypocomplementemia. Immunofluorescence will show a fine granular deposition of IgG and C3 in the mesangium and along the peripheral capillary loops; the reactivity for complement component is usually very strong, commonly stronger than reactivity for IgG. The two components to the treatment of MPGN include treating the underlying cause and assessment of the factors that predict renal prognosis. Factors with favorable prognosis include Non-nephrotic proteinuria, normal serum creatinine or estimated GFR, and normal blood pressure. Factors with poor prognosis include nephrotic syndrome, elevated serum creatinine or a reduced GFR, hypertension and crescents on biopsy. Treatment includes combination of Prednisone, ACE-I and Cyclophosphamide. Dosages and combinations are determined based on the above factors and presentation. REFERENCES: CDC. Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS among Adolescents and Adults. MMWR Recomm Rep. 1992 Dec 18;41(RR-17):1–19. 1993. Rao TK, Filippone EJ, Nicastrì AD, Landesman SH, Frank E, Chen CK, et al. Associated focal and segmental glomerulosclerosis in the acquired immunodeficiency syndrome. N Engl J Med. 1984;310:669–73.
INTESTINAL OBSTRUCTION AND APPENDICITIS AS INITIAL MANIFESTATION OF CROHN’S DISEASE

Introduction: Crohn’s disease (CD) is a condition of chronic inflammation potentially involving any location of the alimentary tract from mouth to anus, but with a propensity for the distal small bowel and proximal large bowel. Case: A 40 year old Hispanic male, non smoker with no significant past medical history came with diffuse upper abdominal pain and 6 episodes of vomiting to the ER. Pain gradually increased overnight and was partially relieved with vomiting. Physical examination revealed diffuse tenderness in the upper part of abdomen more on the right side. Initial labs showed elevated white count of 15,500/cumm and neutrophils of 89%. Rectal examination was unremarkable. CT scan of abdomen without contrast revealed thickening of proximal appendix and periappendiceal stranding and distended cecum and ascending colon with abrupt transition at hepatic flexure. It also showed concentric hypertrophy at level of hepatic flexure. Gastrograffin enema was done which showed apple core lesion at the transition between proximal dilated right colon and decompressed distal colon. At this stage the differential was neoplasia and CD. The patient underwent open right hemicolectomy. Surgical pathology revealed appendicitis and findings suggestive of CD. Colon was filled with hemorrhagic pasty fluid and colonic mucosa showed multiple ulcers. Discussion: CD has been linked to a variety of presentations and complications. It can originate in and be confined to the appendix, yet manifest clinical symptoms leading to emergency laparotomy. Preoperative radiologic findings are similar to those of suppurative appendicitis, but visualization of a markedly thickened appendiceal wall with patent or irregularly narrowed lumen supports the diagnosis of Crohn’s appendicitis (3). Primary CD of the appendix has a favorable long-term prognosis after simple appendectomy, despite a 10% incidence of recurrence as granulomatous ileocolitis. Another complication leading to surgery is intestinal obstruction secondary to chronic inflammation with fibrous scar, which is rarely complete but is high grade (2). Despite the advances in our understanding of CD, the course of the disease in a particular patient remains unpredictable. Surgery is frequently needed, and a large number of patients require more than one operation. (1)

References:

ATTENUATED FAMILIAL ADENOMATOUS POLYPOSIS WITH CHRONIC PANCREATITIS IN A 25 YEAR OLD STUDENT

Introduction- Chronic pancreatitis (CP) is a well recognized entity with numerous causative factors. CP without any identifiable cause is termed idiopathic CP which is rare. Genetic mutations like CFTR gene, CTRC, SPINK1 and PRSS1 are some causes but there are possibly more unidentified mutations that cause idiopathic CP.1 Case- 25 yr old microbiology student came in for evaluation of chronic epigastric pain. In 2007 he had a colonoscopy and CT done for evaluating LLQ pain. Colonoscopy revealed a tubular adenoma polyp in sigmoid colon and the rectum. A repeat colonoscopy in 2012 showed two polyps in sigmoid colon- one tubular adenoma and the other hyperplastic. In 2010 patient was evaluated for acute epigastric pain and an EUS done showed 3/9 EUS criteria for CP with no cysts or masses in the pancreas. EGD was unremarkable. He had no known risk factors for acute or chronic pancreatitis. Since then there has been no recurrence of pancreatitis with any symptoms of CP like steatorrhea or diabetes or pancreatic calculi, except for the epigastric pain. As a child he and his brother were exposed to an insecticide containing organophosphate, for more than 3 months in their new house and were sick for few months. In May 2011 patient had a urine heavy metal screen for Arsenic, Lead, Mercury and Cadmium which was negative. Family History is significant for collagenous colitis in mother. One maternal uncle had pancreatic cancer at age 52 and thyroid cancer at age 51 in another. Maternal grandfather had pituitary cancer and maternal grandmother has polyps in her colon. His younger brother (23 yrs) had a colonoscopy which showed polyps. Discussion- 10% of cases of CP are truly “idiopathic” with no identifiable cause. Recent studies indicate that the idiopathic variety of CP has two subsets—a juvenile form and a senile or late onset form, with distinct clinical features.2 In this patient of ours

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A CASE OF ANTERGRADE AMNESIA IN A MIDDLE AGE FEMALE

Introduction Transient Global Amnesia(TGA) is a rare self limiting condition(1). The incidence is reported to be 0.005% to 0.010% per year. The incidence of TGA increases in those over 50 years of age. Precipitating factors include strenuous physical activity, sexual intercourse, severe pain, and psychological stress. Case description A 55 year old Caucasian female was brought to the hospital by her husband because of sudden change in her mental status. Earlier in the morning the patient and her husband had sexual intercourse. Shortly after this she developed intense frontal headache radiating to the back. She had experienced similar headaches in the past, all occurring shortly after sexual intercourse and resolving spontaneously with time. This time the headache was associated with difficulty incorporating new memories and retaining new information. There were no associated visual auras, nausea, vomiting nor did she have any past history of migraines. She described having no numbness, weakness, slurred speech, fevers, chills or changes in bowel or urinary function or recent trauma to her head. Her medical history was significant for breast cancer status post breast conserving therapy. On physical examination she was resting calm in bed in no distress but kept asking repetitively what was happening and why she was brought to the hospital. She was oriented to place, had full self awareness but was disoriented to time. Remote memory was intact but short term memory was noted to be affected. Her speech was fluent without dysarthria or aphasia. There was no apraxia or nuchal rigidity. Cranial nerve examination revealed no abnormalities. No papilledema was noted. Motor and sensory functions, coordination and gait were all intact. The rest of her physical examination was otherwise unremarkable. CT scan of the head revealed no abnormalities. A lumbar puncture performed did not show any evidence of xanthochromia. Laboratory findings were otherwise unremarkable. Subsequent magnetic resonance imaging and MR angiography were also unremarkable. Over the course of 24-hour she returned back to her baseline normal neurological state and there were no recurrences. She was diagnosed with Transient Global Amnesia and was subsequently send home. Discussion Differentiating TGA from conditions that may lead to a change in baseline mental function is crucial. The hallmark of Transient global amnesia is anterograde amnesia, without alteration in level of consciousness. Cognitive functions are usually intact and remote memory is not disrupted. Focal neurological deficits and seizure activity are absent. In comparison to patients with delirium, these patients have no inattention. Clinicians should always rule out life threatening conditions like subarachnoid hemorrhage and stroke before making the diagnosis of TGA. 1. Quinette P, et al. What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. Brain 2006; 129:1640.

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RIGHT HEART THROMBUS -ACUTE MANAGEMENT IN A SECONDARY MEDICAL FACILITY

Introduction Free floating right heart thrombi although rare, carry a mortality rate of nearly 100% without treatment, 30% with anticoagulation and 11-25% with embolectomy. Case A 62 y/o morbidly obese female was brought to the ER with confusion and possible drug overdose. She was comfortable, neurologically intact, no JVD, normal heart and lung exam with no LE edema. Her ECG showed QT interval 663msec without ST-T changes and a CK of 999. She was admitted to the ICU, treated for Trazodone overdose and rhabdomyolysis. Day 2 the patient was found to have mild SOB and tachycardia. CT-angio was done with high suspicion of PE and lovenox was started empirically. Results revealed bilateral lobar, segmental, subsegmental PE. She was
started on Coumadin. She also had asymptomatic elevation of troponins which were attributed to the PE. Day 3 her INR was supratherapeutic at 3.6 and Coumadin was held. A 2d echo results showed– large mobile right atrial thrombus, right heart dilatation and pulmonary hypertension. On exam the patient was comfortable yet anxious. JVD was present with hepatojugular reflex, and a faint cardiac murmur with clear lungs. EKG showed right axis deviation with atrial enlargement. Heparin drip was started and a tertiary care hospital was contacted to transfer for embolectomy. Early the next morning the patient became unresponsive and ACLS was performed. Unfortunately she could not be resuscitated. Discussion Mobile right heart clots are most commonly complicated by pulmonary embolism. The mortality rate without treatment ranges from 80-100%. There is no gold standard management however general therapy is to prevent further clotting and potentially remove the clot; limited to large medical facilities. In this patient’s case it’s debatable that thrombolysis should have been considered from the start. However would this have been too aggressive as she was hemodynamically stable and with supratherapeutic INR. And could the INR have been a result of liver congestion secondary to acute right heart failure and not a true value. It is difficult to say if the patient would have benefited from thrombolytic therapy, opposed to heparin drip. Each option of treatment seems to carry equally negative side effects. It seems the best management is still controversial. References: 1. Lawrence Lazar, M.D et al. Dilemma of Right Atrial Thrombi, to Dissolve or to Extract. Proceedings of UCLA Healthcare Vol. 16 (2012). 2. Pierre-Justin G. et al. Management of a Mobile Right Heart Thrombi: a prospective series. University of Fort De France, Department of Cardiology. 3. Mark N. Levine, M.D. et al. Hemorrhagic Complications of Thrombolytic Therapy in the Treatment of MI and VTE. Chest (1992): 364 – 373.

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SHORT LASTING UNILATERAL NEURALGIFORM HEADACHE WITH CONJUNCTIVAL INJECTION AND TEARING (SUNCT)

Introduction: Short lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT) is a rare headache syndrome classified among the Trigeminal Autonomic Cephalalgias. Case report: A 64 year old female with no past medical history, complained of new onset, episodic, unilateral, right retro-orbital and peri-orbital headache of 2 weeks duration. Pain was stabbing in quality, with increase in frequency of painful episodes. Pain was precipitated by light, sound, sudden movement of head, chewing or touching on right side of face, associated with tearing and conjunctival injection. Ibuprofen did not relieve her pain. Headache initially started as 2 to 5 episodes/day but in 2 weeks progressed to 30 to 40/day. Episodes lasted between 2 to 10 min. Pain was so severe that she was unable to function or to get out of bed. There was no photophobia, phonophobia or vomiting. Vital signs were normal. Headache could be precipitated by palpation of right fronto-temporal region. Neurological examination was normal. Imaging studies including CT head, MRI and MRA of brain was done. No abnormality noted on VEEG. Initial differential diagnosis were cluster headache and paroxysmal hemicranias but 100% oxygen inhalation, Triptans and Indomethacin failed to produce any response in first 4 days. Pain was severe, disabling, and typically refractory to initial therapy. With suspected diagnosis of SUNA or SUNCT, we started treatment with Oxcarbazepine, Lamotrigine, Topiramate. IV Lidocaine was tried, but due to insufficient pain control and unwanted side effect including severe dizziness, we had to discontinue it after 12 hours of total treatment. Only mild symptomatic relief was achieved by Sphenopalatine ganglia block. Over the course of 10 days, we could control attacks of cephalalgia. Discussion: • The patient described here met the International Headache Society criteria for SUNCT syndrome, i.e. more than 30 attacks of unilateral, moderately severe, orbital or temporal, stabbing, lasting for 120-600 seconds, associated with one of the following cranial autonomic features: conjunctival injection, lacrimation, nasal congestion, rhinorrhea, ptosis and eyelid oedema. It is very hard to differentiate between trigeminal autonomic cephalalgias (TAC). Especially new onset headache with no past medical history (PMH). Etiology and pathology of SUNCT is not well understood. The attack of SUNCT can be sudden without any precipitating factor. Subcutaneous infusion of Lidocaine is effective in only 78%. Reference 1) Max H. Williams, Simon A Broadley et al, SUNCT and SUNA: Clinical features and medical treatment Journal of Clinical Neuroscience, May 2008, Vol 15, Issue 5, Pages 526-534.
DISSEMINATED HISTOPLASMOSIS A PROTEAN DISEASE

Introduction: Histoplasmosis is the most prevalent endemic mycosis in the United States. While most infections are asymptomatic or self-limited, some individuals develop acute pulmonary infections or severe and progressive disseminated infection (1). Approximately 70 percent of patients with disseminated histoplasmosis have involvement of the gastrointestinal tract at autopsy. Gastrointestinal manifestations are recognized clinically in less than 10 percent of cases. Lesions include ulcerations or polypoid masses, leading to misdiagnoses of colitis or malignancy (2). Lesions most often involve the colon or ileum, especially the ileocecal area, but may occur from the mouth to the anus. Other findings include esophageal pain or difficulty swallowing, abdominal pain, gastrointestinal bleeding, colonic perforation, and polypoid lesions resembling cancer (3). Case: A 32 years old Mexican male admitted for watery diarrhea and weight loss for 2 weeks. Developed septic shock, respiratory failure, pulmonary edema, acute respiratory distress syndrome, hepatic injury, renal failure, pancytopenia, disseminated intravascular coagulopathy and was diagnosed with AIDS with a CD4 count of 7. Diagnosis of histoplasmosis was made with bone marrow biopsy. Treatment with Amphotericin B was initiated but at day six of admission patient expired. Autopsy showed bilateral acute bronchopneumonia, diffuse alveolar damage, and disseminated Histoplasma capsulatum involving liver, spleen, gastrointestinal serosa, urinary bladder serosa, mesentery and omentum, adrenal glands and bone. The small and large intestines, appendix, esophagus and stomach showed massive serosal involvement by histoplasma. Discussion: Patients with acute histoplasma infection present with fever, fatigue, hepatosplenomegaly, and pancytopenia. Diarrhea and dyspnea occur less commonly. Severely immunodeficient patients, such as those with AIDS or those receiving treatment with immunosuppressive medications, can present with overwhelming infection manifested by shock, respiratory distress, hepatic and renal failure, obtundation, and coagulopathy. The mortality in spite of amphotericin B treatment approaches 50 percent in such cases (4). Our case demonstrated that clinical presentation of disseminated histoplasmosis, is protean and requires a high index of suspicion, recognition of the common modes of presentation, as well as familiarity with the appropriate diagnostic tests. Treatment is highly effective but mortality is high in disseminated cases. Reference: 1.- Goodwin RA Jr, Shapiro JL, Thurman GH, et al. Disseminated histoplasmosis: clinical and pathologic correlations. Medicine (Baltimore) 1980; 59:1. 2.- Assi MA, Sandid MS, Baddour LM, et al. Systemic histoplasmosis: a 15-year retrospective institutional review of 111 patients. Medicine (Baltimore) 2007; 86:162. 3.- Goulet CJ, Moseley RH, Tonnerre C, et al. Clinical problem-solving. The unturned stone. N Engl J Med 2005; 352:489. 4.- Wheat LJ, Connolly-Stringfield PA, Baker RL, et al. Disseminated histoplasmosis in the acquired immune deficiency syndrome: clinical findings, diagnosis and treatment, and review of the literature. Medicine (Baltimore) 1990; 69:361.

A CURIOUS CASE OF SPONTANEOUS INTRAHEPATIC BLEED

Introduction: Spontaneous intrahepatic bleed(SIHb) is a rare event, and caudate lobe bleeding is even rarer. There have been over 100 cases reported in the literature, with the majority being associated with pregnancy-induced-hypertension as well as primary (adenoma) and metastatic liver tumors [1]. However, hepatic rupture in the absence of an underlying pathology is an extremely rare occurrence. We describe a patient presenting with liver rupture with no clear etiologic cause and without history of abdominal trauma. Case 51 y/o Uruguayan male, previously well, presented to the emergency room with 1 day history of sudden onset of constant bilateral upper abdominal pain, radiating to his back. No similar episodes in the past, loss of appetite, fever, night sweats or trauma. On examination, mild tenderness was present at right upper quadrant. Laboratory evaluation demonstrated white count of 16.8/cmm, hemogobin 13.4gm/dl, platelet count 170 cmm with AST 35 U/L, ALT 36 U/L, Alkaline phosphatase 78 U/L, Total Bilirubin 1mg/dl, serum albumin 4.7gm/dl with normal amylase, lipase, CA 19-9 with negative hepatitis serology. CT scan and MRI of abdomen revealed enlarged periportal lymph nodes (largest measuring 3.7 * 3.6 cm) with hepatosplenomegaly (Liver 22 cm and spleen 16.7 cm) with mild stranding of pancreatic head. During the course of hospitalization his pain worsened with drop of Hgb to 9.0gm/dl. Patient was hemodynamically stable with benign physical examination. A repeat
CT scan showed marked enlargement and heterogeneity localizing adjacent to/within the caudate lobe of the liver with evidence of hemorrhage adjacent to the liver and spleen with no peritoneal leak. Angiography showed similar findings along with a focus of active hemorrhage medial to the caudate lobe. No intrinsic hepatic disease was noted. EGD was essentially normal. No source for the hemorrhage was identified. Patient was managed conservatively with blood transfusion and repeat CTA showing findings suggestive of caudate lobe hematoma with mild increase in perihepatic hemorrhage seen anterior to inferior right lobe of liver. Patient improved clinically, was hemodynamically stable and was discharged home. Outpatient laparoscopic examination is scheduled. Discussion SIHB may be a life-threatening complication of various underlying conditions and may represent their first manifestation. One study showed that the etiology for SIHB was hepatic adenoma in 40% of cases, hepatocellular carcinoma in 25% and various other liver pathologies in the rest [2]. But even in the absence of classical clinical scenarios for SIHB, one must be vigilant for this life threatening condition. References 1. R. A. Sheikh et al, “Spontaneous intrahepatic hemorrhage and hepatic rupture in the HELLP syndrome: four cases and a review,” JP Clin Gastroenterology, 1999; 28: 323-328. 2. Battula N et al, “Aetio-pathogenesis and the management of spontaneous liver bleeding in the West: a 16-year single-centre experience,” HPB 2012;14:382-389.

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COMPICLATED PARAPNEUMONIC PLEURAL EFFUSION

Modern principles of pleural effusion management are based on early diagnosis, correct use of antibiotics and prompt pleural drainage. Ten percent of patients with parapneumonic effusion will develop complicated parapneumonic pleural effusion (CPPE). CPPE is associated with increased morbidity and mortality. It is necessary to differentiate CPPE requiring intervention from uncomplicated effusion. Differentiation is achieved using clinical, pleural fluid (PF) and imaging parameters. Intervention strategies include tube thoracostomy placement and surgical decortication. A thirty year old woman with a medical history of recent caesarean section, gestational diabetes and preeclampsia presented with shortness of breath, right upper quadrant pain and fever for three days. The pain was moderate, sharp, increased with deep inspiration and was associated with progressively worsening dyspnea. She denied cough or sputum. On physical examination BP 153/83 mmHg, T 100.7F, HR 109 bpm, RR 20/min and oxygen saturation 97% on room air. She appeared well nourished and in moderate distress. Lung exam revealed equal expansion of the chest wall bilaterally. There was decreased air entry in right lung base with mild crackles and dullness to percussion. Pertinent laboratory data included a WBC count of 11,000. Chest imaging showed a large, right-sided pleural effusion with associated atelectasis. The patient was diagnosed with health care associated pneumonia with parapneumonic effusion and was started on vancomycin, piperacillin-tazobactam and levofloxacin. An ultrasound-guided thoracentesis was performed with removal of 600 ml of amber, cloudy fluid. PF analysis revealed an exudate, with 61% PMNs and 24% lymphocytes. LDH was 1085 and pH 7.0. No organisms were seen on Gram stain. Fluid culture was negative. Because of the PF characteristics a chest tube was placed to facilitate drainage of the complicated effusion. Although patient had initial clinical improvement, resolution of symptoms and effusion was not complete. Repeated imaging showed hydropneumothorax with large area of consolidation and suggestive findings of collapsed right lower lobe. She ultimately required video assisted thoracoscopic surgery (VATS) for pleural debridement, parietal pleurectomy and decortication. CPPE is suggested by PF pH < 7.2 and elevated LDH. Patient did not have risk factors such as COPD, smoking, alcoholism or drug abuse as typically seen in CPPE. This case illustrates the importance of initial sampling of all parapneumonic effusion and a rigorous stepwise approach in management. It includes antibiotics, chest tube placement for drainage and VATS decortication. VATS should be performed without any delay if inflammation and residual fluid are still present in order to achieve complete lung expansion and reduce hospital stay. References: 1. Rodriguez P, et all. Treatment of complicated parapneumonic pleural effusion and paraneumonic empyema. Med Sci Monit. 2012; Jul:443-9. 2. Hampson C, et all. Diagnosis and management of parapneumonic effusions. Semin Respir Crit Care Med. 2008; Aug:414-26.
INVASIVE DUCTAL CARCINOMA AND PHYLLOIDES TUMOR COEXISTING IN DIFFERENT BREASTS

INTRODUCTION: Available literature reports that Phylloides tumor commonly presents with ductal and lobular in situ breast lesions and less frequently with invasive lesions. If present with invasive breast cancer, it is usually situated inside or adjacent to the fibro epithelial neoplasm in the same breast. CASE REPORT: Patient is a 49 y/o Caucasian female who presented with progressively worsening shortness of breath. She also noticed swelling in the right chest wall below the right axilla one month ago which eventually involved the right breast with palpable lymph nodes. No associated pruritus, pain or nipple discharge. One week ago she started noticing a painless, non pruritic left breast swelling underneath her nipple. No palpable lymph nodes in the left axilla. No relationship with menstruation was noted. No weight loss or loss of appetite was present. She has a history of 1 pack per day cigarette use for the last 33 years, family history of breast cancer in paternal grandmother and aunt. She has never used Oral Contraceptive pills and never had a mammogram. She has never been tested for BRCA. Chest X ray showed massive right pleural effusion which was subsequently drained. CT chest showed bilateral breast masses highly suspicious of malignancy, multiple lung nodules, ovarian, liver and axial skeleton metastases. She had multiple pleurocentesis for relief of her dyspnea. Pleural fluid cytology showed malignant cells. We proceeded to do ultrasound guided needle biopsy of both breasts which showed Invasive ductal breast carcinoma with focal lobular features, about 1.3 cm in the right breast and fibroepithelial lesion with stromal proliferation suggestive of Phylloides tumor in the left. The tumor was positive for Estrogen and Progesterone receptors and negative for p53 and HER2. She was scheduled for VATS pleurectomy and sclerosis, and was started on Tamoxifen. Surgical intervention or radiation therapy is not recommended for her as she has advanced Stage 4 breast cancer with liver, lung, ovarian, bony metastasis and malignant pleural effusion. Patient was scheduled for outpatient palliative chemotherapy. CONCLUSION: Benign phylloides tumor and invasive breast carcinoma coexisting together in separate breasts simultaneously is very rare. It would be worth looking if the presence of the phylloides tumor has an effect on the end prognosis. Also there are no clear guidelines for the treatment modalities and response to treatment in such cases which is a potential area of future research. References: Coexistence of benign phylloides tumor and invasive ductal carcinoma in distinct breasts: Neto G B et al : European journal of medical research, ISSN 0949-2321, 2012, Volume 17, Issue 1, p. 8 Concomitant phyllodes tumour and homolateral breast cancer: Auerbach, Leo The lancet oncology, ISSN 1470-2045, 05/2002, Volume 3, Issue 5, p. 279

PARADOXICAL EMBOLIC STROKE AND ORTHODEOXIA

Introduction: Hypoxemia has many etiologies. Pulmonary and extra pulmonary shunting is one of them. Patients with these shunts can develop paradoxical emboli. In many circumstances neurological deficits or stroke become the first clinical manifestations of such shunts. Case Presentation A 75-year-old man with history of hypertension was admitted with sudden onset left sided weakness for 5 hours. He also had left sided facial droop, left arm drift and slurred speech. His blood pressure was 130/70 mmHg. CT scan of the head without contrast showed no evidence of intracranial hemorrhage or infarct, EKG showed sinus bradycardia and a 1st degree A-V block. Patient did not have any absolute contraindications for thrombolytic therapy. Alteplase was given under the diagnosis of acute ischemic stroke and patient was admitted to the ICU. Control MRI showed recent right pontine and left superior cerebellar lacunar infarcts but noa intracranial hemorrhage. Neurological deficits resolved over the course of 72 hours but patient was found to be hypoxic. Hypoxia increased with changes in position, other causes of hypoxia where ruled out but the probability of a shunt remained. A 2D transthoracic echo with saline contrast (“bubble study”) was done and showed an atrial septal defect. Discussion Intracardiac shunts can cause orthodeoxia, which may be mild, but usually do not respond to oxygen therapy. Causes can range from various types of atrial septal defects, ventricular septal defects or complex cardiac abnormalities. Such patients can present for the first time with paradoxical emboli. 2D transthoracic echo with the bubble study protocol can identify such shunts. Conclusion Patients with neurological deficits and persistent hypoxia should make us think of intracardiac shunts and paradoxical emboli. The saline contrast study is extremely safe, inexpensive and sensitive method to detect such

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A CASE OF BLUE FINGERS

Introduction: Venous thrombosis, a common complication of malignancy, presents most often as pulmonary embolism. Arterial thrombosis, which results in limb/organ ischemia, however, is a much less frequent finding. Arterial occlusion can stem from tumor/cardiogenic embolism, both of which are still rare events (1). We present a case of acute limb ischemia as the presenting symptom of advanced lung carcinoma. Case: A 62 year old female presented to the ED with bluish discoloration of her Left 5th digit which progressed to all the digits over 10 hours and was associated with pain. On ROS she had been suffering from a nonproductive cough for one month. She received antibiotics but cough persisted. Social history was significant for cessation of smoking 5 months prior. Pertinent findings on physical exam included submandibular lymphadenopathy, bibasilar crackles, diminished radial pulse and delayed capillary refill on the left upper extremity. Basic Laboratory studies were normal. Chest x-ray showed bilateral interstitial lung disease and pleural effusions. CT angiogram revealed an occluded left subclavian artery from its origin extending to the origin of the vertebral artery origin and also mediastinal, supraclavicular, and hilar lymphadenopathy. Given the location of occlusion, embolectomy was not performed. Instead patient was started on anticoagulation. Fine needle aspiration was done on pretracheal lymph nodes and showed non-small cell carcinoma most suggestive of poorly differentiated adenocarcinoma of lung origin. Discussion: Acute arterial occlusion originating from an embolus most likely occurs in an area of acute narrowing of the artery. Most frequent locations include femoral (28%), arm (20%), aortoiliac (18%), and popliteal (17%)(2). Further diagnostic evaluation would have been interesting in this patient to determine if the thrombus was malignant or not. One case report has shown tumor invasion of the pulmonary artery which mimicked a sarcoma but was actually extension of lung adenocarcinoma with an overlying nonmalignant thrombus as well (3). Malignant embolism can occur when the tumor invades the walls of the pulmonary vein and fragment into embolus (1). Conclusion: This patient may have formed a thrombus as a complication of tumor extension or from cardiogenic origin. Arterial thrombosis and acute limb ischemia are both rare complications of malignancy and as presenting symptom of lung carcinoma. References: 1. Schreffler S.et al. Spontaneous showering of tumor emboli in a patient with advanced primary lung cancer. Int J Emerg Med. 2012; 5:27. 2. Goto T. et al. Lung Adenocarcinoma with peculiar growth to the pulmonary artery and thrombus formation. World J Surg Oncol. 2012;10:16. 3. Mitchell M. et al. Acute arterial occlusion of the lower extremities. UpToDate 2012.

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A CASE OF EPIDURAL SPINAL CORD COMPRESSION

Introduction: Neoplastic epidural spinal cord compression arises when a tumor invades the epidural sac and compresses on the thecal sac. The symptomatology varies from asymptomatic to paraplegia. The neurologic deficits can be irreversible if not treated promptly. Approximately 20% of the cases are initial manifestations of a neoplasm. Case report A 32 year old male came to the emergency department complaining of dyspnea, sweating, anorexia, vomiting and a 15 pound weight loss in a month, associated with lower back pain, bilateral leg weakness and numbness, and sharp pain behind his thighs. He also complained of constipation and urinary incontinence which had worsened over the course of 5 to 6 weeks, after he sustained a fall from a
Cholangiocarcinoma, despite growing to be 15.6cm x 15.8cm with vascular invasion and survival of approximately 6 months. This case provides evidence that a poorly differentiated perihilar adenocarcinomas being the most common. Despite aggressive anticancer therapy and interventional supportive care (ie, wall stents or percutaneous biliary drainage), the median survival rate is low with an overall survival of approximately 6 months. This case provides evidence that a poorly differentiated perihilar cholangiocarcinoma, despite growing to be 15.6cm x 15.8cm with vascular invasion, can present with minimal,

Cholangiocarcinoma is a malignancy arising from the biliary ductal epithelium and classified as intrahepatic, perihilar, or distal bile duct cancer. The average incidence is 1-2 cases per 100,000 persons per year with perihilar adenocarcinomas being the most common. Despite aggressive anticancer therapy and interventional supportive care (ie, wall stents or percutaneous biliary drainage), the median survival rate is low with an overall survival of approximately 6 months. This case provides evidence that a poorly differentiated perihilar cholangiocarcinoma, despite growing to be 15.6cm x 15.8cm with vascular invasion, can present with minimal,
vague symptoms and negative serum markers. This case highlights the difficulty in using serum markers for screening and diagnosis of cholangiocarcinoma. In addition, this case confirms the aggressive nature of this tumor type with IVC invasion and diagnosis at an advanced stage of disease.

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ATYPICAL PRESENTATION OF METASTATIC PROSTATE CANCER

Objectives: 1) Review prostate cancer metastatic to a vertebral body presenting, seemingly paradoxically, with anterior chest pain 2) Review the management of spinal cord compression  
Case: A 74-year-old African American male presented with one week of intermittent chest pain described as sharp, over the anterior chest wall with band-like radiation toward left side. Denied nausea, diaphoresis, exacerbating or alleviating factors. Activity: able to walk twenty blocks daily. Exam: Normal. EKG, troponins x 3, stress test were negative. When approached for discharge, he stated he "will fall if sent home", reported new onset numbness in his lower extremities, left knee pain, and fell to the floor while trying to walk. EXAM: no trauma, inflammation, or tenderness over joints of the lower extremity or vertebrae. CN II –XII intact. Muscle strength 5/5 upper and lower extremities bilaterally. Normal muscle tone, finger-to-nose and heel-to-shin. Decreased sensation below the T7 dermatome. Diminished reflexes in the patellar tendons. Negative for Hoffman''s, clonus, babinski. Unable to stand without assistance. Rectal: Normal tone with a non-tender diffusely enlarged/nodular prostate. PSA > 154ng/ml. MRI cervical/thoracic spine: lesion at the T6 vertebral body extending into the epidural space compressing the spinal cord. CT pelvis: 5.5 x 4.4 cm nodule in the prostate. CT guided biopsy of the vertebral body lesion revealed metastatic prostatic adenocarcinoma. He was started on corticosteroids, casodex and radiation therapy which resulted in improvement of symptoms.  
Discussion: Prostate adenocarcinoma is the second leading cause of cancer in the United States. The manner in which the patient presented is unique in that his spinal cord compression presented with anterior chest pain without symptoms suggestive of prostate involvement such as weight changes or urinary symptoms. Literature search did not reveal other case reports of metastatic prostate cancer initially presenting as chest pain. Spinal cord compression is a medical emergency that can lead to irreversible spinal cord injury and permanent paralysis, thus early diagnosis and treatment is crucial for neurological recovery. A retrospective study of 153 patients revealed the most common presenting symptoms of spinal cord compression as radicular pain, motor weakness, sensory deficits, and bladder dysfunction. In a retrospective study of 28 patients with neoplastic spinal cord compression, the most common presenting symptoms were back pain (68%), bilateral leg weakness (61%), urinary retention (36%), and bilateral leg numbness (32%). Delays in diagnosis occurred among patients who presented with unilateral leg weakness, pain, ataxic gait, back pain, or symptoms in the neck, chest, and arms. MRI is the most sensitive diagnostic test. Treatment includes dexamethasone to reduce the surrounding edema, surgical decompression, and radiation therapy for spinal cord compression secondary to malignancy.

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SECONDARY HYPERKALEMIC PARALYSIS: MIMICKING GUILLAIN-BARRE SYNDROME

OBJECTIVE: 1) Recognize the rare neuro-muscular presentation of hyperkalemia. 2) Appreciate the lack of electrocardiogram (ECG) findings in severe hyperkalemia.  
CASE SUMMARY: An 80 year old female presented to the emergency room complaining of progressive fatigue and weakness over the past month. Specifically, she had trouble standing from the seated position. Most recently she had been confined to a bed. The evaluation revealed a potassium level of 9 meq/L (normal 3.5-5.0). She had teeth extracted 6 weeks ago and since then her diet had consisted almost entirely of soft foods such as bananas. Her prescription medications included spironolactone for her stress cardiomyopathy. Cardiovascular, respiratory and abdominal exam were unremarkable. On neurological examination, cranial nerves II–XII were intact; motor strength was 5/5 in the distal and proximal upper extremities, however lower extremity motor testing showed that ankle and knee flexion/extension were 2/5, hip flexion was 2/5. Patellar reflexes were 1/2 bilaterally. Sensation was intact to dull and sharp, joint proprioception normal. Fine tremor was present during rest and exertion. Assessment of gait was not done due to inability to stand. ECG showed sinus rhythm with no ST changes or T wave changes. The hyperkalemia was treated with sodium polystyrene sulfonate, insulin/glucose, and hemodialysis.
Hemodialysis brought about complete resolution of symptoms. DISCUSSION: Hyperkalemia is an electrolyte disturbance in which patients are often asymptomatic in routine clinical practice. Neurological symptoms associated with this are often overlooked and misdiagnosed. The most common neurological manifestation is ascending quadriparesis or quadriplegia with diminished or absent reflexes. However, our patient presented with increasing lethargy and muscle weakness pronounced in the proximal muscle groups of the lower extremities, sparing the upper extremities, along with hyporeflexia. In 1998, there were only eighteen reported cases of secondary hyperkalemic paralysis. Fifteen presented as tetraparesis/tetraplegia and only three presented as paraparesis of the lower extremities. Another unique and surprising aspect of our case was the normal ECG was normal with potassium of 9 meq/L. Commonly in the clinical outpatient and emergency department setting, ECG is considered to be a sensitive tool to predict the severity of hyperkalemia and lethal arrhythmias. However, reports have shown poor correlation between ECG changes and serum potassium level. In conclusion, paresis is an important clinical feature of hyperkalemia yet seldom reported in literature. It can be insidious in its presentation and can often be mistaken for Guillain-Barre syndrome due to its ascending nature. This case highlights the importance of not solely focusing on cardiac abnormalities but also the neurological component associated with hyperkalemia.

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THINKING OUT OF THE BOX: PICA RELATED FOREIGN BODY INGESTION AS A SOURCE OF GI BLEED

Objectives: (1) Recognize an uncommon etiology of GI bleed, (2) Review diagnosis and management of GI bleed secondary to foreign body ingestion. Case: 47 year old male with a history of multiple strokes, cognitive impairment and Pica presented with 2 days of 3-4 large bloody bowel movements. Exam: HR 116, BP 100/60. Pale. Abdomen: non-distended, non-tender, normal bowel sounds; bright red blood clots per rectum. Neurologic/Psychosocial: Awake but not alert or oriented; non-verbal; Left Cranial Nerve III palsy. Patient witnessed to ingest paper and other objects within reach in the emergency room. Labs: BUN 74 mg/dL, Cr 3.18 mg/dL. WBC 14.1, Hb 8.8 g/dL, Hct 26.1%, MCV 91.9 fL, INR 1.10. The patient was admitted to intensive care for active hematochezia and borderline hemodynamic instability. Nasogastric tube placement and endoscopy ruled out upper gastrointestinal (GI) tract as source of bleed. Colonoscopy revealed a toothpick lodged in the colon with surrounding edema and mild bleeding, which was removed via endoscopic snare. Follow-up abdominal films were negative for additional foreign bodies. Post toothpick retrieval, hemoglobin remained stable; no further bloody bowel movements were noted. Gastrostomy tube was inserted and the patient was transferred to a supervised facility. Discussion: The majority of ingested foreign bodies pass through the GI tract without medical or surgical consequences. However, sharp objects such as toothpicks, which account for 9% of foreign bodies ingested, are associated with high risk of complications such as perforation, fistula formation and bleeding. Adults who ingest such foreign objects usually have a psychiatric disorder, mental retardation, or substance abuse which makes obtaining history and eventual diagnosis challenging. Early diagnosis and retrieval of foreign objects are imperative, especially in patients with perforation, which is associated with high morbidity rate. Initial diagnostic evaluation includes radiography, which is only useful if the ingested object is radiopaque. Endoscopy which can be diagnostic and therapeutic for visualization and extraction with a polypectomy snare should be attempted prior to surgical intervention. If the cause of uncontrolled bleeding is not elucidated with colonoscopy, definitive diagnosis may be made via laparotomy, and is indicated in peritonitis, abscess, and fistula formation as well. The management of GI bleeding secondary to foreign object ingestion is similar to other etiologies except for the need to retrieve the culprit foreign body. Fluid and blood product resuscitation is indicated for hemodynamically unstable patients. In conclusion, in patients with intractable GI bleeding, it is important to consider foreign body ingestion as a cause, especially
ACUTE STENT THROMBOSIS IN A YOUNG PATIENT POST MYOCARDIAL INFARCTION WITH ANTI-PHOSPHOLIPID SYNDROME

Objectives: (1) Define anti-phospholipid syndrome and clinical manifestations, (2) Recognize a rare case of recurrent acute stent thrombosis and aPL, (3) Speculate clopidogrel resistance in aPL syndrome Case Report: A 47 year-old Caucasian male active tobacco user with family history of coronary artery disease presented with substernal chest pain of 6 hours. EKG revealed inferior wall STEMI emergently sent to cardiac catheterization after aspirin and clopidogrel loading doses. Initial angiograms revealed an occluded and thrombosed right coronary artery (RCA). After successful aspiration thrombectomy and drug eluting stent (DES) placement to proximal and mid-distal RCA she was transferred to the CCU pain free on Reopro infusion, aspirin and clopidogrel. Eight hours post-catheterization he developed recurrent chest pain with an episode of ventricular fibrillation requiring defibrillation. Emergent re-catheterization showed rethrombosis of his entire RCA. Successful reperfusion was established with aspiration, mechanical thrombectomy, and intracoronary thrombolysis. The remainder of his hospital stay was uneventful. PostMI echocardiogram showed preserved left ventricular ejection fraction. Hypercoagulable state was suspected as cause of recurrent thrombosis, and his Anticardiolipin Antibody titer was 26 GPL on 2 occasions, 12 weeks apart. He was diagnosed with Primary Antiphospholipid Syndrome and eventually discharged home in stable clinical condition. Discussion: Antiphospholipid Syndrome (APS) is an autoimmune disorder that damages phospholipids proteins found in cell membranes leading to recurrent thrombotic events. Definitively diagnosed by Sydney criteria as (1) arterial or venous thrombosis, miscarriage and (2) positive titers of at least one type of anti-cardiolipin, anti-B2 glycoprotein or lupus anticoagulant on two separate occasions at least 12 weeks apart. It can be of primary origin or be secondary to a connective tissue disease. Cardiac manifestations of APS include valvulopathy/vegetations, Libman-Sacks endocarditis, myocardial infarction, ventricular hypertrophy/dysfunction, intracardiac thrombus and pulmonary hypertension. It is estimated that aCL antibodies are found in 5% to 15% of patients with MI and the association increases to 21% in patients under 45 years of age. Few case reports have documented acute stent thrombosis in patients with APS. Of particular notice is our patient that developed recurrent thrombosis despite being on three anti-platelet agents. To our knowledge, this is the first reported case of recurrent coronary thrombosis so shortly after an initial thrombosis and despite post-intervention anticoagulation that meet current standards of care. It is also possible that he has clopidogrel resistance. A cohort study found clopidogrel resistance in 8.6% of 13% with DES thrombosis. Another study found prasugrel therapy to have significantly lower rates of stent thrombosis when compared with clopidogrel, but also an increased risk of bleeding in patients with scheduled PCI. Although the concept of using prasugrel for suspected clopidogrel resistance to reduce the risk of stent thrombosis the efficacy in primary APL syndrome warrants further investigation.

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“IS IT REALLY PARANEOPLASTIC PEMPHIGUS?”

Objectives: Review the presentation, diagnostic criteria and treatment of paraneoplastic pemphigus (PNP). Case: A 42 year old man with chronic lymphocytic leukemia for 3 years presented with sore throat and multiple painful mouth ulcerations. He reported a 35 pound weight loss within 6 weeks due to poor oral intake from the oral ulcers. EXAM: multiple oral, lip and skin ulcerations on the extremities and trunk; swollen and tender cervical lymph nodes. Labs: BUN 24 mg/dL, Cr 1.13 mg/dL. CBC normal except WBC: 104 x 103/µL with absolute lymphocyte 84.6 x 103/µL. The patient was admitted to the hospital for parental volume repletion and empiric broad spectrum antibiotics for upper airway infection. Paraneoplastic pemphigus was suspected; thus, he was started on empiric steroid therapy and biopsy of his skin and oral lesions was obtained. The lesions improved and he was transferred to a pemphigus specialist for further management. The biopsy of the oral lesion showed suprabasal bullae filled with keratinocytes and large hyperchromatic nuclei with scattered acute inflammatory cells. Although the oral mucosa biopsy showed no direct immunofluorescence (DIF) staining specific for immunoglobulin or complement, the skin biopsy revealed staining of infiltrating leukocytes for IgG and of the basement membrane zone for IgA and C3. This is compatible with pemphigoid lesions and, in
addition to other criteria, confirms the diagnosis of PNP. Discussion: PNP was first described in 1990 as an autoimmune mucocutaneous disorder associated with lymphocytic malignancies. The pathophysiology is thought to be tumor antigens that evoke both a humoral and cellular immune response leading to mucosal and epithelial blistering. The clinical manifestation varies; however, painful and intractable mucositis is a constant feature. Stomatitis usually presents as extensive erosions and ulcerations affecting the oropharynx, causing soreness and dysphagia. The diagnosis requires either 3 major or 2 major and 2 minor signs. Major signs: 1. Polymorphic mucocutaneous eruption. 2. Underlying proliferative lymphocytic neoplasm. 3. Serum autoantibodies to antiplakin. Minor signs: 1. Histological findings of vacuolar interface change, keratinocyte necrosis. 2. DIF showing intercellular and basement membrane deposition. 3. Indirect immunofluorescence staining with rat bladder epithelium. Due to the rarity of the disease, there is no specific treatment guideline; however, high dose corticosteroids are a mainstay of therapy. Treatment of the underlying malignancy can improve PNP, and an immunosuppressive agent such as rituximab may be considered for refractory cases. The prognosis depends on the nature of the underlying malignancy and is worse with malignant tumors, which are associated with a higher mortality rate. In conclusion, diagnosis of PNP should be entertained in a patient with lymphocytic malignancy who presents with mucositis with cutaneous symptoms.

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DOES STRESS TESTING BEFORE ELECTIVE HIP FRACTURE SURGERY INCREASE LENGTH OF STAY AND/OR ALTER PATIENT MANAGEMENT

Introduction: Retrospective studies suggested that pre-operative cardiac stress testing could identify patients at high risk for peri-operative myocardial infarction who might benefit from coronary revascularization, but a prospective randomized trial demonstrated that revascularization of high risk patients did not decrease mortality. The accepted approach to patients with acute non-traumatic hip fracture is surgery within 24-48 hours of admission. Stress testing is unlikely to alter management, but could cause delays in patient care. The objective was to determine if a preoperative stress test administered to patients with acute hip fractures was associated with a longer length of stay (LOS), and if an abnormal result was associated with an alteration of the care plan. Methods: Retrospective case-control study of patients seen at our hospital 2006-2011. Patients were identified by ICD-9 for hip fracture and procedure codes for persantine or dobutamine stress tests. Patients were matched by age and gender. Results: 42 patients who had a pre-operative stress test (STRESS) were matched to 42 controls (CONTROLS). The mean age was 81 years in both groups, and 70% were female. The respective mean values (days) for the STRESS group vs. CONTROLS were: LOS 8.3 vs. 4.7 (p=0.0002); Time to Operating Room 2.5 vs. 1.4; Time from surgery to discharge 5.7 vs. 3.2. The LOS was 3.6 days longer for the STRESS group [95%C.I. (1.7, 5.5)]. When a cardiologist was consulted 73% had a stress test (n=48) vs. vs. 20% when they were not (Risk Ratio 3.8 [95% CI (1.9, 7.5)]), and the mean LOS was 7.9 vs. 4.5 days (p=0.0004). Among the STRESS group, the time to the operating room for those with ischemia was 2.6 days, suggesting that an abnormal stress test result did not alter management. 60% of patients were on beta-adrenergic blocking agents, and this was true for those with and without ischemia on the stress test. Neither of the patients who died had asthma or COPD, but only 1 was on a beta-blocker. The following were associated by Logistic Regression with stress testing: cardiology consultants, COPD, atrial fibrillation and hypercholesterolemia. Surprisingly, diabetes and a known history of coronary disease were not associated with testing. Conclusion: In acute hip fracture patients, preoperative stress tests were associated with a longer length of stay, delays in getting to the operating room, and longer time from surgery to discharge. Abnormal results did not prompt alterations in management such as revascularization and beta-blockers were not prescribed to 2 patients with stress-induced ischemia. This data suggests that such testing in this population increases LOS without affecting clinical decision making, and education of cardiologists could significantly decrease this “lets just be safe” practice style.
CAN DIFFUSION WEIGHTED IMAGING IMPROVE PREOPERATIVE AXILLARY LYMPH NODE STAGING IN WOMEN WITH NEWLY DIAGNOSED BREAST CANCER?

PURPOSE Axillary lymph node (ALN) status in patients newly diagnosed with breast cancer provides crucial prognostic information. Surgical staging of the axilla is highly accurate but is associated with significant morbidity. Dynamic contrast-enhanced (DCE) MRI is useful for detection of malignant ALNs but has insufficient negative predictive value to obviate traditional surgical staging. Diffusion weighted imaging (DWI) reflects cell density through apparent diffusion coefficient (ADC) values and may provide complementary information to DCE. We assessed the potential of DWI to improve axillary staging by comparing ADC values of biopsy-proven malignant and benign ALNs presenting as suspicious on DCE MRI. METHOD AND MATERIALS After IRB approval, we retrospectively identified consecutive patients with newly diagnosed breast cancer from 3/2006 to 1/2010 who underwent ultrasound-guided core needle biopsy of morphologically suspicious ALNs first identified on DCE MRI. All MRIs were performed at 1.5T with DCE and DWI (b=0, 600 s/mm²) techniques. Benign results were confirmed via surgical ALN staging. ADC values were calculated for suspicious appearing ALNs (biopsy proven malignant and benign) and contralateral normal appearing ALNs and compared by Wilcoxon signed rank test and Mann-Whitney U test. RESULTS We evaluated 41 biopsy-proven ALNs (21 malignant, 20 benign) and 34 contralateral normal ALNs in 41 women (mean age=49±13.2 years). Normal ALNs were not measured in 7 cases (contralateral cancer, n=3; ALN too small to measure on DWI, n=4). Overall, intrasubject comparisons showed ADC values of suspicious ALNs were lower than contralateral normal ALNs (1.02±0.22 vs. 1.12±0.23×10⁻³ mm²/s, p=0.004); However, among suspicious ALNs, there was no significant difference in ADC between pathology-proven malignant and benign ALNs (1.02±0.23 vs. 1.02±0.21×10⁻³ mm²/s, p=0.8). CONCLUSION Although suspicious appearing ALNs exhibit lower ADC than normal appearing contralateral ALNs, no differentiation can be made between benign and malignant suspicious ALNs. This suggests that DWI provides similar information to DCE MRI for assessing the axilla and cannot help to obviate surgical staging. Our study suggests that DWI does not provide complementary information to DCE MRI for preoperative axillary lymph node assessment.

ENERGY DRINK CONSUMPTION AMONG HOUSE STAFF

Purpose: The use of over the counter and prescription medications to enhance concentration and cognitive processing has become habitual among medical students, residents, and faculty physicians. There is very limited knowledge as to why house staff use caffeine, the effects caffeine on their sleep cycle, mood, and behavior, and what are beliefs regarding caffeine use. The purpose of this study was to describe the attitudes, beliefs, and usage patterns of energy supplements among house staff at Jersey Shore University Medical Center Methods: After institutional IRB approval, an anonymous computerized electronic questionnaire was emailed out to all residents at Jersey Shore University Medical Center (JSUMC) in the departments of Medicine, Obstetrics and Gynecology, Pediatrics, Surgery and Dentistry. The questionnaire included 10 multiple choice questions with option for free text. These questions collected information regarding residency department, post-graduate training year, type of drinks/medications and average number of dosages/day (0 to >5), participants’ purpose for use of energy supplements use as well as withdrawal symptoms. Other health-related information such as amount and type of exercise per week, amount of sleep (hours per night), and medical/health problems were also collected. Results: The response rate was 64%. Out of 32 participants that responded to the survey, 59% replied they use energy supplements. The common types of drink/medication used were coffee (86.7%), soda (40%), tea (33.3%), and energy drinks such as Red Bull, RockStar, Monster, 5-hour energy, Amp, Hype, (20%). The average amount is one dosage per day, while 6.3% of participants reported using 4-5 doses per day. The most common reason for usage is to increase wakefulness (100%), followed by mood enhancement (18.5%), improve decision making capacity (11.1%) and improve driving ability (7.4%). Withdrawal symptoms, particularly headaches and crash episodes were noted in 43.3% of the caffeine users. Conclusion: Despite the stressful lifestyle and sleep deprivation, energy supplement consumption among house staff, although quite common, is not as prevalent as hypothesized. The small sample size made
it difficult to assess how popular energy supplements are among house staff at JSUMC. Further research should be done to target a broader range of hospital faculty and to study the correlation between post-graduate year training and the amount of energy supplement consumption.

**PALINDROMIC RHEUMATISM: A RARE INFLAMMATORY ARTHRITIS SYNDROME THAT MAY EVOLVE INTO RHEUMATOID ARTHRITIS**

Objectives: (1) Recognize the presentation and differential diagnosis of palindromic rheumatism. (2) Understand the relationship between palindromic rheumatism and rheumatoid arthritis. Case Presentation: A 57 year-old healthy male presented with progressive weakness, subjective intermittent fevers, and night sweats for the past 6 months. He also described migratory joint pains and swelling involving his left shoulder, right hip, bilateral wrists, and MCP joints over this time. His joint pains were intermittent and lasted several days, and he was symptom-free for a few days in between joint flares. He recently returned from a trip to Mexico and developed watery diarrhea, which resolved after a few days. He has a distant history of scalp psoriasis, and a maternal grandmother had rheumatoid arthritis. On exam, he was afebrile and there was bilateral wrist tenderness with decreased range of motion. There was no synovitis, dactylitis, psoriasis, nail pitting, or other rash. Laboratory values were significant for WBC $18.2 \times 10^3/\mu L$ (4.5-11) with 81.9% neutrophils, ESR 30 mm/h (0-15) and C-reactive protein 6.19 mg/L (0-0.8). Blood cultures were negative, and laboratory testing for Lyme disease, syphilis, HIV, malaria, hepatitis B and C, gonorrhea, Chlamydia, tuberculosis, and Whipple’s disease were unrevealing. ANA and HLA-B27 were negative, rheumatoid factor was elevated at 37.6 IU/mL (<20), and anti-CCP IgG was elevated at 193 U (<20). Over time, his joint symptoms became persistent, involving his bilateral shoulders, wrists, ankles and feet, associated with one hour of morning stiffness. He failed to respond to non-steroidal anti-inflammatory medications, but responded dramatically to an intramuscular 80 mg dose of methylprednisolone. Methotrexate and ultimately etanercept were added to his regimen, and his symptoms are progressively improving. Discussion: Palindromic rheumatism is characterized by episodic inflammation affecting one or more joints for a period of hours to days. It may involve different joints during different episodes; however, there is no cumulative clinical or radiographic damage. Most importantly, there is a symptom-free period that lasts from days to months between episodes. Palindromic rheumatism is a clinical diagnosis of exclusion. The differential diagnosis includes infections such as Lyme disease, syphilis, gonorrhea and Chlamydia. Due to the patient’s travel history, Whipple’s disease was also considered. Workup includes ruling out autoimmune conditions including rheumatoid arthritis, systemic lupus erythematosus, reactive arthritis and crystal arthropathy. Over time, attacks of palindromic rheumatism may persist or spontaneously remit. In 28-67% of patients, palindromic rheumatism may ultimately progress to rheumatoid arthritis. Both rheumatoid factor and anti-CCP antibody may be positive in palindromic rheumatism, as observed in this patient, but the predictive value of these markers is unclear. This case highlights the importance of recognizing this rare syndrome, ruling out other causes, and closely monitoring for progression to rheumatoid arthritis or other autoimmune disease.

**TPA FOR PULMONARY EMBOLISM: IS INTRA-ARTERIAL ADMINISTRATION ASSOCIATED WITH A DELAY IN ADMINISTRATION?**

**BACKGROUND:** Intravenous tissue plasminogen activator (IV-tPA) is an indicated treatment when pulmonary embolism (PE) causes hemodynamic instability. Intra-arterial tPA (IA-tPA) is also used, theoretically because a smaller dose can be administered locally with a lower bleeding risk. IV-tPA can be administered at the bedside, but IA-tPA requires transport to the Interventional Radiology suite, preparation for angiography, and placement of a pulmonary artery catheter. We hypothesized that the additional preparation required for IA-tPA would be associated with delays in administration of the medication. **OBJECTIVE:** Determine the mean time from diagnosis to treatment among patients with PE treated with IV vs. IA-tPA. **METHODS:** The data warehouse was used to identify patients treated between 2000-2011 who received the tPA doses typically used for IV administration (50 or 100mg), and those who received the typical IA dose (12.5 mg). The charts were then
reviewed to determine if PE was the indication. The Shapiro-Wilk test was used to determine that the data was normally distributed. RESULTS: 19 charts were identified, 15 received tPA for PE (6 IV, 9 IA). One IV patient was excluded from the analysis because time to administration was 17 hours. The mean respiratory rates were significantly different in the IA vs IV groups 22+-/4 vs. 29+-/5 (ttest p=0.01), but there were no significant differences in mean age, blood pressure, heart rate, pulse oximetry, hemoglobin, or creatinine. The mean time, in minutes, from diagnosis to tPA administration was 158+-68 for the IV-tPA group and 112+-/28 for the IA group (ttest p=0.07). 4(80%) of IV-tPA patients required mechanical ventilation vs. 3(33%) of IA-tPA patients. 2/5 IV-tPA patients died, and 1/9 of the IA-tPA group died. For those that did not die, the mean length of stay was 22+-11 days for the IV-tPA (n=3) group and 8+-3 for the IA-tPA (n=8) group (p=0.001).

CONCLUSIONS: This preliminary chart review rejected our hypothesis that treating PE with IA-tPA would be associated with a longer time to administration. Like any retrospective study where treatment was determined by a potentially large group of clinicians, there are limitations. It is possible that IA was administered to patients perceived as having more severe disease. The surviving IV patients had a longer length of stay and higher rate of respiratory failure, findings that could be confounded by IA being more effective. The small numbers limited our ability to identify objective measures that could have driven the clinical decision. In summary, this review of PE cases treated over 11 years found that IA-tPA does not appear to be associated with delays in care. The retrospective design and small numbers do not allow us to conclude that it is more effective.

**MONMOUTH (SARA WALLACH, MD)**

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Statin induced Rhabdomyolysis: Unusual and Aggressive Presentation for Pravastatin

Muscle injury is a well-known complication of statin therapy and the spectrum includes myalgias, myositis and rhabdomyolysis. Rhabdomyolysis occurs only in less than 0.1% of patients. The risk appears to be lowest with pravastatin and fluvastatin since they are not extensively metabolized by CYP3A4. We present a patient on pravastatin who developed an aggressive course of statin induced rhabdomyolysis, complicated by acute on chronic kidney injury leading to chronic hemodialysis. A 55 year old male presented to the Emergency Department with a four day history of worsening bilateral lower limb weakness. Past medical history included coronary artery bypass grafting, ischemic stroke, hypertension, dyslipidemia, diabetes mellitus, and chronic kidney disease stage IV. Medications included glipizide, amiodipine, losartan, tamsulosin, aspirin, clopidogrel, carvedilol, clonidine, torsemide and pravastatin (40mg/day). He had a 44 pack year smoking history and denied alcohol or recreational drug use. There was no history of recent viral infections or similar complaints in the family. Neurological examination showed 3/5 motor strength in RLE & 4/5 in LLE with intact reflexes, intact sensations and normal cerebellar examination. Gait was not assessed secondary to weakness. Respiratory, cardiovascular and abdominal examinations were unremarkable. Laboratory results revealed potassium 5.3mEq/L creatinine 4.22mg/dl (baseline 3), BUN 65mg/dl, ALT 388U/L, AST 961U/L, CK 53,756 IU/L, ESR 88mm/hr and aldolase 46.1; hemoglobin 12.3g/dl, WBC 11600 and platelet count 360,000. Urine analysis revealed large blood but only 6-10 RBC. Anti Jo 1 antibody was negative. Pravastatin and torsemide were held and intravenous fluids with sodium bicarbonate administered. His CK reached a peak of 96,662 IU/L on the seventh hospitalization day. He was subsequently started on hemodialysis. Patient recovered with normalization of CK and AST levels within 20 days. Left quadriceps muscle biopsy showed lymphocytic infiltrate and denervation atrophy consistent with statin induced myopathy. The safety of pravastatin (40 mg/day) was confirmed in an analysis of more than 112,000 patient-years of experience in three large controlled trials. The incidence of serum CK elevations was not different from placebo, and there were no cases of confirmed clinical myositis or rhabdomyolysis. Our patient's course demonstrated extensive muscle injury caused by pravastatin leading to AKI. This is a reportable event according to pharmacological literature. There are several other risk factors that predispose to statin-associated myopathy which include acute/chronic renal failure, obstructive liver disease, hypothyroidism, pre existing neuromuscular disorders, genetic risk factors and concurrent drug therapy (particularly inhibitors of CYP3A4). Practitioners should educate patients to discontinue statins and immediately report symptoms of muscle weakness. Patients with a history of statin-induced rhabdomyolysis should generally not be treated with another statin (including pravastatin and fluvastatin) because of the risk of recurrence.
Melanoma is a malignant and aggressive neoplasm affecting the skin, mucous membranes, and central nervous system and accounts for 3% of all malignancies. The incidence of melanoma is increasing and is among the top ten cancer in the United States. Melanoma can metastasize anywhere, with very few cases reported to bone especially spine, in which case the median survival is usually 2-6 months. We present the case of a 64 year old lady with occult primary who presented with multiple metastatic lesions to brain and bone, with bone biopsy confirming the diagnosis of melanoma. A 64 year old lady with history of hypertension, diabetes, dyslipidemia, chronic back pain status post lumbar laminectomy done eighteen years ago presented with worsening back pain for 2 weeks. She denied any fever, chills, bowel or urinary incontinence, tingling, numbness or weakness. She was seen in the Emergency room and was sent home on pain medications. However the patient experienced worsening back pain with radiation to her left knee. The pain was worse with movement, walking and partially relieved with the pain medications. Vitals were stable. Physical examination was unremarkable including neurological examination. Labs revealed a normal wbc count, Hb-12.3 creatinine- 1.05, BUN -22, GFR- 52. MRI lumbar and thoracic spine showed enhancing lesions on T6, T10, T11, L2 - L3, right sacral ala, and pathological fracture T 11, suspicious for metastatic disease. Initial work up ruled out multiple myeloma. CT chest showed 3mm nodule in right lung and asymmetry of left breast. Mammogram was normal. Further evaluation with MRI brain revealed two lesions in the left cerebral and one in the left cerebellar hemisphere. Bone biopsy was scheduled for confirmatory diagnosis. The patient underwent kyphoplasty of T11 compression fracture during which bone biopsies were obtained. The preliminary report revealed an undifferentiated anaplastic tumor of the spine. The final biopsy reported a high grade malignant neoplasm consistent with metastatic malignant melanoma. A thorough examination of her skin and fundus revealed no primary lesions suggestive of melanoma. The plan was to give radiation to T11- T12 spine for pain relief and gamma knife irradiation to the lesions in the brain. Tumors from thyroid, breast, prostate commonly metastasize to the bone. Very few cases have been reported of melanoma metastasizing to the bone (especially spine) with occult primary. In such cases prognosis is very poor. In evaluating bony lesions with no overt primary, melanoma should be considered part of differential diagnosis even though it is a rare presentation. The mainstay of treatment in such cases is shrinking the lesions, pain control and prevention from further spreading. However there has been some progress in treating metastatic melanoma with drugs that target by stimulating the immune system.

ANCA associated vasculitis includes granulomatosis with polyangiitis (GPA), microscopic polyangiitis(MPA), Churg Strauss Syndrome and renal limited vasculitis. There are various classifications based on clinical presentation and serology to differentiate the types of vasculitis. None of the classifications differentiate between GPA and MPA. "The defining pathological difference between the two is the presence of granulomatous change on biopsy in GPA". We present a 73 year old lady who was admitted twice over two months for various symptoms, the constellation of her clinical presentation, lab findings and renal biopsy consistent with the diagnosis of MPA. A 73 year old Caucasian lady came to the Emergency room with complaints of diarrhea and abdominal pain for 1 day. She had loose watery stools with mild lower abdominal pain but denied any fever, chills, nausea, vomiting, recent travel or unusual food intake. Review of system was positive for recent use of antibiotics for ear infection, dizziness and imbalance for which she was seen during previous admission and CT brain showed lacunar infarct posterior aspect of right internal capsule. Biopsy of lung nodule found on CT chest was negative for malignancy. It was decided to get PET scan as an outpatient in 6 weeks. However the patient ended up in the hospital again for abdominal pain. Vitals were stable. Physical examination revealed mild diffuse lower abdominal tenderness. Lab work showed elevated WBC - 31.7 7% bands, Hb 11.7 Hct 36, platelets 598, BUN-49 Creatinine-3.41 baseline Creatinine- 0.5, normal LFT. Urine analysis revealed protein and blood. CT abdomen showed cecal wall thickening suggestive of focal colitis. Stool for clostridium difficile was sent. Patient was empirically started on antibiotics and intravenous fluids. The patient improved significantly however it was noticed that her leukocytosis never trended to normal and platelet "SIMPLE AS IT APPEARS, IS NEVERTHELESS NOT SIMPLE"
count trended to 900. Given the thrombocytosis, lacunar infarct, malignancy was high on differentials. However PET scan was unremarkable. Meanwhile creatinine ranged between 2-3, and vasculitis work up revealed low C4, elevated proteinase 3, ANA positive with 1:160 diffuse pattern. Empirically started on high dose steroids pending kidney biopsy. Kidney biopsy reported as focal segmental necrotizing and crescentic glomerulonephritis consistent with diagnosis of microscopic polyangitis. Microscopic polyangitis is associated with symptoms that affects kidneys, nervous system, lungs and skin. When a patient presents with a constellation of symptoms that involve ears, skin, lungs, kidneys, anemia, thrombocytosis, cerebral infarcts, conduction abnormalities, uveitis, differentials should include ANCA associated vasculitis. Biopsy is required to make a definitive diagnosis. However if clinical suspicion is high patients should be treated empirically. Prompt diagnosis is required to initiate treatment which can be organ and or life saving.

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TO IDENTIFY THE RISK FACTORS THAT MAY CONTRIBUTE TO READMISSIONS AT MONMOUTH MEDICAL CENTER WITHIN 28 DAYS OF DISCHARGE.

Introduction: Healthcare costs are imposing an increasing burden on Federal Budget. Medicare spending has increased incredibly over the past years and is expected to increase to $234.9 Billion in 2019. A report published in NEJM in April 2009 stated that 19.6% of Medicare fee-for-service beneficiaries were readmitted to the hospital within 30 Days of discharge, 34 % within 90 Days and more than 51% within one year. Starting OCTOBER 2012, Medicare will decrease the payments to the Hospitals with higher number of Readmissions. Objective: To carry out a study at our institution to identify the Risk Factors associated with the Readmissions and try to avoid unnecessary readmissions and payment cuts. Methods: This is a Retrospective Descriptive Study which quantifies the readmission rates to medicine services and determines the factors contributing to readmissions. We reviewed 400 medical records of patients who were readmitted to the medical service from January 2011 through December 2011. The records were reviewed for the following factors: age, gender, ethnicity, diagnosis on primary admission and readmission, primary reason for admission, patient disposition, living status and functionality, mental status of the patient, medical errors committed during the readmission, chronic illnesses and outpatient follow-up. Results: Out of 400 charts reviewed, 41% of patients who were readmitted within 28 days of discharge were over 75. Also 54 % of patients were females and 50% of patients discharged to Care facility were readmitted. Other factors which were assessed are: multiple co-morbidities, number of days between admissions, readmission for same diagnosis and preventability of readmissions. Out of 7000 admissions in 2011 at our facility, 865 patients were readmitted within 28 days of discharge. That gives a readmission rate of 12.3 %. This appears to be lower compared to data released from Med PACs 2008 report of 2005 data which was 17.6% at 30 days. We will use this information to establish algorithm to reduce readmissions. Conclusion: Age and multiple co-morbidities play a big role in readmitting patients in the hospital. It is hard to prevent readmission especially in the elderly and sick population because clinical course is very unpredictable. Few limitations to our study include- inability to access data if these patients were hospitalized in other hospitals; the risk factors analyzed for readmissions need to be compared with the control group of non-readmitted patients which we didn’t have at the time we accumulated the data. We also think the comorbidities affecting readmission need to be further analyzed on the basis of direct impact on readmissions.
ENDOBRONCHIAL ASPERGILLOMA MASKING MALIGNANT THYMOMA

Thymomas are rare epithelial neoplasms accounting for 15% of all mediastinal masses. Typically, they are slow-growing and may invade contiguous structures in one third of cases, but they rarely metastasize to distant sites. Endobronchial polypoid extension of Thymic carcinomas is an exceedingly rare and poorly understood phenomenon. Preoperative diagnosis is frequently difficult. Surgical resection is the mainstay of treatment and adjuvant radiotherapy appears to be beneficial. We report a case of a malignant Thymoma with non-contiguous polypoid endobronchial growth and superimposed Aspergillosis. To our knowledge, 19 cases of endobronchial thymomas have been reported in the literature. Only one case describes superimposed Aspergillosis; our case is the second in the literature to describe this presentation. Our patient, a 67 year old male of Asian descent, presented initially with cough and mild dyspnea due to an obstructing right upper lobe lesion. Bronchoscopic biopsy initially showed fibrinoid necrosis, and bronchial washings showed Aspergillus organisms. The patient was initially treated with Voriconazole, with clinical improvement. Several months later, the patient returned with mild hemoptysis and weight loss. Repeat CT showed progression of the obstructing lesion. Repeat bronchoscopy and biopsy with a flexible and a rigid bronchoscope again showed only benign bronchial epithelium and necrotic cells. A thoracotomy revealed a mediastinal tumor invading the right upper lobe and an unattached tan, hemorrhagic, and soft endobronchial mass obstructing the right main bronchus. Both tumors were resected along with the right upper lobe. Histopathology revealed a type A malignant Thymoma. Radiation therapy was initiated postoperatively.

A YOUNG FEMALE WITH GRANULOMATOUS VERTEBRAL OSTEOMYELITIS AND EPIDURAL ABSCESS

We are presenting a case of cervical spine osteomyelitis and epidural abscess secondary to tuberculosis in a 25 year old previously healthy female from India, who immigrated to the United States in 2006. She presented to us with complaints of posterior neck pain of 6 weeks duration radiating to both shoulders without any neurological deficits. She worked as a microscopist in an electronic company. She denied any fever, chills, night sweats, weight loss, cough, dysphagia or hemoptysis. She did not give a history of recent travel, neck trauma, sick contacts, recent oral infection or instrumentation in her spine. She had received BCG vaccine as a child. She had tuberculosis test done at the time of her immigration but she couldn’t remember the results. MRI and CT of the cervical spine showed a large abscess centered at the C3 vertebral body with significant prevertebral and ventral epidural abscesses and cord compression at C3. Serum chemistries, liver panel, leukocytes were within normal limits. ESR and CRP were minimally raised. She was admitted and started initially on intravenous methylprednisone. She had a positive tuberculin test at 72 hours. A bone scan showed moderate uptake at the cervical spine. Quantiferon Gold TB test was negative. She underwent open C2-3 to C4-5 laminectomies with posterior fusion in order to relieve the cord compression. Fungal stain, fungal cultures and gram stain sent from the necrotic tissue were all negative. Acid fast bacilli were not seen. Frozen section biopsy from the lesion showed granulomatous necrosis. Patient was clinically diagnosed with Pott’s disease and was started empirically on antitubercular therapy. Acid fast cultures were found to be positive for mycobacterium tuberculosis at 8 weeks. She was followed by neurosurgery and infectious disease team and had a dramatic improvement. Majority cases of vertebral osteomyelitis and epidural abscess in the United States are due to Staphylococcus aureus. Mycobacterium tuberculosis is the most common organism in the developing world. The patient usually but not always presents with classic triad of fever, spinal pain and neurologic deficits. Non-specific neck or back pain in the absence of constitutional symptoms of fever and neurological symptoms is often overlooked and treated as a benign musculoskeletal problem. Thus, a high index of suspicion is needed while evaluating a young patient presenting with spinal pain. We conclude that though tuberculosis is a rare disease in the west, it represents a minor population of spinal infection. It should be included in the differential diagnosis of patients with spinal pain, especially in those with a history of
immigration from a developing country. Involvement of the spine can be the only manifestation of tuberculosis and it may be seen even in immunocompetent patients without history of previous tubercular infection.

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ACUTE UNILATERAL SWOLLEN PAINFUL THIGH IN A LONG STANDING DIABETIC PATIENT

We present a 65 year old Caucasian woman with a history of long standing diabetes mellitus complicated by retinopathy, hypertension, coronary artery disease with prior placement of drug eluting stents, and chronic systolic heart failure. During a recent admission for heart failure at another hospital she developed pain in her left thigh after a minor fall. She was told that she had a muscle strain and was discharged home. She presented to our emergency room one week later with unremitting and progressive pain in her thigh. She denied fever, chills or arthralgias since her fall. Her exam was notable for warmth, edema and tenderness of her left thigh. Although the pain in her thigh was severe, there was no apparent pain with passive range of motion at the left hip and left knee was without effusion or warmth. A complete blood count, coagulation profile and lower extremity doppler were normal. Metabolic panel was significant for hyponatremia of 128mg/dl, chloride of 90 mg/dl, glucose of 247 mg/dl, BUN of 23 mg/dl and creatinine of 1.5 mg/dl. Radiological assessment with X rays and bone scan were negative for fractures. CT scan demonstrated mild subcutaneous edema within medial and lateral mid to distal thigh. Other labs showed an elevated ESR of 105, CRP of 57, ferritin of 273, total CK of 386 and normal procalcitonin level. Patient remained afebrile but had persistent pain despite treatment with high dose opiate analgesics. MRI of the left thigh showed extensive intramuscular edema throughout vastus medialis and intermedius muscles extending from intertrochanteric region to patella. Appearance was consistent with myositis. Ultrasound guided muscle biopsy was performed and antimicrobial therapy with cefazolin and vancomycin was initiated. Histopathology showed skeletal muscle degeneration consistent with necrotizing myositis with reactive neutrophil infiltration. Culture was negative. Compartment pressures in the left thigh were not elevated and surgical debridement of the necrotic muscle was deferred as the risk of morbidity was felt to be high and the benefit uncertain. She required high doses of parenteral opiates for her pain which subsided after several weeks. During this time she developed progressive renal failure and encephalopathy of unclear etiology. She was started on hemodialysis and underwent renal biopsy which demonstrated moderate nodular diabetic glomerulosclerosis, moderate to severe tubular atrophy, interstitial fibrosis and moderate to severe arteriolosclerosis consistent with diabetic kidney disease. This case report describes diabetic myonecrosis or muscle infarction which is a rare complication of long standing, poorly controlled diabetes mellitus. It often involves thigh and calf muscles and is associated with advanced microvasculopathy. It is usually self limited and responds to conservative management but has a poor prognosis. Diabetic myonecrosis should be considered in diabetic patients presenting with focal muscular pain and swelling.

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ACUTE RENAL FAILURE IN A HEALTHY YOUNG MALE PATIENT

A 20 year old healthy male with no significant past medical history presented to the emergency department with multiple episodes of watery, bilious, non bloody vomiting associated with nausea and poor oral intake for 3 days. He also had an episode of watery diarrhea. The patient denied any fever, chills, abdominal pain, recent respiratory tract infections, ill contacts, new medications, eating outside or loss of weight. Physical examination including abdomen was normal and no tenderness was elicited. His initial labs were significant for white blood cell count of 20.7 k/µL, chloride of 88 mg/dl, blood urea nitrogen of 39 mg/dl and creatinine of 4.3 mg/dl. Urine analysis was negative for cast, sediments, eosinophils, glucose or protein. Urine sodium was 83 mg/dl and fractional excretion of sodium was calculated to be 3.4%. The patient was treated with IV fluid hydration and anti-emetics. Renal ultrasound demonstrated bilateral echogenic kidneys with no atrophy or hydrenephrosis. There was no prior documented imaging of the kidneys for comparison. Nephritic workup including Hepatitis B and C, HIV, C3C4, ASO, Anti GBM, ANA and Anti-DNA were negative. After extensive further questioning, patient admitted to smoking “K2” which is a synthetic cannabinoid for 3 days during the week before he presented. Urine drug toxicity panel was pan negative including for cannabinoids. Other causes for acute
gastroenteritis including Giardia, Salmonella, Shigella, and Campylobacter were ruled out. He improved symptomatically and started tolerating oral feeds, hence renal biopsy was withheld. The patient was discharged after 4 days in the hospital with serum creatinine of 2 mg/dl and blood urea nitrogen of 21 mg/dl. He was advised strictly not to use those drugs anymore. The patient was followed up after 6 weeks and his repeat labs showed normal renal function. This case report describes acute renal failure in a young healthy male possibly due to “K2” abuse which has never been reported in literature. The cause of acute renal failure is explained by intrinsic renal injury with high fractional excretion of sodium despite multiple episodes of vomiting and poor oral intake. Biopsy could have been done but other causes of acute kidney injury and bilateral echogenic kidneys were excluded by exhaustive work up. Use of synthetic cannabinoids marketed as “K2 or spice” has been on the rise as a legal alternative to marijuana due to its easy availability. Despite their alarmingly increased prevalence, very little is known about their clinical effects, metabolism and toxicology. The fact that these drugs are not easily detected by standard toxicology tests presents “K2” abuse a challenge to practicing physician. It should be of high suspicion especially in the young adult population when being evaluated for possible substance abuse.

MOUNT SINAI - JERSEY CITY (DOUGLAS RATNER, MD)

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LEFT VENTRICULAR SPONTANEOUS ECHO CONTRAST IN A PATIENT WITH SICKLE CELL DISEASE

Spontaneous echo contrast (SEC) is a smoke-like echocardiographic finding thought to be caused by a swirling pattern of blood flow that can be observed on tranesophageal or transthoracic echo, most often within the left atrium. SEC has been shown to be most strongly associated with left atrial appendage thrombus and increased risk of embolic events. We herein describe a case of a 20-year-old African American male with a history of sickle cell disease who presents to the emergency department complaining of a sudden onset of shortness of breath for one day. On physical examination the patient was found to have a pan-systolic murmur heard best at the apex, and radiating to the left axilla. An echocardiogram confirms the presence of a mitral regurgitation caused by a ruptured chordae tendineae of the anterior leaflet of the mitral valve and shows a “smoke-like” spontaneous echo contrast in the left ventricular cavity. The mechanism of production of intracardiovascular smoke-like echo is closely related to the formation of erythrocyte rouleaux. When rouleaux are formed, they become larger and appear nearer or larger than the ultrasonic wavelength. Thus reflections are produced and smoke-like echo appears. The increased rouleaux formation in sicklers along with the low flow and reduced shear forces within the left ventricle caused by the mitral regurgitation produced a smoke like appearance within the left ventricle in our patient. This phenomenon may be a predictor of an increased risk for developing thrombo-embolic events, where primary thrombo-prophylaxis appears to be a promising area worth studying in patients sickle cell disease.

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ARG VARIANT OF TP53 GENE POLYMORPHISM ARG72PRO INCREASES THE RISK OF DEVELOPING CERVICAL LESIONS IN MEXICAN WOMEN

Objective. To determine the association between the polymorphism Arg72Pro of TP53 gene and the risk of developing cervical lesions in patients with Human Papiloma Virus (HPV)-16 and / or 18 positive. Materials and methods. The study was carried out in 74 patients infected with HPV-16 and/or 18 with a cytologic and colposcopic diagnosis of a cervical intraepithelial lesion; as control group, 321 healthy hemo-donors ethnically matched were included, all of them, from Mexico. The viral genotype and allele and genotype frequencies of the polymorphism were determined by PCR. The results were analyzed with the statistical programs DeFinetti and STATAintercooled v11.1. netti and STATAintercooled v11.1. Results. 28 patients (37.8%) were positive for HPV-16, 29 patients for HPV-18 (39.2%) and 17 for both genotypes (23%). With regards to the polymorphism analysis, from the 74 HPV-positive patients, 1 (1.31%) was homozygous for the proline allele (P
Peripheral blood (for DNA analysis) was taken from the hemo-donors and after analyzing the polymorphism, it was observed that 12 women were positive for the P/P genotype (9.23%) and 25 men (13.1%). For the P/A genotype, 61 women were positive (46.9%) and 71 men (37.2%). With regards to the A/A genotype, 57 women were positive (43.87%) and 95 men (49.7%). Conclusions. Patients with the A/A genotype showed more susceptibility to HPV-16 and the development of high-grade cervical lesions, while patients with the P/A genotype showed increased susceptibility to infection with HPV-18 and the development of low grade cervical lesions. Complete list of authors (in order): Juan Jose Rios-Tostado, Jesus Salvador Velarde-Felix, Fernando P Gonzalez-Ibarra, Ignacio Osuna-Ramirez, Hector Samuel Lopez-Moreno, Joel Murillo Llanes, Marina Sanchez Leyva, Sylvia Paz Diaz Camacho, Jose Guadalupe Rendon-Maldonado.

Author: Basheer Tashtoush
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VANISHING LUNG SYNDROME IN A PATIENT WITH HIV INFECTION AND HEAVY MARIJUANA USE

Vanishing lung syndrome (VLS), also termed giant bullous emphysema, is a rare, distinct clinical syndrome that usually affects young men leading to severe progressive dyspnea and characterized by extensive, predominantly asymmetric upper lobe giant lung bullae. Case reports have suggested an additive role of marijuana use in the development of this disease in young male tobacco smokers. We report a case of a 65-year-old Hispanic male previously diagnosed with severe emphysema, HIV/AIDS, and a history of intravenous drug use of heroin and cocaine with active marijuana smoking, who presents with severe progressive shortness of breath over one week, and found to have multiple large subpleural bullae occupying more that 1/3 of the hemithorax on chest CT scan; characteristic of vanishing lung syndrome. The patient requires mechanical ventilation and later develops a pneumothorax requiring a chest tube placement and referral for surgical bullectomy. This case emphasizes the importance of recognizing VLS in patients with severe emphysema and a long history of smoking marijuana, as surgical bullectomy has shown high success rates in alleviating the debilitating symptoms and preventing the life threatening complication of this rare syndrome.

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HIP PAIN; A RARE PRESENTATION OF SYRINGOMYELIA

Neuropathic arthropathy is a type of chronic degenerative arthropathy which is associated with sensory loss, and loss of autonomic regulation at the involved joint. It is seen most commonly in diabetes mellitus, syringomelia, and tabes dorsalis. Shoulders and elbows are the most commonly affected joints by syringomyelia-induced neuropathy. Hip neuropathic arthropathy is a rare presentation in syringomyelia patients. We herein present a case of a 36 year old Hispanic male who presents to the emergency department complaining of right hip pain for two days with a history of a slowly progressive weakness in the upper and lower extremities over the last four years. MRI shows evidence of right hip joint articular surface destruction, suggestive of charcot’s arthropathy. Neurological examination reveals a pain and thermal sensory deficit in the upper torso posteriorly in a cape-like distribution, and signs of an upper motor neuron lesion in the upper and lower limbs. An MRI of the spine reveals a chiari I malformation with extensive syringohydromyelia of the cervical and thoracic spine.
JAK2 (V617F) and Factor V G1691A mutations in a patient with Budd-Chiari Syndrome

Myeloproliferative neoplasms (MPN) are considered an important risk factor for Budd-Chiari Syndrome (BCS). The current classification of MPN by the World Health Organization is based on the presence of JAK2 (V617F) somatic mutation, which is present in 29 to 34% of patients with BCS. Factor V Leiden mutation is found in around 53% of patients with BCS, representing the most common prothrombotic disease associated with the disorder. We describe a 48-year-old woman with a past medical history relevant for recurrent episodes of deep venous thrombosis in the left upper extremity and one episode in both lower extremities, one episode of transient ischemic attack, and essential thrombocythemia, who presented with jaundice, ascites and hepatomegaly. Budd-Chiari syndrome was diagnosed based on findings on doppler ultrasound and liver biopsy. Doppler ultrasound showed narrowness of hepatic veins and inferior vena cava in its hepatic portion, diffuse echotexture and portal hypertension. Liver biopsy showed congestion of sinusoids and portal fibrosis. The patient was found to be heterozygous carrier of Factor V and homozygous wild type G20210A prothrombin mutations. The JAK2 (V617F) mutation was detected by allele-specific PCR. The association of these mutations is rare with only a few cases reported in the literature. The patient was treated with oral anticoagulation and antiplatelet with good results and proper follow-up. In conclusion, due to the possible coexistence of multiple prothrombotic factors in patients with Budd-Chiari syndrome, the approach of these patients must be oriented in searching for multiple factors and should include the JAK2 (V617F) mutation. Complete list of authors (in order): Jesus Salvador Velarde-Felix, Jorge Sanchez-Zazueta, Jose Alberto Gonzalez-Valdez, Berenice Salcido-Gomez, Efren Gallardo-Angulo, Joel Murillo-LLanes, Fernando P Gonzalez-Ibarra.

MOUNTAINSIDE (RUTH C. WONG-LIANG, MD)

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A HEMODIALYSIS PATIENT WITH AN UNUSUAL ABDOMINAL FILM

A 47-year-old man who had been on hemodialysis for 3 years, and on Coumadin for hypercoagulable disorder presented with episodes of gross hematuria. Urological workup including abdominal plain x-rays are shown in picture 1 and 2, which revealed an incidental finding of multiple rounded hyperdense structures. The patient had no gastrointestinal complaints. He denied intake of any foreign body. No history of suicidal ideation. No history of abdominal surgery. Metallic foreign bodies were initially suspected. However, on reviewing his medications, lanthanum carbonate (Fosrenol 1000 mg tablet chew) 1000 mg twice daily was used as a phosphate binder to control his hyperphosphatemia. Patient has been swallowing, instead of chewing, the pills in the days around the admission. Subsequently he was instructed to chew the pills instead of swallowing them. He was discharged with no signs of bowel obstruction. Two weeks later, repeated abdominal XR/CT showed no more existence of the hyperdense structure. Lanthanum carbonate is a carbonate salt of the rare-earth metal lanthanum. This salt dissolves freely in the acid of the digestive tract, yielding ionic lanthanum, which has been shown to bind dietary phosphate released from food during digestion. This binding activity prevents the dietary absorption of phosphate in the intestines. Fosrenol is supplied as chewable tablets which should be chewed or crushed completely and be taken with or immediately following meals [1]. The diameter of Fosrenol (1000 mg) tablets is 22 mm. They are radiopaque with a density between that of bone and metal [2]. Thus the hyperdense structures retained in the colon in this case are probably the swallowed lanthanum carbonate pills (picture 1 and 2). Several previous case-reports [2,3,4] demonstrated abnormal abdominal films with high density material in ESRD patients which was suspected to be due to lanthanum carbonate ingestion. To our knowledge, this is the first report that a patient failed to chew the tablets which were shown intact in subsequent abdominal films days after ingestion. The rounded hyperdense structures can be easily misdiagnosed as other metallic foreign bodies, such as batteries and buttons. In addition to education of physicians regarding the radiopaque effects of lanthanum carbonate, patient and nursing staff education regarding the correct administration of the medicine should be emphasized. 1.

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CASE REPORT: 70YR OLD FEMALE WITH GRANULOMATOSIS WITH POLYANGIITIS PRESENTING AS RECURRENT OTITIS MEDIA

Granulomatosis with polyangiitis (GPA), formerly called Wegener’s granulomatosis, is a rare multisystem autoimmune disease. Hallmarks are vasculitis of the small to medium-sized vessels, geographic necrosis, and granulomatous inflammation, particularly in the airways. Most common sites of involvement are the upper respiratory tract, lungs, and kidneys. Ear manifestations may be unusually seen though reported, during the course of the disease, however, not as the initial presentation. The presence of anti-neutrophil cytoplasmic antibodies against proteinase-3 (PR3-ANCA) is highly sensitive and specific for the diagnosis. The early diagnosis and prompt treatment dramatically improves survival in this fatal disease. In this article, we report a case of GPA in a 70 year old female presenting with recurrent otitis media refractory to medical therapy, unintentional 15 lb. weight loss, and non-specific systemic symptoms as the unusual initial manifestations, with subsequent development of rapidly progressive glomerulonephritis.

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INAPPROPRIATE USE OF ACID-SUPPRESSIVE THERAPY IN PATIENTS ADMITTED TO A COMMUNITY HOSPITAL

The use of acid-suppressive therapy (AST), including proton pump inhibitors and histamine H2-receptor antagonists, for the prevention of stress ulcers has been well defined in the guidelines published by the American Society of Health-System Pharmacists in 1999. However, multiple studies have demonstrated the over-use of AST in both critical care and regular floor patients. This study is intended to assess the extent of over-use of AST in a 365-bed community hospital, and evaluate the possible side-effects and the subsequent financial cost. We also investigated into subgroups of patients who contributed the most to the over-use. We randomly reviewed three hundred patients’ charts who were admitted to the hospital ICU or regular medical-surgical floor in 2011. The results demonstrated that 61% of patients were started on AST upon admission, out of which 40% were inappropriately started on AST without a valid indication, which was 23% of all admissions. The average per-patient/admission drug cost of inappropriate use of AST was calculated to be $19, which can be translated to roughly $33,430 cost per year for this community hospital. Of note, nearly half of surgical patients (46%) were started on AST inappropriately upon admission. It is further noticed that 49% of patients on outpatient AST without a documented indication were continued on AST upon admission, for which the cost is unclear because of the incomplete record. Our study failed to detect an increased incidence of Clostridium difficile- associated disease and pneumonia among patients who were on AST compared to those who were not on AST, possibly as a result of the small sample size. The study results indicate that the over-use of AST is still significant in community hospitals. More education of physicians including surgeons regarding AST use, and more careful evaluation of continuing patients on outpatient AST upon admission could improve the AST over-use, and decrease drug cost for community hospitals.

Author: Yousuf Khan

GLUCOSE-6-PHOSPHATASE DEFICIENCY: MAINTENANCE OF EUGLYCEMIA AND FLUID BALANCE IN FASTING STATE

Glucose-6-phosphatase deficiency is a rare genetic disorder of glycogen metabolism predisposing to hypoglycaemia. Patients with this deficiency are dependent on continuous supply of glucose through frequent doses of oral cornstarch to maintain their blood glucose levels. When they are unable to eat or cannot eat, this plan needs to be altered to protect them from severe hypoglycaemia, lactic acidosis and seizures. The
Pregnancy during residency is common. One survey indicates that almost 44% of female residents will have their first child during residency. As per AAMC statistics the number of female residents in internal medicine increased from 38.5% in 1998 to 44.5% in 2008. Few studies on pregnancy during residency training in plastic surgery, family practice and obstetric gynecology have taken place but none in the Internal Medicine, the largest specialty. A 40 questions survey was sent to all the 300 Internal medicine residency program directors to be distributed to their female residents. All responses were anonymous. Only 48 responders completed the survey. Slightly more than half of the pregnancies were planned and most occurred in the 2nd or 3rd year of the training. Residents who became pregnant were more likely to be enrolled in the community hospital program than an academic medical center; 69% vs. 31%. Geographically, 46% of the responders were from Northeast, 19% from the Midwest, 19% from West and 16% were from South. Average age of the responder was 31 years old. In terms of pregnancy outcome, 31% reported some complications; 15% low birth weight, 15% post maturity vs. 10% prematurity, 10% anemia and 10% infection, gestational diabetes was 5%, spontaneous abortion 5%, Preeclampsia 5% and bleeding 5%. Residents who had prior pregnancies reported feeling more tired; 40% with the same number reporting less sleep and average number of sleep was 6 hours. The effect of pregnancy on residency training prolonged the length of training on only 31% or approximately 1/3 of these reported extended training to comply with ACGME requirements reported an additional 1 to 2 months and the remaining 1/3, less than 1 month. About 2/3 pregnant residents reported modified behavior in their first child during residency. As per AAMC statistics the number of female residents in internal medicine increased from 38.5% in 1998 to 44.5% in 2008. Few studies on pregnancy during residency training in plastic surgery, family practice and obstetric gynecology have taken place but none in the Internal Medicine, the largest specialty. A 40 questions survey was sent to all the 300 Internal medicine residency program directors to be distributed to their female residents. All responses were anonymous. Only 48 responders completed the survey. Slightly more than half of the pregnancies were planned and most occurred in the 2nd or 3rd year of the training. Residents who became pregnant were more likely to be enrolled in the community hospital program than an academic medical center; 69% vs. 31%. Geographically, 46% of the responders were from Northeast, 19% from the Midwest, 19% from West and 16% were from South. Average age of the responder was 31 years old. In terms of pregnancy outcome, 31% reported some complications; 15% low birth weight, 15% post maturity vs. 10% prematurity, 10% anemia and 10% infection, gestational diabetes was 5%, spontaneous abortion 5%, Preeclampsia 5% and bleeding 5%. Residents who had prior pregnancies reported feeling more tired; 40% with the same number reporting less sleep and average number of sleep was 6 hours. The effect of pregnancy on residency training prolonged the length of training on only 31% or approximately 1/3 of these reported extended training to comply with ACGME requirements reported an additional 1 to 2 months and the remaining 1/3, less than 1 month. About 2/3 pregnant residents reported modified behavior in particular. 81% reported limiting contact with patients who had infections and 33% reported limiting their participation in resuscitation and 11% reported limited performing invasive procedures. Missed worked time occurred in only 10% of the reporting residents. The missed work days ranged from less than 2 to more than 6 with 46% reporting less than 2 work days; 6% reported missed calls and of these 80% missed less than 2 calls and 20% missed more than 6 calls. Nearly ½ of the residents reported reading less and 8% of residents felt that their pregnancy adversely effected their board scores. Program directors were viewed by responders to be supportive in 88% percent of the programs. Amazingly, 42% of the respondents said that their program lacked a formal maternity leave policy and 85% reported not reviewing the existing maternity leave policy. Fellow residents were perceived to be supportive about 90% of the time. The majority of pregnant residents (69%) would not recommend pregnancy during residency training.
ACUTE PANCREATITIS IN SICKLE CELL CRISIS

Sickle cell disease is an inherited disorder due to homozygosity for abnormal hemoglobin HbS manifested by chronic hemolytic anemia and microvascular occlusion during the crisis. Abdominal pain in sickle cell patients can pose a diagnostic challenge. It is an important component of painful vasoocclusive crisis (VOC) and can mimic acute appendicitis and acute cholecystitis. Acute pancreatitis can also rarely be caused by vasoocclusive crisis. A 33 year old African American Male with Sickle Cell disease presented with back pain and abdominal pain associated with nausea and vomiting for 2-3 days. Back pain was similar to his usual VOC pain. His surgical history included a cholecystectomy 12 years prior to the admission. He had no previous history of pancreatitis, and no history of excessive alcohol intake. His home medications included Acetaminophen-Oxycodone, Hydroxyurea, Methadone, folic acid, amlodipine and lisinopril. Physical exam showed diffuse mild tenderness of the abdomen. His laboratory data included white blood cell of 11.6 &times; 10^9/L, Hemoglobin of 5.4 g/dL, Platelet of 272,000/μL, reticulocyte count of 12.3%, amylase of 1185 U/L, lipase of 1538 U/L, Ca of 9.2mg/dL, AST of 29 U/L and Creatinine of 1.88 mg/dL. An abdominal obstructive series showed no obstruction. CAT scan of the abdomen showed acute pancreatitis. MRCP revealed normal caliber of common bile duct without any stones. A fasting lipid panel was within normal limits. His hospital course was complicated by hypotension, metabolic acidosis and respiratory distress requiring admission to the ICU. Treatment during his hospitalization course included transfusion of a total of 8 Units of blood and supportive therapy. His condition improved significantly, abdominal pain resolved and lipase trended down. He had one episode of recurrence five months later. Acute pancreatitis secondary to sickle cell disease is a diagnosis of exclusion. It is a relatively rare occurrence, with only six case reports published previously. It is thought to occur secondary to ischemia due to microvascular occlusion. Clinical findings and laboratory data are similar to acute pancreatitis secondary to other causes. Reversal of microvascular ischemia is the key for successful treatment in these cases. The therapeutic role of exchange transfusion versus simple blood transfusion remains less clear. Abdominal pain in sickle cell patients with VOC can be a common presenting feature. Acute pancreatitis is important to consider early on, and might be difficult to distinguish clinically from nonspecific pain due to VOC. Early recognition and prompt treatment can prevent complications of pancreatitis and may reduce morbidity and mortality.

THE RISK OF UTILIZING EEG IN THE NEUROPROGNOSTICATION OF POST CARDIAC ARREST INDUCED HYPOTHERMIA

Introduction: The utilization of induced hypothermia and emergent coronary intervention after cardiac arrest has led to improved outcomes in the last decade. Despite treatment benefit, the guidelines regarding neurological assessment are ill-defined and premature prediction of poor prognosis may be a risk. Case Description: An 81 year old Caucasian male with a history of hypertension, poorly controlled diabetes, dyslipidemia, history of recent coronary angioplasty with stent placement for a non-ST elevation myocardial infarction one week prior presented via EMS for lethargy and confusion. Upon arrival patient was found to be in cardiac arrest with pulseless electrical activity. CPR was initiated and, with return of pulse, hypothermic protocol was initiated with emergent coronary intervention. In the interim pertinent laboratory findings revealed potassium of 7.2, glucose of 1167 and lactic acid of 6.1. Coronary angiography revealed no occlusion or stent thrombosis, and patient was placed in induced hypothermia. The etiology of cardiac arrest was assumed to be an arrhythmic event due to hyperkalemia and diabetic ketoacidosis. Patient was transferred to the cardiac care unit for continuation of induced hypothermia, where a forty-eight hour video electroencephalogram (EEG) was utilized with frequent neurological examination. The EEG revealed low voltage activity. Neurological examination forty-eight hours after normothermia revealed no gag reflex, minimally reactive pupils with 8mm dilation and a Glasgow coma score of 3. At this point the medical team conveyed to the family the patients poor prognosis, and they decided to issue a "Do Not Resuscitate" order, with plans to withdraw care within three days. Forty-eight hours later (96 hours post normothermia), spontaneous movement of the lower
extremity followed by spontaneous eye opening was noted. Repeat video EEG again showed low voltage activity consistent with diffuse encephalopathy. Despite the EEG results, withdrawal of care was reversed and supportive management continued with daily improvement in neurological status. Ultimately, the patient recovered without any neurological deficit and was discharged home. Discussion: Induced hypothermia as an adjunct to emergent coronary intervention in cardiac arrest has clearly shown benefit in neurological outcome. However, this case underscores the limits of video EEG in prognostication at forty-eight hour and ninety-six hour post normothermia. Only a thorough neurological examination can account for the effects of hypothermia, sedatives and variations in neurological recovery. The results of low voltage activity on video EEG, as exemplified in this case should not contribute to patient prognosis or family decision to withdraw care. Current guidelines are not sufficient in prognostication of neurological recovery and further studies must be pursued to avoid unnecessary death.

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MYELOID SARCOMA: AN UNUSUAL CAUSE OF SPINAL CORD COMPRESSION

Introduction: Myeloid sarcoma, also termed granulocytic sarcoma or chloroma, is a rare malignant solid tumor resulting from the extra medullary proliferation of myeloblasts. Myeloid sarcoma may involve any organ, but it is most commonly described in the skin, bone, mediastinum and lymph nodes. The involvement of the central nervous system as a first manifestation of myeloid sarcoma is rare, and spinal cord compression is even rarer. Here we report a case of myeloid sarcoma with a posterior medistinal mass with extradural compression of the thoracic spine. Case presentation: An otherwise healthy 37 year old female presented with right sided chest pain. On CT scan evaluation she was found to have a posterior medistinal mass. She underwent a CT guided core needle biopsy but biopsy results were inconclusive. The specimen was then sent to an outside lab for review. While awaiting results of the biopsy the patient developed bilateral lower extremity paraesthesia and urinary retention. MRI of the Thoracic spine revealed the presence of a 10 cm posterior medistinal mass, which infiltrated into the spinal canal at T2-T5, resulting in spinal cord compression. She underwent emergency laminectomy with resection of the extradural tumor. Excisional biopsy of this mass was consistent with myeloid sarcoma. Cells were medium size with scant cytoplasm and irregular contours. Markers were positive for CD 45, CD 99, CD 117, CD 34, TDT, CD 43 and myeloperoxidase. B and T cell markers were negative. The patient’s Blood counts and peripheral smear were normal. Bone marrow studies revealed focal involvement by acute myeloid leukemia. Cytogenetic studies reveal t (8:21). The Patient underwent induction chemotherapy with cytarabine and idarubicin. She also received local radiation therapy, and subsequently consolidation chemotherapy. Discussion: Myeloid sarcoma is a rare disease and is associated with a few clinical scenarios; as a localized tissue mass in a known case of AML, as a sign of impending blast crisis in CML, a leukemic transformation in MDS, or as a forerunner of AML in nonleukemic patients. In the absence of overt leukemia, myeloid sarcoma is rarely considered in the differential diagnosis of a soft-tissue mass, and it may be misdiagnosed as a number of other neoplastic diseases. The critical factors for detecting this rare entity would include an acute awareness of this disease, active coordination between clinicians and pathologists and the application of special stains to detect the myeloid origin. The mainstay of treatment of myeloid sarcoma is treating the underlying leukemia. Most tumors, whether detected prior to or during the therapy of leukemia, respond well to standard chemotherapeutic agents. Evidence of residual tumor may be an indication of surgery or radiation therapy.
NEUROLEPTIC MALIGNANT SYNDROME IN THE CLOAK OF ACUTE CORONARY SYNDROME

Neuroleptic Malignant Syndrome (NMS) in the cloak of Acute Coronary Syndrome: Shilpi Singh, M.D., M.P.H., Raritan Bay Medical Center, Perth Amboy NJ-08861 Neuroleptic malignant syndrome (NMS) is a life threatening neurologic emergency seen with the use of neuroleptic agents, and also due to withdrawal of anti-parkinson medications, characterized by constellation of symptoms including altered mental status, fever, rigidity, and dysautonomia. Mortality rate are very high if left untreated or if there is delay in the diagnosis of NMS. It is therefore essential to recognize the various atypical presentations of NMS. A 49 year old female with past medical history of diabetes mellitus type 2, hypertension and schizoaffective disorder presented to the emergency room with shortness of breath and fever for 2 days, with symptoms worsening in the last 2 hours, associated with chest pain. The chest pain was not relieved by anything and was reported to be radiating to the left arm and jaw. The EKG showed marked ST elevations in the inferior leads and, the patient was sent for emergent cardiac catheterization. She was found to have 99% occlusion of proximal RCA, and 90% occlusion of first obtuse marginal artery. Balloon dilatation of the coronary arteries was done and the first drug eluting stent was placed in the proximal RCA. At that point the laboratory test results showed leukocytosis of 28000, and further cardiac intervention was aborted without placing the second stent in the light of suspected sepsis. The patient was transferred to ICU, where she continued to have chest pain despite the recent intervention and improved blood flow in the coronaries. Shortly after transfer, the patient was diagnosed with NMS with the following positive criteria: fever of 102 degrees Fahrenheit, muscular rigidity, altered mental status, WBC count >28 000, autonomic instability with blood pressure of 90/46, elevated CK levels (1343), with the risk factor of neuroleptic medication (risperidone) use. Risperidone was discontinued, and she was treated with bromocriptine, lorazepam and ample IV fluids. Her symptoms slowly resolved over a period of two weeks and she was discharged from the hospital. She followed with the cardiology for evaluation of further coronary intervention in light of recent incomplete revascularization. This case shows the importance of recognition of atypical presentation of NMS, and the need for high suspicion in patients on neuroleptic medications. Early recognition of NMS is critical in management and preventing potential related and unrelated complication, as in this case, the incomplete cardiac intervention.

SPLENIC INFARCTION CAUSED BY AN UNEXPECTED CO-INFECTION OF EPSTEIN-BARR VIRUS, CYTOMEGALOVIRUS AND MYCOPLASMA PNEUMONIAE

INTRODUCTION: Epstein-Barr virus (EBV), cytomegalovirus (CMV) and Mycoplasma pneumoniae are common infectious pathogens among children and young adults. Although single infection of one of these pathogens is common enough, their co-infection has been rarely reported. CASE PRESENTATION: A 19-year-old woman presented with severe upper abdominal pain for 5 hours. She reported feeling sick in the past 2 to 3 weeks, having fatigue, sore throat, fever, chills, nonproductive cough and jaundice. On examination, she was febrile, tachycardiac and tachypneic. Icteric sclera and diffuse upper abdominal tenderness on palpation were noted. Blood work showed pancytopenia (hemoglobin 9.4 g/dL, WBC 2,600/mm3 and platelet 90,000/mm3), high reticulocyte index (3.12%), atypical lymphocytes (28%), increased LDH (583 U/L), low haptoglobin (< 20 mg/dL) and abnormal liver function tests (total bilirubin 1.7 mg/dL, direct bilirubin 1.1 mg/dL, ALT 193 U/L and AST 324 U/L). The chest x-ray was unremarkable, but the CT of the abdomen showed enlarged paraaortic lymph nodes and one wedge-shaped focal hypodensity at the periphery of the mildly enlarged spleen, indicative of splenic infarction. Infection by virus/atypical organism was suspected. M. pneumoniae IgM was found positive, but the HIV screening test, monospot test and hepatitis viral serology were all negative. Azithromycin was promptly given, but her fever and abdominal pain persisted. A high suspicion of infectious mononucleosis prompted further serology tests, which reported positive CMV IgM, EBV D-early antigen IgG and capsid antigen IgM as well as negative CMV IgG and EBV nuclear antigen IgG. Acute co-infection of CMV and EBV with M. pneumoniae was diagnosed, and supportive care was given. By hospital day 7, her fever and abdominal pain had subsided. On hospital day 10, her liver function became much improved; her WBC and platelet count were normal and hemoglobin was stable. DISCUSSION: EBV/CMV
infection can cause transient immunosuppression and may lead to superinfection of M. pneumoniae or other organism(s). Similar to single infection, co-infection of EBV, CMV and M. pneumoniae caused fever, sore throat and abnormal liver function, but to a more severe degree. Some rare complications, such as splenic infarction and pancytopenia, were also observed here. Severe abdominal pain in mononucleosis often raises suspicion for splenic rupture, but splenic infarction has rarely been reported. In a retrospective study, it was reported that 6% of the patients with splenic infarction had acute EBV/CMV infection, suggesting mononucleosis as an important cause. Diagnosis of multi-pathogen infection requires a high suspicion in patients presented with unusual or more severe symptoms. When mononucleosis is highly suspected but monospot test (81-95% sensitivity) is negative, more sensitive tests that measure EBV/CMV specific antibodies/DNA should be performed next. A greater awareness among clinicians will help to properly diagnose and treat this under-recognized co-infection of multiple respiratory pathogens.

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A PUZZLING ELEVATION OF SERUM AMYLASE

Introduction: Macroamylasemia is a benign condition characterized by persistently elevated serum amylase. It is caused by binding of amylase to large complex molecules (e.g., immunoglobulin or polysaccharide), which leads to a decreased renal clearance with the consequence of prolonged half-life of serum amylase. As measurement of serum amylase is commonly used to screen for pancreatitis in patients who present with acute abdominal pain, macroamylasemia could be mistaken as having pancreatitis. Case Presentation: A 42-year-old female presented with epigastric pain, nausea and vomiting for 1 day. She reported a past history of cholecystectomy, appendectomy, childhood-onset type I diabetes, and recurrent DVT in association with heterozygous prothrombin G20210A mutation. Medications included lantus, humalog and coumadin. On examination, mild tenderness on palpation was noted at the epigastric region. Because pancreatitis was considered, serum amylase and lipase were ordered and reported to be 2,292 units/L (normal: 60 to 180 units/L) and 33 units/L, respectively. Due to abnormal serum amylase, she was admitted for further evaluation. The patient was kept NPO with intravenous fluids administration, her symptoms did not improve. Her serum amylase was persistently elevated, while the serum lipase remained normal. The CT of the abdomen failed to find any evidence of pancreatitis. Upper endoscopy revealed moderate to severe esophagitis and gastritis, with no H. pylori infection on pathological examination. Her condition gradually improved with PPI therapy. Further investigations revealed a pancreatic/salivary amylase isoenzyme ratio of 0.33 and a urine amylase clearance/creatinine clearance ratio of 0.14%, suggestive of macroamylasemia. Presence of macroamylase was confirmed by measurement of monomeric/total amylase ratio after serum ultrafiltration. Over the following 1 year, her serum amylase remained persistently elevated (ranging from 1,275 to 2,668 units/L) with normal serum lipase. Further laboratory tests, including rheumatoid factor, ANA, anti-dsDNA and anti-gliadin peptide antibodies, were all negative. Discussion: Macroamylasemia is estimated to be present in 0.98% of healthy persons. Because macroamylase does not cause any symptom, most of the cases were found incidentally in the workup of acute abdominal pain and could be mistaken as pancreatitis. Serion lipase is helpful to differentiate macroamylasemia from pancreatitis, with the former always having normal serum lipase. As macroamylases are large in size and cannot be filtered through the glomerulus, measurement of urine amylase clearance/creatinine clearance ratio helps make the diagnosis. A ratio of less than 1% is consistent with macroamylasemia. Diagnosis can be confirmed by measurement of monomeric/total amylase ratio in serum. Macroamylasemia does not require any treatment, although it has been reported to be associated with several diseases, including rheumatoid arthritis, SLE, celiac disease and diabetes. Clinicians should be aware of this condition in the differential diagnosis of hyperamylasemia to avoid unnecessary workup of pancreatitis.
AN ATYPICAL CASE OF ACUTE MYOCARDIAL INFARCTION LINKED

Psychiatric patients may develop NMS when taking Neuroleptic medications. Current literature indicates a mortality rate of 10-20% in NMS alone, which significantly rises if linked to acute MI. However, very few reports have been documented regarding this association. We report a case of NMS causing acute MI due to its rarity and great educational contribution. Case Presentation: 49 year old female with a significant history of Schizophrenia and Renal Insufficiency presented to ED with 2-day history of SOB aggravated by a new onset of high grade fever for the last two hours. Consecutively, she developed severe tightening chest pain with radiation to the left arm accompanied with marked sweating. She also reported feeling progressive stiffness throughout her body. She has no history of CAD or pertinent risk factors. Medications included Risperidone, Trazodone, Aripiprazole and Benztropine. In the ED she had a BP 104/60 mmHg, HR 88 bpm, RR 23/min, T 101.1F and SO2 95% [3L NC]. Physical exam showed marked diaphoresis and limited range of motion in all extremities. EKG revealed acute inferior STEMI and underwent emergent Cardiac-Catheterization which revealed > 90% RCA occlusion and consequently stented. Patient remained markedly febrile with mild generalized stiffness for which broad-spectrum antibiotics was empirically initiated. Upon admission to ICU, patient became tachypneic, lethargic, diaphoretic and had clear chest on auscultation with BP 97/46 mmHg, HR 156 bpm regular, SO2 90% [3L NC], T 103.4F but no chest pain. Fever persisted in spite use of antibiotics and Tylenol. ABG’s showed Respiratory Alkalosis, Urine/Blood cultures and Urine Drug Screen were negative, Glucose normal, Calcium was 8.5 and CXR was clear. CPK level and WBC increased from 98 to 1343 and 28.5 to 40.7 (3-4 hrs), respectively. Renal function deteriorated further. Post-Cath repeat EKG was unremarkable. At that time, she developed generalized “lead pipe rigidity” compromising her normal breathing along with autonomic instability. NMS was immediately identified and treated with aggressive IV hydration, Bromocriptine and Benztropine (Dantrolene unavailable). Neurology/ Psychiatric consults were placed. Patient was closely monitored and improved gradually; body stiffness and breathing improved without the need for intubation. She regained full motion of all extremities, renal function improved, cultures continued to be negative, CPK trended down to 733 and finally she became stable (HR 110 bpm, RR 20, SO2 99% [2L NC] and T 98F). Discussion: While clinically rare, NMS should receive high consideration within the differential of a patient on Neuroleptic medications who presents with high fever, muscle rigidity and autonomic instability. Even though unusual, NMS can trigger the onset of an acute MI in the presence of severe chest pain. Since mortality increases tremendously in this clinical scenario, early recognition helps physicians to diagnose and manage this life threatening neurologic emergency.

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AN UNUSUAL CASE OF HYPEREOSINOPHILIC SYNDROME (HES)

Currently a rare condition, Hyperesinophilic Syndrome is considered a myeloproliferative disorder which may ensue into fatal multi-organ damage. Often asymptomatic or clinically vague, this syndrome results from a persistent Eosinophilia. We report a case of HES due to its unique and valuable educational contribution. Case Presentation: 45 year old male and recent immigrant from Dominican Republic with no significant past medical history, presented with six week history of non productive cough, fever, nasal congestion, and myalgia. Patient reported no relief with over the counter medications and initial physical examination was otherwise normal. Laboratory workup revealed a WBC of 25,000 µ/L and a significant Eosinophilia of 20,500 Cells/mm3, which further increased from 50% to 69% following admission. In addition, Hemoglobin and Hematocrit were normal as well as the rest of the basic metabolic panel. Further testing showed elevated ESR (57 mm/hr) and IgE levels (> 1,600 IU/mL), but normal IgA levels. Cultures for blood and urine resulted negative. Analysis for stool and sputum showed no Ova, Parasites or any other organisms. Dengue, Strongyloid, TST and HIV showed negative serologies including a negative blood smear for Malaria. Furthermore, nasal secretions were negative for Influenza Antigen. Imaging testing illustrated atypical pneumonia and mastoid disease on CXR and Head-CT scan, respectively. Upon admission to the regular floor, the patient was treated for community
acquired pneumonia using IV Unasyn® and given respiratory treatment with Albuterol. On third day of stay, patient developed severe respiratory distress needing immediate transfer to the ICU. He was promptly treated with IV steroids and responded positively. In consideration of persistent unexplained Eosinophilia, Transbronchial biopsy, Bronchioalveolar Lavage and Duodenal Aspirates were performed, which tested negative for ova, parasites or malignancy. Consecutively, peripheral blood smear showed marked eosinophilia with no evidence of atypical or malignant cells. After numerous inconclusive studies to determine the etiology of eosinophilia, diagnosis for HES was met by exclusion criteria. After 6 days of IV steroid therapy, WBC as well as Eosinophil count decreased to normal ranges. He continued to be asymptomatic and was followed up for a period 6 months without any complications. Discussion: According to literature, HES manifests with eosinophilia &gt;1500/µL on two occasions without any recognizable etiology. Incidence of HES is estimated at 0.5-1.0 cases per 100,000 persons per year and is 9-fold higher in men than women. Usually asymptomatic, HES is incidentally detected on a CBC performed for other purposes. However, HES could be fatal as a result of multi-organ failure if clinically missed. Physician’s recognition of this syndrome accompanied with prompt diagnosis and management could decrease mortality in this seldom identified condition.

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ACUTE RETROVIRAL SYNDROME-A MAJOR DIAGNOSTIC DILEMMA

Introduction: Detection and treatment of acute retroviral syndrome remains a major clinical challenge. Early detection and therapy determines the risk of disease progression and transmission. We report a rare case of acute HIV infection and its diagnostic dilemma. Case presentation: A 23 year old male was admitted to hospital for evaluation of fever, abdominal bloating and diarrhea for 3-4 days. The patient had been well about three weeks before hospitalization when he had recently gotten back after spending his vacation in Bermuda Island where he visited a mountainous area and had worked in the garden picking few vegetables. One week after his return to home he had mild joint and muscle aches without runny nose or sore throat. He did not remember being bitten by ticks, mosquitoes or bugs during his stay in the island. Two weeks later he started having abdominal bloating and 3-4 episodes of loose watery diarrhea without blood and mucus. He did not eat anything unusual and had no nausea or vomiting. He noticed mild rash on his forearms and dry cough since four days. He had fever (38.4C) but denied chills, night sweats, sore throat or swollen glands in his neck and other parts of the body. He reported decreased appetite for the past 2-3 days but no weight loss. He smoked half pack per day for five years and was sexually active with single male partner (who had no known illness). He was hemodynamically stable, without signs of distress and pertinent physical examination was unremarkable except for small area of macular rash on both his forearms. He had leucopenia (3,200/mm3), lymphopenia(600/mm3) and thrombocytopenia (69,000/mm3) on admission. His routine EKG and CXR were normal. Stool examination revealed Giardiasis. The rapid HIV test done on admission was negative. Serologies for Dengue, RMSF, Ehrlichia species, Haantavirus were done and were negative. The cell counts persisted to be low with profound lymphopenia and also neutropenia. Owing to high index of clinical suspicion P24 assay and HIV PCR were done and were positive for HIV-1 (viral load=7.2 million copies/ml). Patient was counseled thoroughly and after PhenoSense-GT testing was started on enfuvirtide for four weeks with concomitant ritonavir boosted darunavir, tenofovir and emtricitibine. Discussion: Standard diagnostic testing for HIV with screening ELISA followed by confirmation on western blot and CD4 count and viral load (if positive ELISA) is not applicable when the clinical picture and epidemiological information raises high suspicion of acute retroviral syndrome. If the suspicion for acute retroviral syndrome is strong, one should proceed with viral load (PCR) and p24 antigen capture assay despite negative HIV ELISA. The 4th generation tests which detect both antibodies against HIV and capsid p24 antigen will obviate the diagnostic dilemma.
FACTORS AFFECTING OUTCOME OF CARDIOPULMONARY RESUSCITATION AT RARITAN BAY MEDICAL CENTER

Abstract Factors affecting outcome of cardiopulmonary resuscitation at Raritan Bay Medical Center. Authors: Beena Syed, MD, Daniel Storms BA, Mohammad Islam, MD, Linda Lopazanski, RN, Josephus Sanjorjo, MD, Natesh Sharma, MD, Hassan Alazzawi, MD, Reddy Candula, MD, Li Young, MD, and Abdalla Yousif, MD.

Introduction: Cardiopulmonary arrest occurs in a minority of inpatients, and its occurrence is associated with a very high risk of pre-discharge mortality. Code blue, or cardiopulmonary resuscitation is called when a patient is identified in cardiopulmonary arrest. The objective of this study was to identify factors affecting the outcome of code blue.

Methods: All code blue calls initiated during 2008-2010 on patients 18 years of age and older at Raritan Bay Medical Center were included. In case of more than one call on same patient within 24 hours, the outcome of final call was included. Calls initiated in the emergency room or cancelled before arrival of the team were excluded. Data was stratified according to age, service, and location i.e. Intensive Care Unit (ICU) vs. non-ICU. Statistical significance was determined by the Chi-square test. Results: Successful outcome of code blue was higher in non-ICU patients compared to ICU patients (38% successful in ICU vs. 66% successful in non-ICU, p<0.01). There was no difference between admitting service (44% successful in resident service vs. 53% successful in private service, p= 0.24) or age (48% successful if ≤65 years old vs. 52% successful if >65 years old, p=0.63).

Conclusion: Patients in non-ICU setting have a higher chance of successful outcome if a code blue is called vs. patients who are in the ICU. We found no association between age and whether the patient was a private patient or a resident service patient. The higher success in non-ICU setting could be attributed to the fact that patients in ICU were sicker than patients on the floor, and the staff in ICU is better able to identify true cardiopulmonary arrest.

INTRODUCTION There has been refuting evidence regarding the risk of pancreatitis with statin use. We report a rare case of recurrent acute pancreatitis and rhabdomyolysis associated with statin use. CASE REPORT A 68-year-old female was admitted to hospital with a one day history of severe epigastric pain radiating to the back associated with severe myalgias and lethargy but no nausea or vomiting. On examination, there was epigastric tenderness without rebound or guarding. Bowel sounds were normal. The remainder of the physical examination was normal. Her past medical history was remarkable for hypertension (on valsartan), diabetes mellitus type 2 (on metformin), coronary artery disease (on Aspirin and Clopidogrel), and hypercholesterolemia (managed with simvastatin). She had been taking simvastatin 40 mg PO daily since 3 years. There is no history of alcohol use, gallstones, abdominal trauma, or hypertriglyceridemia. There is no family history of pancreatitis. On admission pulse 67/min, BP 128/70, RR 14/min, temperature 37.2 C. Laboratory data showed white blood cells 12,300/mm3, Lipase >2500 U/L, Amylase 1221 U/L, ALT 292 U/L, AST 287 U/L, and CPK 10,606 IU/L. Plasma glucose, BUN, creatinine, alkaline phosphatase, bilirubin, calcium, and lipid profile were normal. Additionally, urine analysis revealed moderate amount of blood on dip-stick without microscopic hematuria. Computed tomography showed acute pancreatitis. No gallstones or common bile duct abnormalities were demonstrated.. Patient was treated with IV fluids and bowel rest and all medications were withheld. A right thigh muscle biopsy was performed revealing scattered regenerating fibers consistent with primary myopathy. Patient recovered completely with supportive care and was discharged home on his previous medications. Unfortunately patient was readmitted with similar complaints and workup showed recurrent acute pancreatitis and rhabdomyolysis. After initial supportive care the statin therapy was discontinued and continued follow-up showed no further symptoms and complete normalization of creatine kinase levels. DISCUSSION Statin therapy has been associated with pancreatitis in observational studies. However several pooled analysis of randomized trial data have showed refuting evidence with lower risk of pancreatitis in patients on statin therapy. Statin-induced acute pancreatitis is uncommon and only few cases
have been reported thus far. In our case, the patient was treated with simvastatin for 3 years prior to admission. The exclusion of common etiological factors along with no family history of pancreatitis and recurrence of rhabdomyolysis and acute pancreatitis with reintroduction of statins strongly suggest causal relationship of statins and acute pancreatitis with concurrent rhabdomyolysis. With increasing amounts of patients being prescribed statins, the importance of appreciating its role in acute pancreatitis and rhabdomyolysis becomes essential. In the absence of obvious etiology, clinicians should consider statin-induced acute pancreatitis when the clinical presentation and history warrant such suspicion.

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PRIMARY HYPERALDOSTERONISM: A CLASSICAL CASE WITH PRINCIPLES OF EVIDENCE-BASED MANAGEMENT

We present the case of a forty-seven year old patient whose chief complaint of sudden severe right sided scapular pain would require admission for control and further work-up of severe hypertension (BP 225/134 mmHg). Physical examination revealed a thin Hispanic female with no point tenderness but evidence of chronic hypertensive retinopathy and hypertensive heart disease. Preliminary investigations found spontaneous hypokalemia, borderline alkalosis, volatage criteria for left ventricular hypertrophy on electrocardiogram and a mitalized left heart border on chest X-ray. Subsequent screening by measuring plasma aldosterone concentration/plasma renin activity (PAC/PRA ratio) suggested primary hyperaldosteronism (PAC/PRA - 31:1). Confirmation testing was done using the saline infusion test. Localization of the source of autonomous aldosterone production was achieved via adrenal venous sampling. Elevated cortisol-corrected aldosterone ratios coincided with a right sided adrenal mass noted incidentally on prior CT angiogram done to rule out aortic dissection. The patient was scheduled for laparascopic right-sided adrenalectomy. Evidence-based work-up is essential to diagnose Primary Hyperaldosteronism even in the presence of an incidental adrenal mass. Recent prevalence estimates of Primary Hyperaldosteronism at 5-15% among 65 million U.S. hypertensive patients warrants a high index of clinical suspicion for this potentially curable disease.

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ANEURYSM OF PANCREATICODUODENAL ARTERY IN A PATIENT WITH CELIAC ARTERY COMPRESSION SYNDROME

Introduction: Celiac artery compression syndrome (also known as median arcuate ligament syndrome or Dunbar syndrome) is an uncommon disorder with a characteristic triad of postprandial abdominal pain, weight loss and abdominal bruit. Pancreatic duodenal artery aneurysm associated with celiac artery compression syndrome is extremely rare. We report a patient who presented with retroperitoneal hematoma without any preceding events or symptoms; the patient was diagnosed with a pancreaticoduodenal artery aneurysm associated with celiac artery stenosis. Patient Presentation: A 69-year-old female with a past medical history of hypertension and angina presented with dizziness, abdominal pain and syncopal episodes. The patient was found to be hypotensive and anemic. Computed tomography (CT) scan of the abdomen and pelvis with contrast revealed a large retroperitoneal hematoma (approximately 10 X 4 X 20 cm) with associated high-grade celiac artery stenosis and a 7mm aneurysm of the pancreaticoduodenal arcade. No extravasation of contrast was seen. The patient had an urgent nuclear bleeding scan, which did not detect active bleeding. After she was hemodynamically stabilized with aggressive fluid resuscitation and transfusion, the patient underwent elective mesenteric angiogram and the aneurysm was successfully embolized. The patient's hemoglobin remained stable after the coil embolization. She was discharged one week after the event, with elective celiac decompression surgery scheduled for the near future. Discussion: Celiac artery compression syndrome, first
introduced in 1967 by Dr. Dunbar, is an uncommon disorder believed to result from changes in the relationship between the aorta and the arcuate ligament over a prolonged period of time. Color Doppler sonography may reveal increased flow velocity during deep expiration, and Multidetector-row CT (MDCT) is considered the most appropriate modality to diagnose celiac artery compression syndrome. Aneurysms of the pancreaticoduodenal artery are extremely rare in celiac artery compression syndrome patients, with a reported incidence of about 3%. Most pancreaticoduodenal arteries aneurysms range in diameter from 8 to 30mm, however size does not predict risk of rupture. Due to the rich collaterals between the superior mesenteric artery and the celiac artery, some patients with celiac artery compression syndrome remain asymptomatic, which makes diagnosis more difficult. Pancreaticoduodenal artery aneurysm associated with celiac artery compression can be treated with embolization or surgery. Median arcuate ligament (MAL) compression is traditionally relieved with invasive surgery; however, laparoscopic MAL release, first introduced in 2000, has demonstrated success and is associated with reduced morbidity and shorter hospital stays. As such, it may be the treatment of choice for MAL compression.

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SUSTAINABILITY OF A PHARMACIST-MANAGED ANTICOAGULATION PROGRAM IN AN OUTPATIENT MEDICAL TEACHING PRACTICE

Background: Warfarin therapy requires close monitoring with frequent international normalization ratio (INR) evaluations. A previous review of data from the Internal Medicine Faculty Practice (IMFP) demonstrated that the institution of a pharmacist-managed, evidence-based, multidisciplinary anticoagulation program led to statistically significant increases in the percentage of INR values within target range. The purpose of our study was to evaluate the sustainability of INR target attainment during the last two and half years that the pharmacist-led monitoring program has been in place. In addition, we sought to evaluate the number of hospital admission related to bleeding events and frequency of vitamin K administration during this period of time. Methods: A retrospective evaluation was conducted that included all patients treated with warfarin at the IMFP between August 1, 2009 and January 31, 2012. Warfarin indication, INR values, INR target range, average time-in-therapeutic range, vitamin K administration and hospital admission related to bleeding events were extracted and assessed. Bleeding events were defined and graded. Results: During the study period a total of 67 patients were enrolled in the program and a total of 1,386 INR values obtained. Of these patients, 39 (58.2%) had an indication of venous thromboembolism, 25 (37.3%) had an indication of atrial fibrillation, one (1.5%) had an indication of each of the following; cardiomyopathy, mechanical aortic valve replacement and cerebral arterial dissection. The average time-in-therapeutic range (INR of 2 – 3 +/- 0.2) was found to be 82% during the study period. The reported time-in-therapeutic range (INR of 2 – 3 +/- 0.2) for outpatient practices in the literature is between 60 – 70%. A total of 28 (2%) INR values were greater than four and a total of 6 (0.4%) INR values were greater than six. There were a total of seven admissions/emergency room visits for bleeding during the study period and all were minor bleeding episodes as defined in the literature. Of these, three patients had INR values within therapeutic range, two had INR values between 3.1 and 6, and two had INR values greater than 6. Vitamin K was administered to four of these seven patients. There were no reports of major or fatal bleeding during the study period. The overall incidence of minor bleeding in our practice was 2.8% per year as compared to a reported annual incidence of 8-15% in the literature. Conclusion: The pharmacist-managed anticoagulation program at the IMFP has been able to sustain target INR attainment and has increased the percentage of INR values within target range over the past two and half years. In addition, patients in the program had a lower frequency of bleeding complications and supratherapeutic INRs compared to reported values in the literature.
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HUMAN IMMUNODEFICIENCY VIRUS ASSOCIATED NEPHROPATHY DEMONSTRATING PATHOLOGICAL FINDINGS CONSISTENT WITH A MEMBRANOPROLIFERATIVE GLOMERULONEPHROPATHY

Background: HIV associated nephropathy (HIVAN) is the most common form of glomerulopathy found in HIV-infected patients. Pathological evaluations typically demonstrate a collapsing focal segmental glomerulosclerosis; however, ten percent of biopsy specimens demonstrate lesions consistent with a membranoproliferative glomerulonephropathy (MPGN.) The underlying mechanism of HIVAN is thought to be a direct HIV-1 infection of the renal parenchymal cells. MPGN is most typical of patients with hepatitis C infection, especially in the presence of a mixed cryoglobulinemia, depleted complement levels and an HIV co-infection.

Presentation: Our patient is a 58 year old gentleman with a medical history of hepatitis C infection, diabetes mellitus type 2, who on outpatient follow-up was noted to have rapidly progressing renal insufficiency reflected by a rise in creatinine from 1.51 mg/dL to 4.24 mg/dL over 4 a month period. This was accompanied by nephrotic range proteinuria (300 mg/dL) and resistant hypertension. Additional studies revealed the presence of a mixed cryoglobulinemia and normal complement levels (C3, C4). Hepatitis C was not detected by polymerase chain reaction analysis. Notable was a positive rapid HIV test with a T-helper/CD4 cell count of 79 cells/mL. Our patient underwent a renal biopsy which demonstrated findings consistent with a MPGN. The patient was treated with highly active anti-retroviral therapy (HAART) leading to significant improvement in the patient’s CD4 count. Additionally, the decline in renal function was halted with creatinine levels declining from 4.07 mg/dL to 3.59 mg/dL over two months.

Discussion: HIV related nephropathy manifesting with a MPGN is a relatively rare but known variant of a condition which typically demonstrates pathologic findings of a collapsing focal segmental glomerulosclerosis. Overlapping hepatitis C infection is felt to be the usual cause for the rapidly progressive renal failure. In our patient the hepatitis C was undetectable and complement levels were within normal limits suggesting that an HIV associated nephropathy may have been the more likely culprit.. The stabilization renal function following initiation of HAART regimen supports this hypothesis.

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CONCURRENT DEVELOPMENT OF ACUTE GASTROINTESTINAL T-CELL LYMPHOMA AND CHRONIC LYMPHOCYTIC LYMPHOMA: A CASE REPORT

Introduction: About 3% of patients with B-cell chronic lymphocytic leukemia (CLL) develop a high grade B-cell lymphoma, an association called the Richter’s Syndrome. These B-cell lymphomas are believed to arise by clonal evolution from the CLL cells. Here we present a patient who developed a gastrointestinal T-cell lymphoma and was also found to have CLL on bone marrow biopsy. Case Description: A 65 year old gentleman with no significant past medical history presented with one month of non-bloody diarrhea and about 20 lbs weight loss. He had about five bowel movements per day with no abdominal pain or hematochezia. On examination, his vital signs were stable and he had a performance status of 80% on the Karnofsky scale. Right cervical and bilateral auxiliary lymph nodes were enlarged, non-tender and hard, and the spleen was palpable below the costal margin. Labs: WBC 4.7 x 10^3/µL with 13.8% lymphocytes, absolute lymphocyte count 621 x 10^3/µL, platelets 81 x 109/µL, AST 48 units/L, ALT 41 units/L, LDH 937 units/L and uric acid 51 mg/dL. CT scan revealed multiple enlarged lymph nodes and splenomegaly. Endoscopic biopsy revealed an atypical lymphoid infiltrate of mixed sizes infiltrating the lamina propria of duodenum, terminal ileum, as well as the right and left colon. Immunohistochemical panel revealed that the atypical cells were T-cells with a Ki-67 proliferative rate of 30%. On bone marrow biopsy, the patient was found to have a T-cell lymphoma and a clonal B-cell population consistent with CLL. The plan was to treat the T-cell lymphoma first since CLL has a relatively indolent course. The patient received chemotherapy with CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisolone) but unfortunately he developed severe pancytopenia and was hospitalized for sepsis and respiratory failure. He was treated successfully and discharged home. Discussion: Patients with
CLL may develop a high grade B-cell lymphoma which is believed to arise by clonal evolution from CLL cells. This condition is known as the Richter’s syndrome. However, it is uncommon for these patients to develop a lymphoma of T-cell origin. A literature review revealed only one other such case reported in 2001 by Gottesman and colleagues. That patient was diagnosed with CLL but over a period of ten years was found to have developed a T-cell lymphoma. The authors suggested that in patients with CLL, immuno-dysregulation can result in the proliferation of T-cells, which may mutate and result in the development of a new malignant clone. In our patient, CLL is a coincidental finding with the gastrointestinal T-cell lymphoma, thus it is not evident which one developed first. We believe the CLL was likely present for some time and that the gastrointestinal T-cell lymphoma, being more aggressive, made the patient symptomatic with weight loss and diarrhea.

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**PSEUDOMELENA IN A PATIENT WITH FACTOR V LEIDEN DEFICIENCY**

Though a common occurrence, familiar to most experienced clinicians, pseudomelena (the false appearance of blood in stools, often caused by the ingestion of various substances) still often goes unrecognized in the clinical settings and often leads to unwarranted testing. Case: A 53 year old female with a past medical history of Factor V Leiden deficiency, pulmonary embolism, and DVT presented to the ER with a 3 day history of brownish red stools. Patient was on 8 mg of warfarin daily. She denied eating any “red foods”. On presentation she was afebrile and hemodynamically stable. Her physical exam was completely unremarkable and included a benign abdominal exam. Lab tests revealed hemoglobin of 12 and INR of 2.7. An occult blood test was not carried out as the presence of blood was obvious in the stool sample. Urine sample appeared grossly pink but was negative for RBCs. Serial CBC monitoring did not reveal any drop in hemoglobin and EGD was negative. Later on patient reported that 5 days ago she had a “beet rich” meal. She had forgotten to mention this earlier and didn’t make the connection because the beets were a much different color than the subsequent stools. Discussion: The term pseudomelena often serves as a misnomer (as melenic stools are generally described as dark red, tarry, or black, while stools considered as “pseudomelena” are bright red, purple or reddish brown; perhaps more appropriately termed pseudohematochezia). Etiologic factors include the ingestion of iron, bismuth, licorice, beets, spinach, carrots, blueberries, red or orange gelatin. Therefore, a commonly asked diagnostic question: “Did you eat anything red” may be wholly inadequate. As seen in this list, depending on patient’s conceptualization of “redness” (she later described beets as purple) many of these substances fall outside that definition. Furthermore, in situations where the index of suspicion for a gastrointestinal bleed is elevated, as occurred in this patient on warfarin therapy, questions regarding food intake may be afterthoughts, and the necessary inquiry into dietary habits curtailed. As with real melena and hematochezia, pseudomelena can present in nearly all shades of red, purple, brown and black. Therefore, the color is not a reliable indicator for the presence of blood. True melena has a distinct smell from the enzymatically processed blood. However, while odors can be a diagnostic boon, it cannot be the foundation on which a diagnosis (either melena or pseudomelena) is made. Conclusion: Pseudomelena can imitate symptoms of gastrointestinal bleeding so closely that even the expert clinician may overlook the dietary intake of foods such as carrots, beets or orange gelatin. A comprehensive investigation into the dietary habits of any patient presenting with melena or hematochezia should be performed. References available upon request

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**IS THERE AN ASSOCIATION OF INPATIENT FALLS AND HYponATREMIA?**

INTRODUCTION: Falls are a major problem causing injuries that can not only be life threatening but also very costly to patients and institutions, and can lead to increased morbidity and mortality. Reduction in falls should be a priority for hospital quality and patient safety. In the literature, advanced age, treatment-related factors and change in mental status were among the risk factors for falls in an inpatient setting. It is described in the
Literature that patients with hyponatremia are at increased fall risk possibly from marked gait and attention impairments. **METHOD** We performed a retrospective review of medical records to examine the association of falls with hyponatremia in the hospital setting. The records of all patients admitted to Saint Barnabas Medical Center between January 2009 and May 2012 were reviewed using a computer assisted search. Inclusion criteria were patients 18 years or older who had a documented fall during hospitalization, hospital stay greater than one day, and at least one serum sodium level measured at admission and one within 24 hours before or after the fall. A total of 969 patients were identified using the inclusion criteria noted. We then compared admission serum sodium levels with levels at the time of the fall. **RESULTS:** Nine hundred and sixty-nine patients between the ages of 19 to 101 years had falls during the study period. Of these, 495 (51%) were males and 475 (49%) females and the mean age was 66 years. Fifty six percent (543) of the patients were on medications that affect the nervous system, 41% (401) were on vasodilators, 24% (233) were on diuretics, and 82% (795) were on more than four medications. Twelve percent (113) had a history of stroke. The average sodium level was 137 mmol/L (normal 135-145) at admission and was no different around the time of fall. Only 20% (190) of patients had hyponatremia (sodium <135 mmol/L) during the fall which was similar to the number of patients with hyponatremia on admission 19% (182). **DISCUSSION:** Our study suggests that there is no significant increase in the incidence of hyponatremia among hospitalized adult patients at the time of in-hospital falls. Hyponatremia might not be a predictor of falls in the hospital setting as it has been described in an outpatient setting. The results should be confirmed by other studies.

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**A CASE OF ISOLATED CEREBRAL WHITE MATTER LESIONS IN CELIAC DISEASE**

Introduction Celiac disease (CD) is common (almost 1% of the Western population) and has many extra-gastrointestinal manifestations. Neurological complications including ataxia, peripheral neuropathy, and epilepsy are well-recognized. However, white matter lesions (WMLs) are relatively rare. We describe a patient with unexplained neurological symptoms and WMLs found to have CD without gastrointestinal symptoms.  

**Case Description**  
A 58 year old woman with a history of depression and aortic regurgitation was referred for neurological evaluation as she was suffering from intermittent headaches and impaired memory for three years and episodic left arm and leg weakness for one year. She attributed her symptoms to an injury she endured three years ago when she fell and hit the back of her head. She denied diarrhea or weight loss. Family history was not significant for gastrointestinal or neurological disease. On neurological exam, the patient was fully oriented and had no speech or short term memory deficits. Cranial nerves, motor function and coordination were intact. The sensory exam was remarkable for a mild decrease in vibration sense in the right toe. Brain magnetic resonance imaging (MRI) showed nonspecific WMLs scattered in the left frontal and parietal lobes without enhancement. Laboratory studies were unyielding, including: complete blood count, complete metabolic panel, thyroid function tests, serum iron, vitamin B12, vitamin E, and heavy metals. Evaluation for Human Immunodeficiency Virus, Human T-Lymphotropic Virus 1 and 2, Lyme disease, Ehrlichiosis, Babesiosis, and anaplasmosis were unremarkable. Additionally autoimmune antibodies were not present and examination of cerebrospinal fluid including assessment for oligoclonal bands was unremarkable. Magnetic resonance angiography (MRA) of the head and neck did not show stenotic or vasculitic changes and a 72-hour ambulatory electroencephalogram did not reveal seizure activity. After Anti-tissue transglutaminase IgG, anti-gliadin IgA and anti-gliadin IgG were positive at 49 U/ml (normal <5), 27 (normal <11), and 25 (normal <11), respectively, the patient underwent duodenal biopsy and CD was confirmed with Marsh 3c histology.  

**Discussion** This case supports the suggestion that evaluation for CD be part of the work-up of patients with neurological symptoms of unknown etiology particularly with the presence of WMLs, even in the absence of enteropathy. MRI findings vary from numerous foci to wide spread confluent lesions in both hemispheres. The WMLs may be due to vasculitis or demyelination. Headache seems to be the dominant symptom in most similar cases reported. Most of these patients, however, were already diagnosed with CD, unlike our patient.
IMPACT OF ELECTRONIC HEALTH RECORD ON DOCUMENTATION AND PATIENT OUTCOMES AT OUTPATIENT PRACTICE

Objectives: We wished to involve all medicine residents in a performance improvement (PI) project that evaluated the delivery of preventive care and management of common chronic conditions in our resident clinic before and after implementation of electronic health records (EHR). Methods: The residents conducted a chart review of 396 patients in 2010, to evaluate their performance on 20 parameters regarding documentation and attainment of various health goals as per current guidelines. The results were compared to HEDIS national benchmarks. We set the goal as 85% for those measures that did not have published benchmarks. They then developed system based plans for quality and performance improvement to surpass benchmarks for each measure. Follow-up review in 2011 demonstrated the positive impact of this project on delivery of care. In August 2011, EHR was introduced. This year, residents reviewed 319 records to determine the impact of EHR on quality measures. We hypothesized that EHR would improve documentation of care. Results: In 2010, we exceeded benchmarks in 10 out of 20 measures. After dissemination of results, our residents developed system based plans for improved delivery of care and documentation. A specific example was the development of diabetic eye exam form that ophthalmologists could complete and return to resident clinic which helped to improve documentation of yearly eye exams in diabetics (28% in 2010, 43% in 2011, 48.68% in 2012). Follow up analysis in 2011 revealed that we surpassed benchmarks in 12 measures. In 2011, after implementation of EHR, we only surpassed benchmarks in 9 out of 20 parameters. Among residents, PGY3, PGY2 and PGY1 surpassed benchmarks in 8, 9 and 7 parameters respectively. Lower compliance was noted in parameters related to documentation such as pain scale and smoking cessation counseling. This was attributed to lack of experience with EHR. The overall compliance in all measures was 64.35% in 2010 which improved to 69.45% in 2011. After introduction of EHR, it decreased to 64.45%. Residents are now working on ways to use EHR to improve our performance on all quality measures. Conclusions: By having residents perform this practice based improvement exercise, we were able to obtain data to give feedback to them on their individual performance. This led to implementation of processes and behavioral changes in our physicians. The result has been measurable improvement in quality parameters. Although the implementation of EHR caused a slight decline in performance measures, we expect improvement as residents, faculty and staff learn to use the system to its fullest potential. Awareness of performance data can increase self-evaluation, improved feedback for trainees and can lead to better patient care.

SOMETIMES HOOF BEATS ARE FROM ZEBRAS

INTRODUCTION Clinicians make their initial diagnosis by way of pattern recognition. Diagnostic algorithms are often based on methodical evidence-based research, and therefore offer a sense of security and reliability. However it is important to keep in mind that multiple etiologies can yield similar presentations and clinicians must be alert to exceptions to the norm. Dyspnea is an excellent example of this phenomenon. A Patent Foramen Ovale (PFO) and Chronic Obstructive Pulmonary Disease (COPD) both may cause dyspnea and hypoxia but through different mechanisms. We present a patient who had multiple office visits and hospitalizations for COPD exacerbations though objective evaluation was inconsistent with that diagnosis. Therapeutic intervention for COPD was initiated with each presentation resulting in minor improvement but never resolution of her subjective dyspnea or objective hypoxia. Despite the data and results her admitting diagnosis with each hospitalization was “COPD exacerbation”. PATIENT PRESENTATION: An 83 year old woman presented with cough, dyspnea, wheezing, lethargy, and confusion. She was hypoxic on room air and supplemental oxygen. She had no associated fever, sputum production, chest or calf pain, palpitations, nausea or vomiting. Chest radiographs, arterial blood gasses, pulmonary function tests, lower extremity dopplers, computed tomography angiogram of the chest and trans-thoracic echocardiography (TTE) were inconsistent with COPD or associated conditions. She was started on bronchodilators, systemic steroids, antibiotics, and non-invasive ventilation with bi-level positive pressure ventilation. She improved symptomatically and was transferred to pulmonary rehabilitation on nasal oxygen. She re-presented with the same symptoms, this time
requiring endotracheal intubation and admission to the intensive care unit. Weaning was forestalled by persistent hypoxia that was responsive to pulmonary artery vasodilators. Review of her prior presentations, test results and current lung function led to investigation for non-pulmonary causes of hypoxia. Transesophageal echocardiography (TEE) with bubble study revealed a large PFO with right-to-left shunt. She underwent percutaneous closure of the PFO with an AMPLATZER septal occluder device. She was extubated successfully and sent to the medical floor on nasal oxygen. CONCLUSION Results of prior diagnostic evaluation were inconsistent with COPD as the cause of her symptoms and hypoxia. More aggressive pursuit of non-pulmonary causes of her pulmonary disease should have been undertaken. Sometimes the hoof beats are not horses but in fact zebras.

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A CASE OF HYPERTHYROID CARDIOMYOPATHY MIMICKING ACUTE CORONARY SYNDROME

Case: A 54 year old Hispanic woman with a medical history significant for hypertension and hyperlipidemia presented to the emergency room with substernal chest pain at rest, shortness of breath and palpitations. The pain radiated to her left arm and back. There was no history of smoking, diabetes, coronary artery disease and no family history of premature heart disease. On examination she was diaphoretic with a heart rate of 130 beats per minute. Her blood pressure was 180/120mmhg and oxygen saturation was 88% on room air. She had bibasilar lung crackles but no jugular venous distention or peripheral edema. Electrocardiography (ECG) showed sinus tachycardia with ST segment depressions in the lateral leads and evidence of left ventricular hypertrophy. A CT scan of the chest with contrast revealed pulmonary edema with bilateral pleural effusions; there was no evidence of pulmonary embolism or aortic dissection. The patient had another episode of chest pain with diaphoresis in the emergency room, which was relieved with sublingual nitroglycerin. Stat ECG at that time showed no changes. The patient was treated for presumed unstable angina/non-ST elevation myocardial infarction. She was started on a &"61538; blocker, angiotensin converting enzyme inhibitor, diuretics, heparin, aspirin and statin. Echocardiography on the day of admission showed an ejection fraction of 35% with mid-anteroseptal hypokinesia with no significant valvular disease. Creatinine kinase MB increased from 7.7 ng/ml to 8.2 ng/ml over 8 hours and decreased to 4.4 ng/ml in following 8 hours. Her troponin-T levels remained in the intermediate range. Cardiac catheterization two days after admission, demonstrated normal coronary arteries and an ejection fraction of 55%. The patient’s dyspnea improved with use of diuretics. Thyroid studies were also ordered as she had persistent sinus tachycardia. TSH was suppressed at <0.008 UIU/ml with an elevated free T4 of 2.13 ng/dl (normal 0.89 to 1.76). Her thyroid peroxidase antibody and thyroglobulin antibody levels were not detectable. The patient was discharged on methimazole, prednisone, statin and &"61538; blockers. Discussion: Hyperthyroid cardiomyopathy with resultant heart failure is a well documented complication of hyperthyroidism. The literature shows that congestive heart failure is the initial clinical presentation in approximately 6% of patients with hyperthyroidism1. Atrial arrhythmia and hypertension are other common complications. This patient presented with features of heart failure that appeared to be secondary to acute coronary syndrome, but was found to have normal coronary anatomy. Hyperthyroidism should routinely be considered as a cause of heart failure as it is reversible.

1. Incidence, clinical characteristics and outcome of congestive heart failure as the initial presentation in patients with primary hyperthyroidism. Siu CW, Yeung CY, Lau CP, Kung AW, Tse HF Heart. 2007; 93(4):483

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FALSE-NEGATIVE PET SCAN IN SETTING OF BIOPSY-PROVEN BONE MARROW METASTASIS IN A PATIENT WITH ADENOCARCINOMA OF THE RECTUM

Introduction: Positron Emission Tomography (PET) is characteristically known for its high specificity and sensitivity in finding metabolically active cells in ruling out metastatic malignancy. We report a case of a false negative PET scan on a patient with biopsy-proven bone marrow metastases. Case Presentation: We present a 68-year-old man with recurring rectal bleeding who was recently diagnosed with adenocarcinoma of
the rectum. Immediately after PET-CT scan he developed lightheadedness, dyspnea, tachycardia and hypotension. Laboratory work up revealed profound anemia (6.2g/dL) and he was admitted to the general medical floor for transfusion and observation. Patient ultimately required multiple transfusions and concordantly developed severe thrombocytopenia that was refractive to platelet transfusion. During the patient’s lengthy stay, the outpatient PET-CT scan was read as negative for metastasis and the patient was staged T3N1M0. Due to high clinical suspicion of bone marrow involvement, the patient underwent a bedside bone marrow biopsy of the right posterior iliac crest. Results showed metastatic signet-ring cell adenocarcinoma with origin from the patient’s known rectal primary. The patient was treated with chemotherapy consisting of FOLFOX (5-flurouracil, leukovorin, and oxiplatin). Patient responded well and was subsequently discharged with hemoglobin levels above 10 g/dL and a platelet count stable of 70,000/mL. Interestingly, a repeat PET-CT scan done several weeks later showed metabolically active metastatic sites in the bone marrow. Discussion: A literature search using Ovid and PubMed search engines was performed between 1992-2012. Multiple articles reported false-negative PET scans for metastatic disease, particularly involving lymphomas, breast cancer, and neurological pathology, with sensitivities as low as 84%. However, reports of false-negative results involving metastasis located in the bone marrow are not as common. This case report presents such an occurrence.

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**LARGE UTERINE LEIOMYOMATA CAN LEAD TO LIMB LOSS**

Introduction: Uterine leiomyomata are the most common benign tumors of the female genital tract. Some tumors can reach a large size without causing any symptoms whereas some small tumors can cause bleeding and abdominal pain. Medical therapy is not useful at preventing the development of leiomyomata or reducing morbidity. Hysterectomy is the definitive treatment. Asymptomatic fibroid tumors can be left untreated as long as they are monitored closely. There are reports of large leiomyomata causing compression of adjacent structures. Here we describe a case of a large leiomyoma causing limb ischemia and subsequent limb loss. Case description: A 46 year old African American woman with diabetes mellitus and hypertension presented to the emergency room in diabetic ketoacidosis with severe respiratory distress and a gangrenous right foot. The patient was intubated and admitted to the intensive care unit for stabilization. Laboratory studies revealed severe anemia and ketoacidosis. Physical examination demonstrated a palpable abdominal and pelvic mass. CT scan of the abdomen and pelvis with intravenous contrast identified a calcified 25 cm mass emanating from the uterus extending into the abdomen. Moreover a 4.6 cm long embolus at the aortic bifurcation was revealed. Venous doppler of the lower extremities revealed an acute deep vein thrombosis of the right posterior tibial vein. The patient underwent total abdominal hysterectomy, bilateral salpingo-oophorectomy and aortic thrombectomy. Pathology report confirmed the subserosal and submucosal fibroids and the aortic thrombus. Repeat venous doppler of the lower extremities four days later showed progression of the posterior tibial vein clot while on intravenous heparin drip. The patient underwent amputation of the gangrenous right foot. An inferior vena cava filter was also placed. The patient had a recurrence of gangrene of right leg and required below knee amputation. Pathology report post amputation of the right leg showed thrombotic occlusion of the anterior tibial artery and severe atherosclerosis of the posterior tibial artery. The large subserosal fibroid compressing on the aorta causing thromboembolic disease compromising the blood supply led to the loss of right lower extremity. Discussion: Large leiomyomata can not only cause signs and symptoms related to bleeding and infertility, but can compress nearby organs with resultant morbidity. Cases of fibroid compression of the bladder, inferior vena cava and the common iliac artery have been documented leading to urinary retention, inferior vena cava thrombus, and tissue loss respectively. Physicians should counsel patients regarding such complications. A thorough follow up plan in patients should be instituted irrespective of fibroid size.
YOUNG MAN WITH EPILEPTIC DISORDER – WITH NEW EKG FINDINGS SHOWING BRUGADA SYNDROME: A UNIFYING DIAGNOSIS?

Introduction: Brugada syndrome is a rare autosomal dominant disorder that is caused by myocyte sodium channel dysfunction. It is characterized by electrocardiographic (ECG) changes and manifests clinically with syncope, arrhythmia and sudden cardiac death. Patients usually have a characteristic ECG pattern consisting of a pseudo-right bundle branch block and persistent ST segment elevation in leads V1 to V3. Some forms of epilepsy have also been linked with dysfunction of the sodium channels in neurons due to a mutation in the SCN1A gene. We present a patient with these two distinct maladies and posit that they may be a relationship between the sodium channel dysfunction in two different organ systems. Case Report: A 37 year old man with a past medical history of intractable seizures since age nine was transferred to our facility for further evaluation. The patient had poorly controlled epilepsy and a number of medications had been tried with variable success. Interestingly, the patient had several first and second degree relatives who died suddenly at a young age without an obvious cause. Mapping electrodes were to be used to pin-point the epileptic focus for possible surgical excision. During the pre-op evaluation the patient was found to have ECG changes consistent with saddle-back type-2 Brugada Syndrome. During the course of stay in the hospital his antiepileptic medications were tapered down while he was being monitored with electroencephalography and cardiac telemetry, when the patient had a seizure. Cardiac telemetry showed that the patient did not have any ventricular arrhythmias at the time of the seizure event. The epileptic focus was narrowed down and later excised. The patient tolerated the procedure well and discharged with plans to see a cardioligist for further evaluation for Brugada syndrome. Discussion: This patient has two different disorders that may actually be discrete manifestations of essentially the same pathology—sodium channel dysfunction. Brugada syndrome has been linked to SCN5A mutation linked to sodium channels on myocytes while epilepsy has been linked to a similar mutation in the central nervous system. Seizures may be seen in patients with Brugada Syndrome and have been thought to be to be from cerebral hypoperfusion and ventricular arrhythmias. Conversely epilepsy has been associated with sudden death. In this case however, arrhythmia was ruled out during the time of seizure, suggesting two separate disorders. Thus, dysfunction of the sodium channel may present as two unrelated syndromes rather than one organ-specific disorder. We conclude that this patient along with potentially many more has a systemic sodium channel dysfunction which presented with two clinical syndromes that seem unrelated but might have a unifying pathology.

A DEADLY TRIAD: THE VALUE OF DIAGNOSTIC BRONCHOSCOPY IN THE IMMUNOCOMPROMISED PATIENT

Introduction: Diagnostic bronchoscopy should be strongly considered in critically ill patients who are immunosuppressed with respiratory insufficiency, in whom pneumonia is suspected. Case description: 75 year old gentleman status post renal transplant and on immunosuppressants (tacrolimus and prednisone) developed periampullary carcinoma. Post pancreaticoduodenectomy he was admitted to ICU in anticipation of fluid shifts and abdominal compartment syndrome. He developed hypoxia and wheezing on day 3 requiring high flow oxygen. Chest radiographs suggested perihilar and basilar densities and bronchodilators and diuretics were initiated. On the fifth ICU day he developed bacteremia with Methicillin Resistant Staphylococcal Epidermidis and Enterococcal fecalis, and was started on vancomycin and piperacillin – tazobactam. Upon stabilization, the patient was transferred to the floor. However, he developed sepsis after a few days, despite being on broad spectrum antibiotics. He was intubated and readmitted to ICU for septic shock with multiorgan failure. Bilateral patchy infiltrates were noted on chest x rays with pneumonia suspected as the source of sepsis. Piperacillin-tazobactam was changed to meropenem while vancomycin was continued and caspofungin was added for better fungal coverage. Computed axial tomogram (CT) of chest revealed increasing patchy infiltrates in bilateral lung fields with enlarging consolidation at left lung base suggestive of pneumonia and possible septic emboli with worsening intraabdominal ascites and mild posterior pancreatic fluid collection on CT scan of abdomen. A transesophageal echocardiography ruled out endocarditis while blood cultures were
negative. Bronchoscopy was done on day 3 of ICU readmission with bronchoalveolar lavage of left and right lower lobes. Friable mucosa in left lower lobe and several small white papillary endobronchial lesions in left lung at the carinas were noted which were brushed. Infectious Diseases consultant recommended adding amikacin and changing caspofungin to amphotericin B for possible aspergillosis or mucormycosis. The next day his clinical condition worsened and he died later that day. Results obtained from the bronchial alveolar lavage and brush specimen revealed herpes simplex virus type 1, Aspergillus fumigatus and Candida krusei. Conclusion: Lower respiratory samples can be obtained with or without bronchoscopy. Cochrane data base analysis has shown that sampling via bronchoscopy does not improve mortality, length of ICU stay or duration of mechanical ventilation. However it is considered to be more accurate, leading to faster de-escalation of antibiotic usage. Case in point is the above clinical vignette, where bronchoscopy sampling was found to be positive for not one but three organisms which are associated with high mortality in the critically ill immunosuppressed patients. This reiterates the fact that bronchoscopy with cytologic and microbiologic examination of sample is an important diagnostic tool to be strongly considered in immunosuppressed critically ill patients, especially in presence of high clinical suspicion for an underlying pneumonia.

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BRAIN ABSCESS AND INFECTIVE ENDOCARDITIS CAUSED BY STREPTOCOCCUS INTERMEDIUS IN A PATIENT WITH PATENT FORAMEN OVALE

Introduction: Streptococcus intermedius is a part of the normal flora of mouth, gastro-intestinal tract and genito-urinary tract. It is not uncommon for this organism to cause abscesses in the liver and brain but it only rarely leads to infective endocarditis. We present a patient with a patent foramen ovale (PFO) who developed brain abscess and infective endocarditis due to Streptococcus intermedius. The association between PFO and paradoxical microbial embolization has not been studied extensively. Case Presentation: A 37 year old lady presented with sudden onset of weakness in both upper and lower extremities associated with slurred speech, diplopia, vomiting, fever and lethargy. In the Emergency Department, she was found to have a temperature of 101° F while other vital signs were normal. Physical examination revealed lethargy, dysarthria, dysconjugate gaze, left abducens nerve paresis, right facial droop, right sided hemiparesis and right hemisensory loss. The rest of the physical exam was unremarkable. A CT scan of the brain showed hydrocephalus and a 19 mm hemorrhagic, left para-median pontine lesion with incomplete ring enhancement and extensive edema in the midbrain and left pons. An external ventricular drain was placed in the right prepontine cistern which drained bloody, purulent fluid. The cerebrospinal fluid cultures showed a heavy growth of Streptococcus intermedius. Blood and urine cultures were negative. A thorough review of history to identify the source of infection revealed that she did not have any recent medical or dental procedures except for tubal ligation which was done two weeks prior. The search for the primary source of infection included transesophageal echocardiography (TEE) and CT scan of the abdomen and pelvis with intravenous contrast. TEE revealed a PFO with inter-atrial shunt and two vegetations at the junction of the superior vena cava and right atrium. CT scan of the abdomen and pelvis showed a wedge-shaped infarct in the left kidney but no abscess or phlebitis. A diagnosis of possible infective endocarditis was made based on modified Duke’s criteria. Patient showed clinical improvement during the hospital stay and was discharged home on antibiotics to complete six weeks of therapy. Follow up TEE after two months revealed complete resolution of the vegetations. Discussion: PFO is often an under-recognized cause of brain abscesses. Studies have shown that a PFO can serve as a pathway for paradoxical embolism via right to left shunting in cryptogenic stroke. A transient right-to-left gradient is sufficient to induce shunting and commonly occurs in patients without net right-to-left shunt. A similar pathophysiology could explain paradoxical microbial embolization leading to development of a brain abscess.
EXEMPLARY ARTERIAL THROMBOSIS AND ACUTE LIMB ISCHEMIA IN AN ANTICOAGULATED PATIENT

Introduction: Acute limb ischemia can result from a sudden decrease in limb perfusion causing a potential threat to limb viability. It may present as rest pain, an ulcer and/or gangrene within two weeks of the acute event. The management of acute arterial occlusion remains a challenge despite the availability of surgical thromboembolectomy, bypass grafting and thrombolytic therapy. Morbidity, mortality, and limb loss rates from acute lower extremity ischemia remain high. Independent of the treatment modality used, early diagnosis and rapid initiation of therapy are essential in order to salvage the ischemic extremity. Case Presentation: A 64 year old woman with atrial fibrillation, hyperlipidemia, diabetes mellitus, coronary artery disease, and hypertension presented to the emergency room with low back pain radiating down the left lower extremity associated with weakness and patchy areas of numbness of the left foot not in specific nerve distribution. On physical examination the limb was warm with palpable pulses. MRI of the lumbar spine revealed spinal stenosis with marked foraminal narrowing at L4-L5. A venous doppler study demonstrated deep vein thrombosis of the left popliteal vein. The patient had been on warfarin therapy for atrial fibrillation and at presentation her INR was supratherapeutic at 4.9. The following day, the patient developed sudden worsening of pain and paraesthesia in her left leg. The affected limb was cold to touch and pulseless, distal pulses were not audible by bedside Doppler study. An immediate arterial duplex study revealed severe arterial occlusive disease. Flow was not appreciated in the common femoral, superficial femoral and peroneal arteries, and poor flow was noted in the popliteal and posterior tibial arteries. She was taken to the operating room where embolectomy and femoropopliteal bypass were performed. Extensive tissue damage led to an above the knee amputation. Pathology examination of the specimen revealed extensive atherosclerosis with thromboembolus. Hypercoaguability studies including protein C, protein S, antiphospholipid antibody, Factor V Leiden, homocysteine and lupus anticoagulant were unremarkable. A minor deficiency in protein C and S was observed, however this was not considered significant as warfarin and recent acute thrombosis affect their levels. Discussion: This case demonstrates that arterial thrombosis can occur in the setting of seemingly therapeutic anticoagulation therapy. When evaluating patients with multiple risk factors, physicians should have a high index of suspicion for arterial thrombosis if they present with atypical symptoms (eg. patchy paresthesias). A simple non-invasive diagnostic test such as an arterial ultrasound is reasonable as an early diagnostic tool to reveal thrombotic changes in an effort to prevent limb loss. In this case, the supratherapeutic INR and the spine MRI data directed the physicians to an alternative diagnosis. This unusual presentation underscores the need to consider arterial thrombosis even in appropriately anticoagulated patients.

SPONTANEOUS DISSECTION OF INTERNAL CAROTID ARTERY PRESENTING WITH "PAINFUL HORNER'S SYNDROME" AND MONOCULAR VISUAL FIELD DEFECT

Introduction: Spontaneous internal carotid artery dissections (ICAD) have an annual incidence rate of 2.5 per 100,000 to 3.0 per 100,000 in United States. Usual presentations of these patients include headache (68%), neck pain (26%), Horner’s syndrome, lower cranial nerve palsies and hours or days later signs of cerebral or retinal ischemia. We present a case of spontaneous ICA dissection with a painful Horner’s syndrome and monocular visual field defect that was treated promptly with anticoagulation. Case Report: A 36-year-old man presented with sudden onset of right eyelid droopiness that started three days prior to admission. He also had a continuous, aching right temporal headache and right neck pain, which was not fully relieved by acetyl salicylic acid. He complained of blurred vision in the right eye, accompanied by floaters on and off in “zigzag” manner. There was no recent trauma, loss of consciousness, weakness or numbness in the extremities. On initial examination, his BP was 150/90 mmHg. No carotid bruit was noted on neck exam. Pupils were reactive to light bilaterally but unequal in size, with the right 1mm smaller than the left. There was loss of the temporal visual field in the right eye. The right eyelid was mildly ptotic. The rest of the neurological exam was unremarkable. A magnetic resonance angiogram (MRA) of the neck showed absent flow signal in the right ICA from the bifurcation to base of skull, through the petrous segment, with minimal flow in the cavernous segment,
likely secondary to collaterals. There was an intimal flap seen in the right ICA. At this time, diagnosis of spontaneous carotid artery dissection was made and the patient was started on anticoagulation with enoxaparin and eventually continued on warfarin. Repeat MRA after three months showed persistent occlusion of the right ICA, with good flow signal in the middle and anterior cerebral arterial territories from extensive collaterals and communicating arteries. There was partial recanalization of the petrous segment of the ICA. His visual symptoms and carotidynia continue to gradually improve. Discussion The clinical case presented here is of medical interest in understanding the medical emergency in patients presenting with classic symptoms of a painful Horner’s syndrome and retinal ischemia. Internal carotid artery dissection is an increasingly recognized cause of stroke that accounts for up to 20% of ischemic strokes in young adults. The prognosis after dissection is related to the severity of the initial ischemic insult and the extent of collateral circulation. Only 7.7% of strokes in patients with carotid dissection have a presumed hemodynamic etiology (insufficient flow), whereas 92.2% are caused by embolic infarction. This case emphasizes the need to rapidly identify the classic signs of dissection to enable institution of appropriate treatment.

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A CASE OF CARDIAC TAMPONADE AND PROTEINURIA: IS THERE AN ETIOLOGICAL RELATIONSHIP?

INTRODUCTION: Cardiac tamponade, as a cause of proteinuria has not been described in the literature to the best of our knowledge. We demonstrate a temporal relationship between the onset of proteinuria occurring with cardiac tamponade and its resolution subsequent to pericardial window on two occasions in the same patient suggesting a possible etiological relationship between the two conditions. CASE DESCRIPTION: A 75 year old lady with metastatic pancreatic adenocarcinoma was admitted with acute kidney injury and proteinuria. Physical examination was unremarkable. Laboratory data revealed a serum creatinine of 2.33 mg/dl that was elevated from her baseline value of 0.75 mg/dl. Urinalysis was significant for protein of 300 mg/dl. On day three, she developed shortness of breath and palpitations. A large pericardial effusion with features of cardiac tamponade was seen on echocardiography. A pericardial window was performed and 500ml of clear fluid was drained. Cytology revealed adenocarcinoma. Serial measurements of her spot UPCR revealed progressive decline from a maximum of 4.39 to 3.6 to 2.3 to 1.66 g/g creatinine on consecutive days. On discharge, her serum creatinine had returned to 1.2 mg/dl. Four months after discharge, she was admitted with shortness of breath and was diagnosed with a loculated pericardial effusion and cardiac tamponade. She had proteinuria of 100 mg/dl on dipstick and serum creatinine of 2.66 mg/dl. Interestingly, her proteinuria and acute kidney injury completely resolved after the pericardial window. DISCUSSION: We suggest a possible mechanism of proteinuria in cardiac tamponade by extrapolation from known, similar pathophysiology. In acute febrile illnesses and subsequent to vigorous exercise, one can see proteinuria related to presumed hemodynamic stress. "Orthostatic Proteinuria", is theoretically due to an exaggerated response to angiotensin II (AT-II) and norepinephrine upon assuming the upright posture. This response causes increased efferent glomerular vasoconstriction with a resultant acute increase in intraglomerular pressure and subsequent proteinuria. Proteinuria can be seen in "Nutcracker phenomenon", where the left renal vein traverses under the superior mesenteric artery and becomes trapped between the aorta and the superior mesenteric artery. This theoretically causes an increased glomerular back-pressure and increased release of AT-II with resultant proteinuria. Cardio-renal syndrome is yet another condition in which the physiology may be similar. Decreased cardiac function, with activation of the renin-angiotensin-aldosterone system, along with increased renal venous back-pressure define this syndrome. Our case of cardiac tamponade can be categorized within a subset of "Type I" cardio-renal syndrome. We suggest glomerular capillary hemodynamic changes due to renal venous back-pressure and elevated AT-II levels may influence glomerular permeability to macromolecules and can evoke proteinuria. The relationship between proteinuria and cardiac tamponade has not, to our knowledge, been specifically studied, and warrants further research.
CEFTRIAXONE INDUCED THROMBOCYTOPENIA

Introduction- Ceftriaxone is known to cause hematological complications such as eosinophilia (incidence of 6%), thrombocytosis (5.1%), leucopenia (2.1%) and hemolytic anemia (<1%) but ceftriaxone induced thrombocytopenia is rarely reported. We report a case of the patient presented with life threatening thrombocytopenia on ceftriaxone therapy. Case Presentation- A 73 year old man with history of hypertension, cutaneous T-cell lymphoma and prostate cancer visited his primary care physician with a two week history of cough, chest pain, fatigue and low grade fever. On review of systems he complained of an ongoing toothache for several months. Chest radiograph revealed a right upper lobe (RUL) consolidation and he was given a course of azithromycin. However, the patient's symptoms worsened and a CT of the chest was performed and showed a RUL mass with an air fluid level. CT guided biopsy grew Actinomyces odontolitycus and Streptococcus viridans. The patient was initially treated with penicillin G but discharged home on ceftriaxone because of its less frequent administration. The patient's clinical condition continued to improve on intravenous ceftriaxone. On outpatient follow up one month later, the patient was found to have platelet count of 5000 per cubic mm and was sent to ER for further evaluation. Fortunately the patient had no active bleeding and hemoglobin was unchanged since last admission. At this point, ceftriaxone was stopped and single donor platelets transfusion was given. Platelet count increased to 30000 per cubic mm after transfusion and outpatient follow up one week later showed a normal platelet count. Discussion – A variety of hereditary conditions, diseases and drugs can cause thrombocytopenia. Drug induced thrombocytopenia (DITP) is a serious, sometimes life threatening complication of drug treatment and ceftriaxone is one of the culprits. The pathophysiology is immune mediated destruction of platelets. The patients are sensitized by prior exposure and experience acute platelets destruction on re-exposure or after continuous drug exposure for at least one week. To diagnose DITP one needs a high degree of suspicioun. Clinical signs (ecchymosis, petechiae, bleeding), blood count, peripheral blood smear and serological tests to detect anti platelets antibodies support the diagnosis. Thrombocytopenia resolves quickly after stopping the offending agent. In general, any long-term antibiotics can increase complications such as hemolytic anemia, thrombocytopenia or kidney and liver failure. Checking blood counts and a complete metabolic panel one to two weeks after initiation of antibiotics can identify complications in early stages so that appropriate measures may be taken.

PERICARDIAL EFFUSION IN CHRONIC MYELOMONOCYTIC LEUKEMIA

Introduction: Chronic Myelomonocytic Leukemia (CMML) is a clonal hematopoietic stem cell disorder that is characterized by the presence of an absolute monocytosis in the peripheral blood and the presence of mixed myelodysplastic and myeloproliferative features in the bone marrow. There have been few case reports of pericardial effusion in CMML. Here we present a case of a patient with CMML who presented with a pericardial effusion caused by leukemia cells. Case Description: A 64 year old Caucasian woman initially presented with hot flashes, night sweats, easy skin bruising, vaginal bleeding and left upper quadrant abdominal pain. Laboratory workup revealed leucocytosis of 60 K/cmm with 5% blasts and 10% promonocytes and 15% monocytes. Hemoglobin was 10.7 g/dl and platelet count was 14 K/cmm. A Peripheral smear showed nucleated erythrocytes, decreased platelets, rare teardrop cells and a myelomonocytic series of blasts and monocytes. Flow cytometry detected CD-13, CD-117, CD-56 and CD-34 predominance, suggestive of myeloproliferation of monocytic lineage. FISH analysis showed Trisomy 8 with no other chromosomal abnormalities. Mutations for bcr-ablx and PDGFR were negative. A bone marrow biopsy showed increased blasts, promonocytes and lysozyme levels. She was diagnosed with CMML with Trisomy 8. Two weeks later, she presented with dyspnea and chest pain of four day duration. There was tightness of the chest which was aggravated on leaning forward. On physical examination a pericardial friction rub was audible but the patient was hemodynamically stable without signs of tamponade. A 2-D echocardiogram showed a normal left ventricular ejection fraction of 55-60% and a moderate pericardial effusion. Following pericardiocentesis, flow cytometry of the effusion revealed CD14+ (monocytic differentiation antigen) predominance. Gram staining and cultures of the effusion were negative. She received two cycles of decitabine and was referred for

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AGGRESSIVE SYSTEMIC MASTOCYTOSIS WITH SEVERE MALABSORPTION/SIGNIFICANT IMPROVEMENT ON THERAPY

Introduction: Systemic Mastocytosis (SM) is a rare, clonal disorder of the mast cells and CD34+ mast cell precursors. Degranulation of tissue mast cells accumulated in the bone marrow, skin, gastrointestinal tract (GI), liver, and spleen produces mediator-related symptoms. SM is usually an indolent disease, but an aggressive form can produce abdominal pain, nausea, vomiting, diarrhea, malabsorption, anaphylaxis, hypotension and shock. We present a patient with aggressive SM who improved on combination therapy. Case Report A 31 year-old woman was referred to our metabolic center because of chronic malabsorption with malnutrition. She had a history of asthma and skin reactivity. Her GI symptoms began at age 20 after a traumatic injury. She suffered from severe recurrent vomiting, abdominal pain and diarrhea. She had poor oral intake with multiple food intolerances. She failed nasogastric tube feeding because of vomiting. It was thought that she may have idiopathic gastroparesis but failed pyloric botox, GI stimulants, dopamine antagonists and gastric pacemaker therapy. She failed jejunostomy tube feeding and required total parenteral nutrition. Intestinal biopsies showed no significant findings on pathology except for decreased enterochromaffin cells. Cystic fibrosis profile was negative, the autoimmune regulator (AIRE) gene study was normal, and extensive work up for GI and autoimmune disorders did not lead to a specific diagnosis. Physical exam revealed a cachetic caucasian female with a BMI of 13.5. She was edentulous after tooth extraction for profound dental abscesses. The abdominal examination showed tenderness around the site of the removed jejunostomy and diminished bowel sounds. She was mildly anemic with a hemoglobin of 10.3 g/dl and a MCV 88 fl. Her serum albumin was 3.2 g/dl and total protein 6.3 g/dl. Bilirubin, transaminase levels, amylase and lipase were all within normal limits. Given the findings, a work up for SM was performed. The serum tryptase level was elevated on two separate occasions at 17.2 ng/ml and 22.5 ng/ml, respectively (normal <11.4 ng/ml). The 24-hour urine methyl-histamine level was also elevated at 215 mcg/g (normal 30-200). A presumptive diagnosis of SM was made and she was started on montelukast, cromolyn, famotidine and lansoprezole. On follow up, patient reported significant improvement in her symptoms with better oral diet tolerance. A bone marrow biopsy for confirmation of SM has been scheduled. Discussion SM is an under-recognized disorder which can produce significant morbidity in its aggressive form. We believe that clinicians should be more aware of the diagnosis and management of SM. This case illustrates the effect of available treatments to control symptoms in a difficult case.

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CONCURRENT DEVELOPMENT OF ACUTE GASTROINTESTINAL T-CELL LYMPHOMA AND CHRONIC LYMPHOCYTIC LYMPHOMA: A CASE REPORT

Introduction: About 3% of patients with B-cell chronic lymphocytic leukemia (CLL) develop a high grade B-cell lymphoma, an association called the Richter’s Syndrome. These B-cell lymphomas are believed to arise by clonal evolution from the CLL cells. Here we present a patient who developed a gastrointestinal T-cell
lymphoma and was also found to have CLL on bone marrow biopsy. Case Description: A 65 year old
gentleman with no significant past medical history presented with one month of non-bloody diarrhea and about
20 lbs weight loss. He had about five bowel movements per day with no abdominal pain or hematochezia. On
examination, his vital signs were stable and he had a performance status of 80% on the Karnofsky scale. Right
cervical and bilateral auxiliary lymph nodes were enlarged, non-tender and hard, and the spleen was palpable
below the costal margin. Labs: WBC 4.7 x 103/µL with 13.8% lymphocytes, absolute lymphocyte count 621 x
103/µL, platelets 81 x 109/µL, AST 48 units/L, ALT 41 units/L, LDH 937 units/L and uric acid 51 mg/dL. CT
scan revealed multiple enlarged lymph nodes and spleenomegaly. Endoscopic biopsy revealed an atypical
lymphoid infiltrate of mixed sizes infiltrating the lamina propria of duodenum, terminal ileum, as well as the right
and left colon. Immunohistochemical panel revealed that the atypical cells were T-cells with a Ki-67 proliferative
rate of 30%. On bone marrow biopsy, the patient was found to have a T-cell lymphoma and a clonal B-cell population consistent with CLL. The plan was to treat the T-cell lymphoma first since CLL has a
relatively indolent course. The patient received chemotherapy with CHOP (cyclophosphamide, doxorubicin,
vincristine, and prednisolone) but unfortunately he developed severe pancytopenia and was hospitalized for
sepsis and respiratory failure. He was treated successfully and discharged home. Discussion: Patients with
CLL may develop a high grade B-cell lymphoma which is believed to arise by clonal evolution from CLL cells.
This condition is known as the Richter’s syndrome. However, it is uncommon for these patients to develop a
lymphoma of T-cell origin. A literature review revealed only one other such case reported in 2001 by
Gottesman and colleagues. That patient was diagnosed with CLL but over a period of ten years was found to
have developed a T-cell lymphoma. The authors suggested that in patients with CLL, immuno-dysregulation
result in the proliferation of T-cells, which may mutate and result in the development of a new malignant
clon. In our patient, CLL is a coincidental finding with the gastrointestinal T-cell lymphoma, thus it is not
evident which one developed first. We believe the CLL was likely present for some time and that the
gastrointestinal T-cell lymphoma, being more aggressive, made the patient symptomatic with weight loss and
diarrhea.

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FUNCTIONAL PARAGANGLIOMA IN A YOUNG PATIENT PRESENTING WITH SYNCOPE

Paragangliomas, also known as extra-adrenal pheochromocytomas, are catecholamine secreting
neuroendocrine tumors. Middle mediastinal paragangliomas arise from aortopulmonary paraganglia and
posterior paragangliomas arise from aortosympathetic paraganglia. These neoplasms usually present with
hypertension, palpitations, headache, flushing and episodic perspiration. Syncope, dyspnea, cough and weight
loss are rare findings. We present the case of a young patient with paraganglioma who presented with chest
pain and syncope. An 18 years old female, with a history of quiescent systemic lupus erythematosus,
presented to the emergency department with a single episode of syncope. She reported acute onset anterior
chest pain and lightheadedness followed by syncope. There was loss of consciousness for about one minute
with no witnessed tonic-clonic activity, tongue biting or bladder/bowel incontinence. She was brought to the
emergency department where physical examination demonstrated orthostatic hypotension. She was treated for
dehydration with intravenous fluid resuscitation. Her electrocardiogram, echocardiogram and CT scan of the
head were all reported to have no abnormalities. In view of her chest pain and syncope a CT angiogram was
performed to rule out a pulmonary embolus. The scan failed to identify a pulmonary embolism but revealed a
posterior mediastinal mass which measured 3.8 X 2.1 X 4.8cm. Because of her continued orthostatic
hypotension despite adequate hydration the possibility of a neuroendocrine tumor was raised. Plasma
normetanephrine levels were found to be 1420 (0-145 pg/mL) and norepinephrine levels of 608 (0-111
µg/g creatinine). During her hospitalization she was never found to be hypertensive. The patient was
discharged with a plan for possible elective excision at a later date. Neuroendocrine tumor spect scintigraphy
was performed and identified a posterior chest paraganglioma. On a subsequent outpatient visit she was found
to be hypertensive and was started on amiodipine. Three months later, she underwent an elective excision by
video-assisted thoracoscopic surgery. Biopsy of the mass confirmed the lesion as a paraganglioma. Genetic
studies performed on the sample were negative for the VHL gene, SDH gene and RETPTC1 and RETPTC3.
Upon review of the published literature, we have been unable to identify any previous reports of a patient with
syncope as the sole presenting feature of a mediastinal paraganglioma. Additionally our patient did not have
hypertention at the time of presentation, which is unusual. In a case series reported by Brown et al. (2008) at
Mayo clinic, of the 14 patients with mediastinal paragangliomas all had a history of hypertension. We would like to emphasize that a syncopal episode in a young, otherwise healthy, adult should be evaluated with a thorough history and physical examination to search for possible etiologic clues such as orthostatic hypotension.

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LARGE GRANULAR LYMPHOCYTIC LEUKEMIA: THE GREAT IMITATOR OF FELTY’S SYNDROME

Thirty percent of patients diagnosed with Large Granular Lymphocytic (LGL) leukemia occurs in patients with Rheumatoid Arthritis (RA), the manifestations (neutropenia and splenomegaly) of which phenotypically resemble Felty’s syndrome. We hereby report a case of a patient with a long standing history of rheumatoid arthritis who presents with neutropenic fever and splenomegaly which was subsequently diagnosed as T-LGL based on bone marrow flow cytometry. A 67-year-old caucasian women presented with a 10 day history of high grade fever without any localizing symptoms of infection. She was previously prescribed ciprofloxacin by her PCP, which failed to improve her symptoms. Past medical history was positive for rheumatoid arthritis, which had been in remission since past 25 years. She denied any current use of medications for her disease. On examination she was febrile with a temperature of 101.4 F and tachycardic with pulse of 110/min. Rheumatoid factor was negative but anti-cyclic citrullinated protein antibodies was positive. Abdominal examination revealed mild splenomegaly by palpation and Castell’s point dullness on percussion. Non reducible, non tender deformities of the metacarpophalangeal and proximal interphalangeal joints were present with ulnar deviation of the fingers. There was no evidence of rash, hepatomegaly, lymphadenopathy, subcutaneous nodules or synovitis. Complete hemogram revealed pancytopenia with hemoglobin of 10.7 gm%, leucocyte count of 900 cells/mm3 with the absolute neutrophil count (ANC) of 108 cells/mm3 and platelets of 95,000. Computerized tomography of the abdomen revealed hepatosplenomegaly. Blood cultures were drawn and broad spectrum antibiotics were started for neutropenic fever. The differential diagnosis in the light of previous RA, neutropenia and splenomegaly included Felty’s syndrome and its close mimicker Large Granular Lymphocytic leukemia (LGL). Bone marrow biopsy was done which demonstrated CD 3+/CD5-/CD8+/ TCR αβ +ve T cells with reversal of CD4:CD8 ratio on flow cytometry. PCR for T cell receptor gamma (TCR γ) gene rearrangement was positive thus establishing monclonality of T cells. A diagnosis of T-LGL & CD8+ TCR & TCR & gene rearrangement was positive thus establishing monoclonality of T cells. A diagnosis of T-LGL & CD8+ TCR & gene rearrangement was positive thus establishing monoclonality of T cells. A diagnosis of T-LGL & CD8+ TCR & gene rearrangement was positive thus establishing T cell monoclonality. Methotrexate 15 mg/week was started as patient had symptomatic neutropenia. After 3 weeks of treatment there was no clinical improvement, prompting discussion of future alternative therapies. T-LGL is commonly associated with RA. Multiple mechanisms have been postulated for its pathogenesis including an antigen driven immune response and dysregulation of apoptosis. Diagnosis is based on TCR positivity for monoclonal proliferation of T cells. The treatment is mainly based on immunosuppressive therapy, with fludarabine and alemtuzumab considered as alternative options. As LGL in patients with RA shares clinical, hematological, immunological, and immunogenetic features with Felty’s syndrome, it may be an extension of the same disease spectrum.

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STOP: NO BLUE TO TREAT BLUE

Rasburicase is a recombinant urate oxidase that is used in chemotherapy induced acute Tumor Lysis Syndrome (TLS) with severe hyperuricemia. However this agent is known for causing methemoglobinemia in patients with Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency due to unopposed peroxide mediated oxidative stress. A 55-year-old African-American male presented with a left hip fracture after trivial trauma. The patient was hypercalcemic on presentation and a CT scan of the abdomen and pelvis revealed multiple lytic bone lesions. A diagnosis of lambda light chain multiple myeloma ISS stage II was established with a bone marrow biopsy, b2-microglobulin, and serum and urine protein electrophoresis. He developed acute renal failure which resolved with hydration and acute cord compression with paraplegia which did not respond
Diagnosed by CT scan. Air embolism, though a rare complication of vascular catheters, should be quickly suspected in an appropriate clinical setting. Our case was unique for a patent foramen ovale in the presence of sudden increase in right-sided pressure. Diagnosis is based on a high index of travel to lungs causing bubbles that may obstruct the pulmonary outflow tract producing air likely entered from either a fracture of catheter connection or from needle hub during insertion. Large artery catheters, arterial catheters, pacemaker placement, and intravenous contrast injections. Intravascular catheters have been described with central venous catheters, hemodialysis catheters, and pulmonary embolism was presumed. Air embolism secondary to radiation therapy. Lenalidomide was added, and bortezomib was reinitiated after radiotherapy. Upon starting lenalidomide, the patient developed massive tumor lysis syndrome (TLS), an undescribed phenomenon for multiple myeloma. A single dose of rasburicase 13.6 mg (0.2 mg/Kg) was administered. Ten hours later, the patient developed cyanosis with oxygen saturation of 80% by pulse oxymetry and 100% by ABG with Po2 of 453 mm Hg on 10 LPM oxygen. Methemoglobinemia was suspected due to saturation gap, and methemoglobin levels were elevated at 9.7%. Levels normalized without any intervention. G6PD level was sent and was low suggestive of underlying deficiency. This case involved two extremely unusual manifestations: The first was his extremely aggressive light chain myeloma, which rapidly progressed on treatment with bortezomib, and developed massive tumor lysis on lenalidomide. The second was rasburicase-induced methemoglobinemia, likely due to underlying G6PD deficiency. Lenalidomide is an agent commonly used for multiple myeloma. It has been described to cause tumor flare and tumor lysis syndrome in leukemia/lymphoma patients, though not in multiple myeloma. We hypothesize that TLS in our patient was due to the addition of lenalidomide, given the temporal relationship between its administration and the development of TLS. Approximately 10 cases of rasburicase-induced methemoglobinemia have been reported in the literature. It is recommended to check levels of G6PD prior to administration of rasburicase. However, the long turnaround time of G6PD screening precludes its use in severe TLS. Antidotal effects of methylene blue (MB) can prove deleterious in patients with G6PD deficiency as it can precipitate hemolysis from unreduced MB. Hence it is relatively contraindicated in patients with G6PD deficiency. The aim of this report is to recognize the fatal effect of TLS with lenalidomide in patients with multiple myeloma and recognize methemoglobinemia as a cause of hypoxia with saturation gap. Rasburicase which can cause methemoglobinemia in G6PD deficient patients results in cyanosis and the traditional treatment with methylene blue should be avoided in this subset of patients due to risk of hemolysis.

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"TO AIR IS HUMAN": IATROGENIC VENOUS AIR EMBOLISM FROM A CENTRAL VENOUS CATHETER

Venous air embolism is a potentially catastrophic complication related to vascular catheters. Acute hemodynamic derangements result from air lock at the pulmonary outflow tract leading to obstructive shock. Preventing further air entry and supportive measures are the cornerstone of management. We report a case of mass embolism of venous air embolism following the insertion of a central venous catheter, which was incidentally diagnosed by CT scan. A 87-year-old woman was admitted to the intensive care for hemorrhagic shock from gross hematuria. She was on warfarin for atrial fibrillation. On presentation, there was a sudden drop in hemoglobin to 4.9 gm% from 9.8 gm% with INR of 2.9. An 8 Fr introducer cordis was placed in the right internal jugular vein under ultrasound guidance. A triple lumen central catheter was introduced through the sheath. Standard precautions were taken during the procedure which included the Trendelenburg position during the insertion, flushing, and attachment of self-sealing valve at all the ports. The position of central line was confirmed with a chest radiograph and patient was volume resuscitated with crystalloids, blood, and blood products. One hour after central line insertion she underwent a CT of the abdomen for potential diagnosis of source of bleeding other than urinary tract. A large amount of intracardiac air was noted along the nondependent right atrium, right ventricle, and pulmonary outflow tract. The patient was hemodynamically stable and responsive to commands. She was placed in the left lateral Trendelenburg position and started on high flow oxygen. An attempt was made to aspirate air from the central line, without success. There were absence of any electrocardiographic findings revealed the absence of right ventricular strain, severe pulmonary hypertension or visualization of air. Agitated normal saline was negative for right to left intra-cardiac shunt. The patient remained hemodynamically stable and resolution of air embolism was presumed. Air embolism secondary to intravascular catheters has been described with central venous catheters, hemodialysis catheters, pulmonary artery catheters, arterial catheters, pacemaker placement, and intravenous contrast injections. In our patient the air likely entered from either a fracture of catheter connection or from needle hub during insertion. Large bubbles may obstruct the pulmonary outflow tract producing obstructive shock whereas small bubbles can travel to lungs causing non-cardiogenic pulmonary edema, shunt hypoxia or paradoxical emboli from patent foramen ovale in the presence of sudden increase in right-sided pressure. Diagnosis is based on high index of suspicion in an appropriate clinical setting. Our case was unique in that the air embolism was incidentally diagnosed by CT scan. Air embolism though a rare complication of vascular catheters, should be quickly
recognized as prompt restoration of circulation through supportive measures (left lateral position, hyperbaric oxygen therapy) can prevent peri-procedure mortality.

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CORRECT IS TO RECOGNIZE WHAT DISEASES ARE AND WHENCE THEY COME

A 45 year old Caucasian male presented with multiple painful rashes on his legs, low grade fever (100.6-102 degree F), tiredness, and mild pain in the wrists for 4 weeks. He applied hydrocortisone 0.5% cream on the rash, which did not help improve his rash. He had been treated for 2 episodes of uveitis in the prior 6 months. He denied cough, shortness of breath, weight loss, or night sweats. Physical examination was remarkable for tender, warm, raised nodular swelling over the anterior aspects of the lower legs that started as bright red lesions, later evolving into dusky red rashes suggestive of erythema nodosum. He was also found to have mild scattered wheezing over bilateral chest fields. His peak expiratory flow rate was 350 liter/min. A complete metabolic profile, complete blood count, ANA, and ESR were in the normal range. A chest X-ray and CT scan showed bilateral hilar adenopathy and mild lower lobe interstitial infiltrates. A PPD test was negative. Based on clinical and diagnostic features, our patient was diagnosed as having Lofgren’s Syndrome. Our patient was treated with prednisone 40 mg daily that was tapered to stop in 2 weeks. Patient had complete resolution of erythema nodosum and pulmonary symptoms. Patient’s broncoalveolar lavage results suggested sarcoidosis. At 6 months follow up patient was initiated on prednisone 10 mg daily for recurrence of pulmonary symptoms. The incidence of Lofgren’s syndrome is rare in Caucasian males; hence it requires a high index of suspicion and ruling out other possible diagnoses. Short term prognosis is excellent.

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WHERE DOES THE WOLF MEET RED RIDING HOOD? THE CASE OF LUPUS AND NEUROMYELITIS OPTICA: TWO DIFFERENT DISEASES OR ONE CONTINUUM?

Introduction: Neuromyelitis optica (NMO), or DeVic’s disease, is a rare inflammatory demyelinating disease that principally targets the optic nerves and spinal cord. Although it can mimic multiple sclerosis, the epidemiology, pathogenesis, clinical presentation, and prognosis distinguishes it from its more common counterpart. We present a case of confirmed NMO in a patient with newly diagnosed systemic lupus erythematosus. Case: A 41 year-old female presented with a three-week history of rapidly ascending quadriparesis, intractable vomiting, diaphragmatic weakness, and urinary retention. On initial presentation, physical examination revealed vital signs within normal limits and neurological motor exam showing 5/5 strength in all muscle groups except 4/5 on the left lower extremity with significant decreased grip strength in the left upper extremity. Sensations were intact throughout to all modalities. Dee tendon reflexes were 3+ in the upper extremities bilaterally and 2+ in both lower extremities. She exhibited bilateral dysmetria secondary to weakness. Her cardiac, gastrointestinal and pulmonary examination were within normal limits. MRI revealed a demyelinating lesion in area postrema. Based on the location of the area postrema lesion she was empirically treated for neuromyelitis optica with high dose steroids and plasmapheresis, but her weakness increased despite these treatments. At this point, additional testing revealed positive serologies for ANA, SSA, and SSB as well as significant proteinuria that suggested a diagnosis of systemic lupus erythematosus. She was started on cyclophosphamide followed by oral steroids and her symptoms improved to the extent that she became ambulatory. However, during the steroid taper she relapsed. She was found at this point to have high titers of aquaporin-4 antibodies, thereby meeting the revised diagnostic criteria for NMO. Discussion: Neuromyelitis optica is a demyelinating disease characterized by the development of cervical myelopathy and bilateral optic neuritis. Unlike multiple sclerosis, the disease tends to be monophasic, causes more severe myelopathy, has less brain involvement, and more commonly afflicts non-caucasian populations. A serum IgG autoantibody (NMO-IgG) directed against aquaporin-4 is thought to be a specific marker of this disease. There is a paucity of data indicating the optimal treatment of NMO, but most patients are treated with a combination of immunosuppression and plasmapheresis. Both NMO and SLE may be associated with longitudinally extensive transverse myelitis (LETM). However, the acute presentation of NMO with elevated aquaporin-4 antibodies in a patient with newly diagnosed SLE has never previously been reported.
DIABETIC MUSCLE INFARCTION: AN UNDERDIAGNOSED COMPLICATION OF DIABETES MELLITUS

Introduction: Diabetic muscle infarction (DMI), or diabetic myonecrosis is a rare complication of diabetes resulting from poor perfusion in the micro-vasculature. Patients present with acute onset of pain and swelling in an isolated muscle. Patients commonly have coexisting micro-vascular complications of diabetes including retinopathy, nephropathy or neuropathy. While the presentation is characteristic, the differential diagnosis is broad, often leading to extensive, sometimes invasive testing. Diagnosis can be established with T2-weighted MRI or histology. Case: A 42 year-old male with history of insulin-requiring diabetes mellitus complicated by ESRD due to diabetic nephropathy presented with acute onset of pain and swelling in his right anterior thigh. The pain was described as 8/10, sharp, continuous, non-radiating, exacerbated by movement and relieved only by rest. He denied any associated erythema, warmth, fever, motor weakness, or sensory loss of the extremity. He also denied any trauma or injections at the site. On presentation, the patient was afebrile and physical examination revealed significant tenderness over the right quadriceps muscle without motor or sensory deficits. Lab studies were notable for mildly elevated CK of 256, elevated ESR of 102 and WBC of 11.3. Arthrocentesis of the hip and duplex scan were negative. The patient was discharged with Trimethoprim-sulfamethoxazole for presumed cellulitis but was readmitted for persistent symptoms after completing the full course. Vancomycin and Piperacillin-tazobactam were started empirically and he underwent MRI without gadolinium that revealed subcutaneous edema and increased water content. Upon transfer to our institution, the clinical diagnosis of diabetic muscle infarction was made. The patient was treated with corticosteroids as NSAIDs were contraindicated due to ESRD, and his symptoms began to improve prior to discharge. Discussion: Diabetic muscle infarction is a rare complication of diabetes with about 200 reported cases. Cases have been reported from ages 13 to 81, averaging around age 40, more common in women than men. Pre-existing micro-complications of diabetes are among the most important risk factors for DMI. Most reported cases involve the thigh with cases involving the calf, upper extremities and abdominal or thoracic wall muscles also reported. Lab results in patients with DMI are often nonspecific. The most helpful study for diagnosis is T2-weighted MRI showing increased signal intensity in the infarcted muscle due to increased water content from edema and inflammation. Muscle biopsy reveals necrosis of muscle, associated edema and occasionally, fibrin occluding the lumen of small vessels. However, this is discouraged due to poor wound healing associated with micro-vascular compromise. Management of DMI is mostly conservative. Anti-inflammatory and anti-platelet agents lead to the most rapid resolution compared with rest and analgesics or surgical excision. Physiotherapy of the infarcted muscle may worsen the condition. Even after recovery, recurrence rates are high and long-term prognosis is poor.

RECURRENT VENOUS THROMBOEMBOLISM AS THE SOLE MANIFESTATION OF A MYELOPROLIFERATIVE DISORDER

Introduction: Although venous thromboembolism is a common clinical manifestation of the myeloproliferative disorders, it may present a diagnostic challenge when a patient who does not present with the other classical clinical manifestations or hematologic abnormalities that define these disease entities. Case: A 48 year-old Caucasian female with PMH of transverse sinus thrombus presented with the acute and progressive onset of epigastric pain described as a constant, non-radiating, pressure localized to the epigastric area that fluctuated in intensity from a 4/10 to a 10/10. The pain was exacerbated by food consumption but there were no other aggravating/alleviating factors. The pain was associated with multiple episodes of non-bilious, non-bloody vomiting and watery diarrhea. She denied the presence of constitutional symptoms, other gastrointestinal symptoms, livedorectilaris, headache, visual disturbances, erythromelalgia, or aquagenic pruritus. There was no personal history of recurrent miscarriages or family history of venous thromboembolism. Physical examination was significant only for epigastric tenderness, facial plethora and hepatosplenomegaly were not appreciated. Right upper quadrant ultrasound was performed which was consistent with occlusive portal vein thrombosis and mild splenomegaly. CT angiography with and without IV contrast for further evaluation of thromboembolism confirmed occlusive thrombus in superior mesenteric vein and non-occlusive thrombus in...
splenic vein in addition to the portal vein thrombus. The hemogram revealed a mild elevation in hemoglobin that ranged from 14.1 to 15.2 g/dL without leukocytosis or thrombocytosis. An acquired hypercoagulable state was strongly suspected due to recurrent venous thromboembolism in unusual locations, although an extensive previous work-up including factor V Leiden, cardiolipin antibodies, homocysteine, and deficiencies in protein C, protein S, and anti-thrombin III deficiencies was negative. JAK2 mutation, beta-2 glycoprotein, lupus anticoagulant, and flow cytometry to evaluate CD 55/59 were obtained during this admission. While awaiting test results, therapeutic anticoagulation was initiated to prevent progression and/or recurrence of thromboembolism and her abdominal pain improved. JAK2 mutation was identified after the patient was discharged on lifelong anticoagulation. Discussion: This case describes a patient who presented with recurrent venous thrombosis in unusual locations who tested positive for the JAK2(JAK2V617F) mutation but initially lacked the other phenotypical clinical features for polycythemia vera. The JAK2 mutation is commonly encountered in myeloproliferative disorder, especially in polycythemia vera where the sensitivity approaches 95%. However, this patient did not initially possess the clinical manifestations of polycythemia vera where the sensitivity approaches 95%. Therefore, it is essential to consider myeloproliferative disorders in the differential diagnosis of a patient who presents with thromboembolism in unusual sites even when the other typical symptoms, signs, and hematological abnormalities for these disorders are absent.

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INFLAMMATION GONE WILD: PRES IN THE SETTING OF ACUTE SEVERE PANCREATITIS

Introduction: Posterior Reversible Encephalopathy Syndrome (PRES) is a rare neurological condition that typically presents with acute onset of headaches, focal neurological deficits, seizures and nausea. The radiologic findings on CAT scan of the head include symmetric white matter defects in the occipital and parietal lobes; while MRI has the advantage of showing smaller, more focal abnormalities (1). Known causes of PRES include hypertension, eclampsia, certain anti-neoplastic or immunosuppressive agents, renal insufficiency, and other rheumatologic diseases (2,3). Case: A 25 y/o Caucasian female, with no past medical history, was admitted for acute severe pancreatitis. Her course was complicated with abdominal compartment syndrome requiring a total of 6 abdominal surgeries, anemia requiring transfusions, and acute renal failure requiring hemodialysis. She then developed subhepatic and peripancreatic abscesses that required drainage and antibiotics. Her stay in the ICU was complicated with HCAP, the need for pressors and a tracheostomy. She had a witnessed generalized seizure and CAT scan of the head revealed hypo-attenuating foci involving both the cortical gray matter and underlying white matter in the bilateral occipital lobes, consistent with PRES. Repeat CAT scan showed progression of the hypo-attenuating foci in both parietal occipital regions with development of low-density foci in the right frontal area of the brain. MRI of the head revealed abnormal flair/T2 confluent hyperintense areas in the parieto-occipital cortical and subcortical regions with scattered areas of blood products within the bilateral parietal lobes. Her systolic blood pressure was elevated up to the 180s and she remained febrile with a max temperature of 102F. Lumbar puncture revealed an opening pressure of 31 cm H2O but with normal cell count and protein. HSV PCR and AFB were all negative. Abdominal fluid collections continued to persist all through her recovery period. Conclusions: This young, otherwise healthy woman had her first bout of acute severe hemorrhagic pancreatitis, which was concurrently associated with Posterior Reversible Encephalopathy Syndrome. Since PRES is now diagnosed more on radiologic findings, it would be interesting to analyze if acute pancreatitis may be a potential cause of this devastating syndrome. Previously noted mainly in eclamptic patients, the newer diagnostic radiology may lead us into a better understanding of the PRES syndrome and its varied associations and causes. The rapid control of blood pressure still remains the cornerstone treatment of PRES, and should always be immediately addressed. References: 1. Hinchey, J., et.al. A Reversible Posterior Leukoencephalopathy Syndrome. N Engl J Med. 1996 Feb; 334: 494-500. 2. Kadikoy, H., et.al. Posterior reversible encephalopathy syndrome in a patient with lupus nephritis. Saudi J Kidney Dis Transpl. 2012 May; 23 (3): 572-6. 3. Kur, J.K., Esdaile, J.M. Posterior reversible encephalopathy syndrome—an underrecognized manifestation of systemic lupus erythematosus. J Rheumatol. 2006 Nov; 33 (11): 2178-83.
A HARD AND PERSISTENT BOIL: A CASE OF CUTANEOUS LEISHMANIASIS

Title: A Hard and Persistent Boil: A Case of Cutaneous Leishmaniasis  Authors: Selina Davis MS-IV*, Satyajeet Roy MD FACP*@
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Background: Persistent cutaneous abscesses point towards a myriad of clinical possibilities. A diagnosis of cutaneous leishmaniasis can be challenging due to rare clinical encounters and a variety of clinical presentations. Case: A 36 year-old male landscaper, who recently migrated from Nicaragua, with no significant past medical history presented to the outpatient internal medicine office with a 4 week history of blistering, pruritic, erythematous, edematous, and draining rash in the right forearm. Physical examination was conclusive of folliculitis and indurated lesions over right forearm, arm lymphangiectasia, and lymphadenopathy in the right medial elbow. He was prescribed Bactrim DS and Mupirocin ointment. A complete blood count and comprehensive metabolic panel showed no abnormality. The patient returned 2 weeks later with worsening lesion. On physical exam there was a 3 cm x 2 cm bilobed ulceration and accompanying adenopathy extending from the right medial elbow to axilla with thickening of lymphatic channels. Due to progression of the lesions and unresponsiveness to empiric antibiotic therapy the patient was admitted to the hospital for further evaluation. Skin biopsy did not demonstrate any organisms. Bacterial and fungal cultures from the lesion and AFB were negative. Due to the negative cultures and ulcerative pattern of the lesion, the patient was then started on a trial of itraconazole to cover suspected sporotrichosis or leishmaniasis, and a percutaneous smear was obtained. The results of the smear showed the presence of Leishmania. Due to lack of medical insurance coverage in the US, the patient decided to receive treatment in Nicaragua. At 1 month follow up visit and after completion of injectable sodium stiboglucone therapy his arm lesions and lymphangiectasia improved significantly. Discussion: Cutaneous leishmaniasis is transmitted by the female sandfly and its incubation period may range from two to eight weeks. It usually presents with papules that progress to ulcerations. The lesions may heal spontaneously within months or years; however they may also become disseminated. Due to its prevalence in the Americas from Texas to Argentina as well as in North African Countries and the Middle East, it is important to consider cutaneous leishmaniasis in the differential diagnosis in travelers to and immigrants from these regions. Conclusion: The differential diagnosis of non-healing lesions should be broad. In the case presented, the patient’s skin lesions were refractory to antibiotic therapy and progressed. A consideration of the patient’s travel history and geographic background were important in making the diagnosis.

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A PERFECT STORM FOR VALVULAR CATASTROPHE

Background: Infective endocarditis (IE) is a catastrophic illness with mortality near thirty percent within first year of diagnosis. Here, we present a young female, chronically immunosuppressed from multifactorial morbidities who developed extensive, destructive endocarditis with aortic root abscess. Case: A 25 year-old female, diagnosed with systemic lupus erythematosus (SLE) at age 14, presented with abdominal pain and fever. At age 18, she developed End-Stage Renal Disease (ESRD) secondary to lupus nephritis and hemodialysis (HD) was initiated. At age 21, she had a renal transplantation and was started on chronic immunosuppressive therapy. Soon after transplant, she again required HD. The patient had refused multiple offers for arteriovenous fistula placement and was being dialyzed through multiple Shiley catheters. Blood cultures drawn as part of her fever evaluation grew methicillin-resistant Staphylococcus aureus (MRSA). Transthoracic echocardiogram revealed a large vegetation and abscess on the aortic valve (AV), for which she underwent an extensive cardiothoracic surgery, including placement of a mechanical aortic valve, with coronary re-implantation, replacement of the ascending aorta, and tricuspid annuloplasty. The procedure was successful. Discussion: Staphylococcus aureus is the most prevalent species responsible for IE, isolated from one-third of all cases. Of S. aureus bacteremia cases in the United States, 7.8% (200,000) per year are associated with intravascular catheters. Without appropriate therapy, S. aureus endocarditis can progress rapidly to damage or destroy heart valves. Intracardiac consequences of endocarditis range from valvular vegetations, to catastrophic, extensive destruction beyond the valve leaflet. Complications include congestive heart failure,
myocardial abscesses, clinically relevant arrhythmias, purulent pericarditis and fistulous tracts. The aortic root is the most common site for intracardiac abscess, which is essentially a mycotic aneurysm of one of the sinuses of Valsalva. This life-threatening complication requires urgent attention. A contributory factor predisposing to valvular infections is immune phenomena like SLE, where accumulation of immune complexes, mononuclear cells and other agents form vegetations on heart valves, damage the valves, thus predisposing them to infection. Conclusion Our report presents valuable insight into the development and progression of IE/AV abscess in immunocompromised individuals. Medical conditions like chronic indwelling intravenous catheters highlight the immunologic susceptibility of this population and warrant timely risk stratification and intervention.

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CHARACTERISTICS OF HIGH GRADE GLIOMAS IN PATIENTS DIAGNOSED AT AGE 18 THROUGH 45

Objectives: To identify the epidemiological, clinical and molecular characteristics in patients less than 45 yrs of age diagnosed with high grade gliomas. Background: High grade gliomas are extremely aggressive tumors associated with a very poor prognosis. They are usually seen in the older age groups (median age of 45- and 60 yrs for patients with Grade III gliomas and GBM respectively). Methods: We have identified at least 24 patients less than 45 years of age with histological findings of Grade III glioma or GBM between 2005-12. We have examined the objective data obtained from patients’ charts to detect any potential risk factor(s). We have also examined archived tumor tissues from these patients to look for molecular differences at the RNA/ protein level. Conclusion: Our study shows the characteristics of high grade glial neoplasms in the younger age group of patients as compared to the older population in an attempt to identify better therapeutic targets that can improve the outcome for this patient population.

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REVELATION OF LEFT VENTRICULAR NON COMPACTION

Left ventricular noncompaction (LVNC) is a rare disorder of endomyocardial morphogenesis during embryonic development. The clinical presentation of LVNC can vary greatly due to the temporal variation of myocardial maturation. We report a case of LVNC in a 25-year-old male presenting after a traumatic injury to his chest that followed a rapidly deteriorating course. Case: A 25-year-old male presented with new onset dyspnea and orthopnea two weeks after blunt trauma to his chest. He denied other constitutional symptoms other than subjective symptoms of weakness and malaise. Physical examination was pertinent for tachycardia, caudally displaced PMI, and a positive hepatojugular reflex. Patient was noted to have a 2/6 systolic murmur at the right sternal border, split S1 normal S2 along with a S3 and S4. Chest examination was positive for decreased breath sounds on the right. Initial lab work showed a marked elevation of Pro-BNP. A CT of his chest was done which showed bilateral pleural effusions and cardiac enlargement. Subsequently, echocardiogram showed a poor ejection fraction of 15% with global hypokinesis, LV thrombus, and marked trabeculation of the left ventricle. He was diagnosed with acute decompensated heart failure and treatment was initiated. Due to the trabeculations an MRI was ordered. MRI confirmed lateral and inferolateral deep intertrabecular recesses. Hospital course was further complicated with episodes of ventricular tachycardia. Heart failure therapy was continued with plans for ICD placement. Discussion: Left ventricular noncompaction (LVNC) is a rare disorder that affects myocardial maturation during embryonic development. Absence of symptoms may cause a delay in diagnosis. Symptomatic patients usually present with heart failure, arrhythmias, thromboemboli, or even sudden death. It is believed that the lack of coronary arterial flow causes subendocardial hypoperfusion leading to ventricular failure. Echocardiography is usually the first diagnostic test performed. Better visualization of the trabecular pattern can be seen on cardiac MRI, which is now considered the method of choice. Management involves treatment of heart failure and prevention of complications such as arrhythmias and thromboembolic events. Definitive treatment with cardiac transplantation may be indicated in severely decompensated cases. Conclusion: Given the potential for life threatening consequences and deteriorating clinical course with a dismal prognosis in symptomatic patients, diagnosis of this condition is imperative. It remains challenging given the lack of a diagnostic gold standard test for diagnosis. Echocardiography and cardiac MRI both have various
diagnostic criteria which aide in the diagnosis of left ventricular noncompaction. The sudden onset of symptoms in a young patient after a traumatic injury with rapid deterioration in clinical course should raise questions of a cardiomyopathy.

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ASSOCIATION OF HYPOTHYROIDISM AND OSTEOPOROSIS-OSTEOPENIA WITH LEVOTHYROXINE REPLACEMENT THERAPY

Background: Thyroid hormone and hyperthyroidism have a clear association with osteoporosis and bone loss due to the direct effect of thyroid hormone on osteoclastic activation. Hypothyroidism is a chronic disease requiring longstanding treatment with levothyroxine. The effect of levothyroxine therapy on bone mineral density (BMD) in hypothyroid patients with a therapeutic range of thyroid stimulating hormone (TSH) remains understudied. Objective: To study the effect of levothyroxine therapy on BMD in hypothyroid patients with normal range of TSH. Method: Retrospective study of the first 30 adult internal medicine office patients with hypothyroidism presented in 2011, who had a normal range of TSH with levothyroxine therapy, and underwent a DEXA scan based on current guidelines. Results: All patients were females in 40 to 91 year age range. Seventy three percent of the patients had a low BMD (osteoporosis or osteopenia), while only 27% had a normal BMD. Twenty percent three percent patients had vitamin D deficiency, 6.7% had chronic kidney disease, and 6.7% had rheumatoid arthritis. Twenty three percent patients were taking hydrochlorothiazide while 17% took other diuretics, 27% were on proton pump inhibitors, 17% were on prednisone, 13% were taking bisphosphonate, and 7% were on methotrexate. None of the patients had a history of small bowel disease, malabsorption, or parathyroid disorder; and no one was on antiepileptic agents or progestins. Seventy percent patients were on supplemental vitamin D therapy. All patients had normal levels of calcium, magnesium, and alkaline phosphatase. Limitations: Possible confounding variables such as genetic predisposition to osteoporosis, coexistent diseases and use of medications other than levothyroxine that may alter calcium homeostasis may result into secondary osteopenia or osteoporosis. Conclusion: Patients with well controlled hypothyroidism on levothyroxine replacement therapy have high association with low BMD. Additional studies are needed to consider routine BMD assessment in this group of patients who otherwise do not qualify under current guidelines of BMD assessment, in order to assess potential bone loss and implement measures to prevent it.

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A NOT SO SWEET SYNDROME

Introduction: Leprosy, caused by Mycobacterium leprae, is a rare disease seen in United States predominantly among immigrants from developing countries. We report a case of a 27-year-old male presenting with a recurrent rash that resolved with steroids, leading to a delay in diagnosis. Case: A 27-year-old male immigrant from Brazil presented with a 6-month history of a nodular, erythematous painless rash without accompanying pruritus, numbness, or paresthesias. The rash initially appeared over the anterior aspect of his lower extremities and resolved after receiving prednisone and amoxicillin. Three months later, the rash recurred over his extremities, trunk, and face and was now associated with constitutional symptoms. The lesions continued to respond to multiple courses of steroid treatment, only to recur after completion of treatment. On examination, a diffuse, non-tender, blanching erythematous nodular rash was present over the face, ears, trunk, and extremities but spared the palms and soles. Sensation to all modalities was intact over the lesions. The differential diagnosis included Sweet syndrome (neutrophilic dermatosis), bacillary angiomatosis, erythema nodosum (panniculitis), urticarial vasculitis, and infections secondary to mycobacteria and fungi. His labwork was significant for an elevated ESR, but serologies for HIV, syphilis and hepatitis were negative. A biopsy of the rash was performed and the patient was started on prednisone for a presumed diagnosis of Sweet syndrome. Biopsy subsequently revealed lepromatous leprosy. Discussion: Leprosy, caused by Mycobacterium leprae, represents a spectrum of clinical and histopathologic diseases determined by the interplay between host responses and bacterial factors. Rudley and Jopling classified leprosy into Tuberculoid, Borderline tuberculoid, Mid-borderline, Borderline lepromatous, lepromatous, and an indeterminate category. Leprosy is diagnosed with the help of the slit and scrape technique where biopsies are procured from cooler
regions of the body including the ear, nasal mucosa, and distal extremities where the mycobacterial load is greatest. For treatment purposes, leprosy is classified into paucibacillary and multibacillary types based on mycobacterial burden. Antileprosy medications include dapsone, clofazimine and rifampin. Two types of immune reactions may be seen in leprosy and may either precede or follow treatment. Our patient presented with Erythema Nodosum Leprosum (ENL), or a type II lepra reaction, resulting from a type III hypersensitivity reaction to the high bacillary burden of leprosy. It is characterized by systemic symptoms and evolution of new lesions and classically responds to treatment with corticosteroids, only to recur when tapered. Conclusion: The internist should be cognizant of the incidence and clinical presentation of this rare disease in the immigrant population in order to confirm the diagnosis in a timely manner and avoid empiric steroid use that may mask the disease and delay the diagnosis.

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A STUDY OF VITAMIN D STATUS IN PRIMARY CARE PATIENTS

Background: Vitamin D deficiency has been reported worldwide in all age groups. Current medical research has shown a role of vitamin D deficiency in cardiovascular, immunological, and neuromuscular pathophysiological processes, in addition to the alteration in bone metabolism. Objectives: 1. To study the prevalence of low vitamin D level in patients presenting with various medical conditions in a suburban primary care office. 2. To study the association of chronic medical conditions with low vitamin D levels. Method: Retrospective review of medical records of 365 patients who presented in a suburban primary care office with various medical conditions and had an assessment of vitamin D levels from January 1, 2011 through December 31, 2011. Results: Forty four percent of the patients had a low vitamin D level (25 OH vitamin D <30 ng/mL). These patients ranged from 22 to 90 years of age, and 90% were females. Associated chronic medical conditions include cardiovascular disease (8.79%), stroke (4.4%), diabetes mellitus (10.06%), hypertension (45.91%), hyperlipidemia (57.86%), hypothyroidism (28.93%), and osteoporosis (5.03%). None of the patients had a history of small bowel disease, malabsorption, or parathyroid disorder; and no one was on antiepileptic agents or progestins. All patients had normal levels of calcium, magnesium, and alkaline phosphatase. 57.23% percent patients who had low vit D were taking over-the-counter vitamin D supplements, while 50.97% of patients with normal vitamin D level took supplemental vitamin D. Discussion: Vitamin D is an essential fat-soluble vitamin, traditionally known to play a crucial role in calcium and phosphate metabolism. However, recent studies have indicated a role of vitamin D in diverse processes as cellular proliferation, insulin production, regulation of angiogenesis, renin inhibition, production of macrophage cathelicidin and possible role in cardiovascular protection- though the role remains debated. As fewer foods have vitamin D, it is not surprising that sub-clinical Vitamin D deficiency is easily under-reported. Many patients who are seen in routine primary care practice harbor co-morbid medical conditions that may involve one or more of the above patho-physiological pathways. Diagnosis of vitamin D deficiency and prompt correction may prevent development of and possibly, improve the co-morbid conditions. Conclusion: Vitamin D deficiency is common in suburban community, and it is associated with chronic medical conditions, like cardiovascular disease, stroke, diabetes mellitus, hypertension, hyperlipidemia, hypothyroidism, and osteoporosis-osteopenia. Daily intake of over-the-counter supplemental vitamin D3 in different strengths does not prevent development of vitamin D deficiency.

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DOES THE URGENCY OF ENDOSCOPIC RETROGRADE CHOLANGIOPANcreateography (ERCP)/PERCUTANEOUS BILIARY DRAINAGE (PBD) IMPACT MORTALITY AND DISEASE RELATED COMPLICATIONS IN ASCENDING CHOLANGITIS? (DEIM-I STUDY)

Background: The Tokyo Guidelines have greatly impacted the management of ascending cholangitis [3-6]. Though ERCP is the favored modality for biliary decompression, no evidence exists for the timing of ERCP [7]. The DEIM-I study set out to determine if the time from patient presentation to biliary decompression impacted in hospital all cause mortality in ascending cholangitis. Methods: DEIM-I cohort study was a single-blinded and consisted of 250 subjects with moderate to severe ascending cholangitis who underwent ERCP/PBD. Subjects were randomized into quartiles based upon time from presentation until ERCP/PBD. The primary
outcome utilized logistic regression to estimate relative risk (RR) of all cause, in hospital mortality with time to procedure as the predictive covariate. Secondary outcomes were analyzed using multivariate logistic regression and included; multiple organ failure (MOF), sepsis, systemic inflammatory response syndrome (SIRS), surgical incidence, hospital readmission and length of stay (LOS). Results The risk for hospital mortality was significantly less when biliary drainage was performed within 11 hours, compared to >42 hours (RR 0.34, 95 % CI 0.12 to 0.99, P= 0.049)(Figure 2). Hospital readmission was lower in subjects who underwent biliary decompression less than 11 hours, when compared to those greater than 22 hours (Figure 3). Subjects who underwent biliary decompression within 21 hours had significant higher risk for surgery compared to those 22-42 hours (Figure 3). Conclusion The relative risk of all cause in hospital mortality was lower in subjects who underwent biliary decompression in under 11 hours compared to greater than 42 hours.

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SUCCESSFUL BRONCHOSCOPIC THERAPY OF A LARGE POLYPOID ENDOBRONCHIAL GRANULAR CELL TUMOR

Introduction: Granular cell tumors (GCTs) are uncommon benign neoplasms that typically occur in the skin, tongue and breast and rarely in the lungs as endobronchial lesions. Surgical resection is recommended for large tumors. We present a case of successful local bronchoscopic therapy of a large endobronchial GCT in a patient with high surgical risk. Case Report: A 34 year-old female presented with progressive positional dyspnea and cough worsening since 2 months. CT chest revealed a 2.9 x 2.7 cm tracheal mass at the level of the thyroid gland invading the surrounding tissue with severe luminal narrowing; and a 4.4 x 2.5 cm polypoid mass in the left mainstem bronchus extending beyond the airway lumen into the mediastinum. Flexible bronchoscopy demonstrated significant narrowing of the proximal trachea and a polypoid lesion occluding the left mainstem bronchus that was biopsied. Pathology showed a submucosal GCT. Surgical resection was discussed with the patient but she refused as this carried a significant operative morbidity and mortality and the possibility of a permanent tracheostomy. A decision to perform bronchoscopic therapy of the left mainstem tumor was taken. During flexible bronchoscopy the pedunculated tumor was completely detached with a snare loop and removed from the lumen using the cryotherapy probe. The left lower lobe bronchus was still occluded by submucosal tissue and argon plasma coagulation was used to remove it. Then the left mainstem bronchus was dilated with a 12 mm balloon obtaining good results. The patient had significant improvement in symptoms and will have a control bronchoscopy in one year. Discussion: GCTs are slow growing benign tumors from neuroectodermal origin. GCT occurs commonly in the skin, tongue, and breast, with only 6%–10% of bronchial GCT occurring endo- bronchially. They are usually located in the trachea or mainstem bronchus and can present with hemoptysis, recurrent pneumonia, atelectasis and cough. Full thickness invasion of the bronchial wall occurs in 40% of cases and multifocal disease in up to 25%. Surgical resection is recommended for lesions of more than 8 mm as they are likely to invade the bronchial wall and as they have a high rate of recurrence after local therapy. The high operative risk in this case precluded this approach, so bronchoscopic removal was done with significant symptomatic improvement. This case demonstrates that local therapy for large GCTs can be effective in patients who are not surgical candidates. Conclusion: Bronchoscopy plays a fundamental role in the management of these patients. Even though the treatment of choice is surgical resection of the tumor we have successfully shown that bronchoscopic removal is not impossible.

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A CASE OF MISSED PULMONARY EMBOLISM PRESENTING AS SYNCOPE

Introduction: Syncope is defined as a symptom of transient loss of consciousness due to global cerebral hypoperfusion. The etiologies of syncope include neurocardiogenic, orthostatic hypotension, cardiovascular, and neurologic causes. This is a case of missed pulmonary embolism (PE) which presented with syncope, EKG changes, and elevated cardiac enzymes, and was initially misdiagnosed as acute coronary syndrome. Case Presentation: A 70 year old Caucasian male with a past medical history of type 2 diabetes mellitus, hypertension, dyslipidemia, and chronic back pain presented to the Emergency Department after a pre-syncopeal episode. He did not have complaints of chest pain, palpitations, or dyspnea. He lived alone, was independent of activities of daily living, and denied recent immobilization. His vital signs were: BP 180/84, HR
123, RR 18, and SpO2 96% on room air. Physical exam was notable for trace bilateral lower extremity edema. EKG showed RBBB with ST elevation in aVR and ST-depression in Leads I and aVL. On admission troponin-T was elevated at 0.13 and peaked to 0.34 and CK was within normal limits. D-dimer was not ordered. Transthoracic echocardiogram showed LVEF> 65%, a moderately enlarged right ventricle cavity, severely reduced RV systolic function and borderline pulmonary artery hypertension. The patient was discharged when no angiographic evidence of coronary artery disease was found on cardiac catheterization. Seven weeks later the patient was readmitted after progressive dyspnea and a syncopal episode. He was tachycardic and O2 saturation was 92% on room air. CT of the chest found massive, bilateral, nearly occlusive pulmonary emboli. A repeat echocardiogram showed a large thrombus floating from the tricuspid valve into the right atrium. Duplex ultrasound of the lower extremities confirmed an occlusive DVT in the right common femoral vein and nearly occlusive DVT of the left popliteal vein. The patient was admitted to the ICU and treated with TPA. He improved clinically and was discharged home on warfarin. On initial workup of hypercoagulable state, factor V Leiden mutation was negative and CT of chest, abdomen and pelvis did not reveal malignancy. Discussion: Pulmonary emboli occur when thrombi in the deep veins lyse and dislodge in the pulmonary arterial system. Although the classic presentation of PE includes tachypnea, tachycardia, acute dyspnea, pleuritic chest pain, cough, and hemoptysis; pulmonary emboli have a widely variable presentation ranging from asymptomatic to fatal. Findings on CXR (Hampton Hump, Westermark sign) and EKG (RBBB, S1Q3T3 pattern) are neither sensitive nor specific. This case demonstrates the importance of forming a thorough differential diagnosis and pursuing secondary ideas if initial testing does not reveal the underlying cause of the chief complaint. A broad approach will aid in diagnosing a potentially life threatening condition that may not initially present with classic signs and symptoms.

UMDNJ - JOHNSON (RANITA SHARMA, MD)

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THE EFFECT OF LANGUAGE CONGRUENCY ON THE OUT-OF-HOSPITAL (OOH) MANAGEMENT OF CHEST PAIN

Background: Although language barriers are associated with disparities and delays in cardiovascular care in the hospital, little is known about how they affect care in the prehospital setting. The out-of hospital (OOH) management of chest pain is protocol driven, however language barriers likely affect the care provided by Advanced Life Support (ALS) paramedics aboard Emergency Medical Services (EMS) units. Methods: This is a four-year retrospective cohort study of New Jersey patients who called 911 for chest pain from April 2008 to January 2011. Using an electronic medical record system, we examined the association between language barriers and total On-Scene-Time (OST) spent by ALS paramedics from one of the state''s largest EMS systems. A series of linear regression models were built to examine this association and we adjusted for confounding by demographic and clinical variables. Results: 11, 249 patients cared for by a NJ-EMS system for the treatment of chest pain were included in our study. Of these, 222 had a perceived language barrier (1.98%). Contrary to expectations, language barriers were associated with less OST (β=0.85400, p<.0028). After adjusting for demographic and clinical variables, a language barrier between patient and paramedic persisted as a significant independent predictor of less OST (β= -.7149, p<.0146). Paramedics spent less OST with Hispanics (β= -.3717, p<.0228), Asian/Others (β= -.5647, p<.0101) and patients with abnormal heart rates (β= -.007, p<.0001). Additionally, we found that the association between language barriers and OST varied significantly among racial/ethnic groups in adjusted models. Discussion: Among patients with chest pain, language barriers between patients and paramedics are associated with decreased total On-Scene-Time. Adjusted models indicate that paramedics spend less OST with Hispanics and Asian/Others (compared to whites) and those with abnormal heart rates, independent of the presence of a language barrier. Our study suggests that for those with chest pain, disparities and delays in cardiovascular care do not occur in the prehospital setting. Further research ought to concentrate on the quality of cardiac care rendered in formal medical setting in order to improve existing disparities.
TITLE: HEPATITIS B VIRUS AND HEPATOCELLULAR CARCINOMA: AN UNUSUAL CASE HIGHLIGHTS THE COMPLEXITIES OF SCREENING AND MANAGEMENT AMONG VULNERABLE PATIENTS

Introduction: Hepatocellular carcinoma (HCC) is the 3rd leading cause of cancer death worldwide and Hepatitis B Virus (HBV) accounts for 50% of cases. Although HBV transmission in developed nations has decreased, chronic infection persists in regions with suboptimal preventative health services. This case highlights the need for HBV screening among those who have been without access to healthcare. Case: A 38 y.o. male presented with a three week history of progressive nausea, vomiting and abdominal pain. The right upper quadrant pain was dull, without radiation and associated with emesis during meals. He reported dark urine and increasing jaundice of his skin for 1 week. He was without chest pain, dysuria, melena, hematochezia, pruritus, rashes and change in stool color. He denied weight loss, fevers and night sweats. He denied alcohol use, recent infections, previous hepatitis, blood transfusions, surgeries or use of illicit drugs. He emigrated from Mexico 6 years ago and last saw a physician in childhood. His physical was remarkable for scleral icterus and jaundice without asterixis or mental status changes. His epigastrium was diffusely tender; hepatosplenomegaly was present without shifting dullness or JVD. A Triphasic-CT revealed a nodular liver with innumerable hypo-attenuating lesions and an expansive mass invading the portal vein. An US-guided liver biopsy confirmed HCC. Diagnostic evaluation was positive for HepB surface-Antigen and HepB Core-Antibody (HBV-DNA:1,830 copies and AFP:2.3 units); HepB Core-AblgM and HBeAntigen were negative. His INR and bilirubin increased steadily and on day seven he developed tense ascites and became markedly hypoxic. A bedside paracentesis removed 2L of bloody fluid. He remained hypotensive despite intravenous albumin and fluid and was transfer to the ICU. He went into hemorrhagic shock, acute liver and renal failure and expired on hospital day nine. His family was counseled on HBV screening and the importance of prenatal care and vaccinations. Discussion: This case highlights the complexities of HBV and HCC. The patient’s presentation was rather unusual and it is likely that he has been a chronic carrier since birth. The point at which the patient transitioned from being a chronic latent carrier to an active sufferer is unknown. Epidemiological data is relatively sparse with regard to HBV transmission in non-endemic areas such as Latin America. US healthcare reform will lead to increased access to care for Latino patients such as this one who have been without medical care and age-appropriate screening. Vigilance with regard to Hepatitis is important and the identification of HBV carriers has the potential to decrease unnecessary mortality and morbidity.

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BEWARE OF BOVIS

Meningitis associated with Streptococcus bovis (SB) bacteremia (SBB) is an extremely rare complication based on our review of the literature dating back to the 1950’s. Its incidence, interestingly, has not increased proportionately in the HIV era. SB’s association with colon cancer is well documented. We present a case of SB meningitis in an HIV patient without definitive evidence of its source. A 58-year-old male with HIV and Hepatitis C presented with acute headaches, neck stiffness, and high fevers. He denied respiratory or abdominal symptoms, focal weakness, seizures or rashes. His spouse denied any behavioral changes. There was no history of dental procedures or infections, sick contacts, hospitalizations or specific infections related to HIV or Hepatitis C. He denied tobacco or alcohol use and reported a remote history of illicit drug use. His recent CD4 count was 129 with a viral load of 154,000. He reported taking antiretroviral therapy but occasionally missed doses. On exam, he did not appear toxic with a temperature of 100.3, heart rate 112, and a blood pressure of 135/73. He was lying in bed in a well lit room. Sharp disc margins were noted on fundoscopy. Oral thrush was absent. The neck exam was positive for Jolt sign. There were no focal neurological deficits, murmurs, or evidence of decompensated liver disease. Admission labs included a WBC count of 9200 (higher than baseline) with bandemia, hemoglobin of 11.2 (baseline) and a platelet count of 54,000 (baseline 120,000). Creatinine was 1.5 with a TBili of 2.2, AST 232 and ALT 109. DIC panel was
negative. With a leading diagnosis of meningitis with sepsis, a CT brain was first obtained and did not reveal any abnormalities. A lumbar puncture was performed. CSF results included normal protein and glucose levels with 29 nucleated cells (93% neutrophils). CSF gram stain showed abundant gram positive cocci in chains and pairs. Fluids, high dose Ceftriaxone, Vancomycin and empiric Dexamethasone were started until the organism was identified. Abdominal ultrasound raised suspicion for cirrhosis and noted gallstones without evidence of cholecystitis or choledocholithiasis. Blood, urine and CSF cultures grew SB susceptible to Ceftriaxone. Other medications were discontinued. Antiretroviral therapy was continued. Clinical improvement was noted over the ensuing days. The CBC returned to his baseline, hepatobiliary values improved and kidney injury resolved. The echocardiogram was normal. Repeat blood cultures were sterile and 2 weeks of parenteral Ceftriaxone was completed as an outpatient. The notorious association of colonic carcinoma with SBB prompted a pre-discharge colonoscopy. Surprisingly, no mass, polyps, diverticuli or ulcers were noted. Intact rectal varices and internal hemorrhoids were discovered. Could these grossly intact lesions have played a role in microscopic bacterial seeding or could his hepatobiliary system have been the culprit?

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EOSINOPHILIC ESOPHAGITIS PRESENTING AS ESOPHAGEAL DESQUAMATION: AN UNUSUAL CASE OF CHEST PAIN

Esophageal desquamation is a rare entity that is typically due to caustic ingestions and various autoimmune dermatological disorders. However, it has never been described in a pathologically confirmed case of eosinophilic esophagitis (EoE). Here, we describe a case of a previously healthy 43 year old female presented with 3 days of epigastric/chest pain. The pain was described as a dull discomfort, non-radiating, with associated odynophagia. Exacerbating factors included eating and deep inspiration. She has no history of asthma or atopy. The severity of the odynophagia lead to an upper endoscopy, which showed esophageal mucosal desquamation. Mucosal biopsies from the proximal and distal esophagus were obtained. A pathologic diagnosis of EoE was rendered based on the eosinophil count, presence of eosinophilic microabcess, eosinophil degranulation and involvement of proximal esophagus. The patient was started on a methylprednisolone dose pack which dramatically improved her symptoms over the course of one week. The patient eventually transitioned to swallowed fluticasone for maintenance therapy. A follow up endoscopy six weeks later showed normal esophageal mucosa and biopsies revealed normal squamous epithelium. The incidence of EoE has been steadily increasing since it was first described in the late 1970's. EoE is “a chronic, immune/antigen-mediated, esophageal disease characterized clinically by symptoms related to esophageal dysfunction and histologically by eosinophil-predominant inflammation.” Clinically, patients present with a variety of symptoms including dysphagia, heart burn/chest pain and food impactions. Esophagogastroduodenoscopy findings include linear furrowing, white papules, strictures, attenuation of the subepithelial vascular pattern and small caliber esophagus. Although the diagnosis of EoE is fairly common, the morphological appearance of a desquamative process is indeed a rare, and to our knowledge, an undocumented presentation of EoE. Esophageal desquamation has been associated with certain medications (bisphosphonates, NSAIDS and potassium chloride), irritants ranging from hot beverages to corrosive irritants, collagen vascular disorders and celiac disease. In the case presented, the patient was not on any medications that could cause esophageal desquamation nor had any history of ingestion of any potential irritants. Additionally she did not have a history suggestive of collagen vascular disorder or celiac disease. Finally, certain dermatological conditions with esophageal involvement must be excluded that have a similar morphological appearance on EGD such as Pemphigus vulgaris or Lichen planus. Ultimately, the patient did not have any dermatologic complaints or symptoms that would point to a dermatological cause. Clinicians should consider EoE in the differential diagnosis of patients presenting with desquamative injury to the esophagus. In the absence of caustic ingestion, biopsies should be taken from the proximal and distal esophagus to allow accurate diagnosis of EoE.
CISPLATIN NEPHROTOXICITY: THE ROLE OF POST-CHEMOTHERAPY HEMODIALYSIS

Cisplatin has a wide toxicity profile, including dose-dependent nephrotoxicity. As a result, the utilization of cisplatin in patients with renal insufficiency presents a difficult therapeutic challenge. We present the case of a patient with testicular cancer who underwent hemodialysis for preservation of renal function during chemotherapy. A 41-year-old male was admitted with a large right scrotal mass associated with 30-lb weight loss and intractable back and testicular pain. The mass had slowly enlarged over the course of one year, and had recently become ulcerated and necrotic. Upon admission, the mass was identified via lymph node biopsy, laboratory tests, and MRI as an intermediate-grade mixed germ cell tumor, with extensive local, lung, retroperitoneal, and paraaortic node metastases but without brain or bone metastasis. His presentation was further complicated by bilateral hydronephrosis and acute kidney injury (initial Cr 3.5, CrCl 21.6, no prior baseline available). Chronic high dose NSAID use, hypercalcemia of malignancy, and bilateral ureteral obstruction contributed to his renal dysfunction. Bilateral renal stent placement and a right nephrostomy tube resolved the hydronephrosis but the renal impairment persisted. At the time of diagnosis, the testicular mass was too extensive and necrotic to undergo immediate resection. Consequently, the patient underwent 4 cycles of neoadjuvant chemotherapy with cisplatin and etoposide. To preserve residual renal function during the nephrotoxic chemotherapy treatments, the novel approach of performing dialysis 60-90 minutes after each treatment was undertaken. Using this approach, the patient’s creatinine remained between 3.1-3.5 and he did not require regular hemodialysis. The patient had a protracted hospital course, which included several unusual opportunistic infections including Alcaligenes xylosoxidans sepsis and a Stenotrophomonas maltophilia urinary tract infection. Ultimately, the tumor burden was markedly reduced, allowing eventual surgical excision of the remaining tumor. Cisplatin is an antineoplastic agent with a wide toxicity profile that includes dose-dependent nephrotoxicity. As up to 35% of the cisplatin dose undergoes renal elimination, dose adjustments in patients with renal impairment are often necessary to avoid worsening renal function and progression of kidney disease. Cisplatin is additionally highly protein bound and undergoes a unique biphasic elimination, which includes a rapid initial clearance from the circulation (half-life 35.5-49.0 minutes) followed by a prolonged loss from the circulation and tissue compartments (half-life 58.5-73.0 hours). Dialysis of platinum compounds results in effective drug clearance only within a relatively short time after drug administration. Others have suggested lowering the dose of cisplatin in conjunction with dialysis; this may help to further limit nephrotoxicity, but at the potential expense of suboptimal chemotherapy treatment. In the setting of prompt hemodialysis, this dosage reduction may be unnecessary. This case illustrates the effectiveness of hemodialysis administered immediately post-chemotherapy to preserve residual renal function and optimize cancer treatment.

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IMMUNOSUPPRESSION LEADING TO AN INFECTIOUS TRIPLE THREAT

Multimodality immunosuppression is often the lifesaving treatment for many diseases. However, it also increases the risk for serious infections. We report a case of Granulomatosis with polyangitis (GPA, formerly known as Wegener’s granulomatosis) where the patient was challenged by an infectious triple threat with a bacterial, invasive viral and an overwhelming parasitic infection. A 58-year-old Nigerian male diagnosed with GPA by kidney biopsy two months prior to admission presented with abdominal pain and episodic blood-tinged diarrhea without fevers, chills, or dizziness. On exam, he did not appear toxic, was not febrile, orthostatic or tachycardic. A shallow, tender buccal mucosal ulcer was noted along with mild abdominal tenderness, hemipositive stool and evidence of healed vasculitic lesions on his lower extremities. Medications included cyclophosphamide, prednisone, and trimethoprim/sulfamethoxazole (PCP prophylaxis). He was HIV-negative. Labs revealed an elevated baseline creatinine with no other abnormalities. With a leading diagnosis of infectious colitis, empiric metronidazole was started while awaiting stool studies, cytomegalovirus (CMV) titers, blood culture results and colonoscopy. Cyclophosphamide was held and prednisone continued. Stool studies were positive for Clostridium difficile. High CMV antigenemia was reported and gancyclovir was initiated. The colonoscopy was complicated by an episode of emesis with aspiration, hypotension and progression to
respiratory failure requiring invasive mechanical ventilation. Profound hemoptysis from his endotracheal tube was noted. Pulse-dose steroids and plasmapheresis were initiated for suspected diffuse alveolar hemorrhage secondary to a possible GPA flare. Patient developed septic shock and worsening renal failure requiring vasopressor support, antibiotics, and dialysis. By this time, colon biopsies and previously collected fecal smears revealed Strongyloides stercoralis and CMV colitis. Steroids were immediately discontinued. With extended Ivermectin therapy (including liquid formulation for better absorption) and the addition of albendazole, eventual clearance of the parasite from tracheal aspirates and stool samples was reported. However, the patient succumbed to Klebsiella bacteremia and multi-organ failure. This case highlights the complications of immunosuppression, including Strongyloides hyperinfection syndrome in the setting of co-infection with C. difficile and CMV – the triple threat described above. While C. difficile and CMV are the more easily recognized pathogens in this setting, the patient's country of origin and travel history to Nigeria two years ago, uniquely predisposed him to Strongyloides. The sequence of clinical events are best explained by the aspiration of the parasite from the gastrointestinal tract during the episode of emesis, leading to an immediate aspiration pneumonitis followed by Strongyloides hyperinfection syndrome. Diffuse alveolar hemorrhage has been reported in this setting. The CDC estimates approximately 3 to 100 million people are infected with Strongyloides worldwide. Failure to detect Strongyloides infections in its asymptomatic state may result in potentially fatal disseminated strongyloidiasis. Currently, there are no screening guidelines for parasitic infections before starting patients on immunosuppressive therapy.

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NORMOKALEMIC THYROTOXIC PERIODIC PARALYSIS: A RARE ENTITY

Thyrotoxic periodic paralysis (TPP) is a rare complication of hyperthyroidism. It is most commonly associated with Graves’ disease but may occur with any etiology of thyrotoxicosis. It is characterized by transient, recurrent episodes of flaccid muscle paralysis affecting proximal muscles more severely than distal muscles. It is most often seen between the third and fifth decades of life, with a predilection for Asian men. It is almost always associated with hypokalemia. There have been very few reported cases of TPP associated with normokalemia. Here we present the case of a 41 year old Asian man with TPP found to have normal potassium levels. The patient had no significant past medical history and presented to the emergency department with an episode of bilateral lower extremity and proximal upper extremity weakness that lasted about 5 hours upon waking up. He did not have sensory disturbances in any extremity. His symptoms resolved spontaneously after a few hours. He reported another episode of lower extremity paralysis upon awakening two weeks ago that lasted about ten minutes. He had been noticing some soreness along his medial thighs for the last month. His review of systems was positive for increasing fatigue with exercise, intermittent diarrhea, unintentional weight loss of 10 pounds in the past three months, heat intolerance, occasional palpitations, anxiety, and hand tremors. His physical examination was remarkable for tachycardia, a diffusely enlarged thyroid gland with small palpable nodule on the right, presence of a thyroid bruit, normal neurological exam, and a fine hand tremor. His laboratory studies were significant for a normal potassium level of 3.9, thyroid stimulating hormone (TSH) of less than 0.005, and free thyroxine (fT4) elevated at 3.90, normal MRI of the brain and lumbar puncture studies. His thyroid uptake scan was consistent with Graves’ disease and he was started on propranolol and methimazole which resolved his symptoms. Most cases of TPP are associated with hypokalemia. Thyrotoxic normokalemic periodic paralysis should be kept in mind as a cause of acute muscle weakness to avoid missing a treatable and curable condition. Symptoms of TPP usually resolve once hyperthyroidism is treated appropriately.
ISOLATED NEUROSARCOIDOSIS: A DIAGNOSTIC CHALLENGE AND A MASQUERADING DISEASE

Intro: Sarcoidosis with involvement of the CNS occurs in a small subset of patients (5-15%). However, isolated neurosarcoidosis without signs of systemic disease is a rare and challenging diagnosis. Case: 45 year old African-American woman presented with 2 weeks of worsening right frontal headache and confusion, without associated symptoms suggestive of a systemic inflammatory or infectious process. On initial presentation to the emergency department, she was treated for acute sinusitis and discharged. Upon a second presentation ten days later, she was noted to be encephalopathic and incontinent. No structural abnormalities were noted on CT head, and CSF studies revealed 120 nucleated cells with a lymphocytic pleocytosis (61%), low glucose (23), and elevated protein (131). Aseptic meningitis, likely viral in etiology, was considered the most likely diagnosis at this point and patient was discharged with close follow up. The patient returned to the emergency department 5 weeks later with recurrent headache, blurry vision, nausea, vomiting, and night sweats. She was re-hospitalized. Repeat CSF analysis was unchanged and additional CSF studies including West Nile, Cryptococcus, HSV, VDRL, Lyme, and HIV were all negative. The patient reported a negative PPD 4 years prior and no interim exposure. A MRI brain revealed leptomeningeal enhancement, particularly of the basal cisterns and left frontal lobe, which were concerning for tuberculous meningitis. A large volume CSF AFB culture was collected and empiric therapy for meningeval tuberculosis was initiated with rifampin, isoniazid, pyrazinamide, ethambutol, pyridoxine, and dexamethasone. One month later, the patient was again hospitalized because she failed to improve. AFB culture remained negative, as did TB PCR, PPD and Quantiferon test. CT chest/abdomen/pelvis showed two stable 5-6mm pulmonary nodules and pelvic adenopathy. Both serum and CSF ACE levels were found to be modestly elevated. Additionally, a meningeal biopsy was performed and yielded non-necrotizing granulomata, supporting the diagnosis of a granulomatous inflammatory disease. Because the CT chest was negative for pulmonary lymphadenopathy, the patient was diagnosed with isolated neurosarcoidosis. Treatment with high dose corticosteroids resulted in symptom resolution. This case illustrates the diagnostic challenge of isolated neurosarcoidosis. Biopsy can be difficult to obtain; however, if feasible, remains vital for diagnosis and treatment.

LIVER ABSCESS TO EMPIEYMA: COMPLICATION OF PERCUTANEOUS DRAINAGE

Introduction: Current standard treatment for a pyogenic liver abscess consists of percutaneous drainage and antimicrobial therapy. Although complications from percutaneous drainage and catheter placement are rare, these complications can have profound sequelae and clinicians should be aware of them. Case Presentation: A 43 year-old male presented to the ED with severe right sided chest and flank pain for one day preceded by malaise and rigors for the past 2 weeks. CXR showed elevation of the right hemidiaphragm and small right pleural effusion. CT scan of the abdomen and pelvis showed a 6.6x4.4cm hypoattenuating lesion within the right posterior liver, and a moderate right sided pleural effusion with no apparent communication to the liver lesion. On the second day of hospitalization, the patient underwent CT guided percutaneous drainage of the liver abscess and catheter placement. 170cc of pus was drained during the procedure. Liver abscess culture grew a pan sensitive Klebsiella pneumoniae strain and the patient was started on IV antibiotics. On hospital day 3, the patient developed respiratory distress with acute onset of shortness of breath and hypoxemia. Repeat CXR showed complete opacification of the right lung field. Emergent thoracentesis was performed and 1 liter of fluid was removed. Chest tube was not initially placed out of concern for thoracobiliary fistula. ERCP was performed and showed no evidence of filling defect or leak suggestive of a fistula. Pleural fluid culture demonstrated the same Klebsiella strain as above. Repeat CT scan of the chest demonstrated a moderate right sided pleural effusion into which a chest tube had been placed, and a separate loculated large subpulmonic effusion with decrease in the size of the liver abscess. Following the CT scan, a second chest tube was placed into the subpulmonic collection. Cultures of the collection again grew the same Klebsiella strain. The patient improved with chest tube drainage of the collections, but did eventually require VATS for removal of fibrous debris and empyema with lysis of adhesions and decortication. He was discharged on hospital day 12 with a course of oral antibiotics. Discussion: Pulmonary manifestations are seen in 50% of
cases of liver abscesses ranging from atelectasis to elevation of the hemidiaphragm to sympathetic pleural effusions. However, the acute respiratory decompensation and the dramatic change in CXR findings in this patient immediately post-procedure suggest a complication of the procedure itself (i.e., rupture of the abscess and/or inoculation of the pleural space). Complications of percutaneous abscess drainage are infrequent, but as this procedure is increasingly commonplace, internists must be cognizant of rare complications to expedite both their diagnosis and treatment.

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ASYMMETRIC HYPERTROPHIC CARDIOMYOPATHY AND UNEXPLAINED SYNCOPE AUTHORS:
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Authors: Meruka Hazari MD, Kelly Hanretta MD, Kashif Janjua MD Authors’ Affiliation: University of Medicine and Dentistry of New Jersey- Robert Wood Johnson Medical School Introduction: Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disorder, and asymmetric hypertrophic cardiomyopathy (ASH) is an infrequent, but known variant. Absence of physical exam findings, inconclusive ECG, and lack of definitive echocardiographic findings in the setting of ASH is rare. We report a case of unexplained syncope secondary to ASH diagnosed conclusively by cardiac MRI. Case description: A 46 year old female with a history of hypertension and alcohol abuse presented one day after her first syncopal episode. Several hours after the episode she developed chest tightness which prompted the hospital visit. She reports sub-acute, progressive dyspnea on exertion without any limitation in her routine activities over the prior few weeks. Her syncopal episode was without prodromal or post-ictal symptoms. Initial physical exam was significant for a blood pressure of 204/97, pulse of 124, and facial contusions, however there was notable absence of any murmurs or focal findings. Presenting ECG showed sinus tachycardia with QT prolongation and left ventricular hypertrophy (LVH). Blood pressure and pulse readily responded to beta-blocker administration. Troponins were negative. Subsequent telemetry monitoring failed to demonstrate any arrhythmia aside from sinus tachycardia. However, an ECG obtained the following morning showed intermittent anterolateral ST segment elevations despite the patient being asymptomatic. Echocardiography showed mild LVH without signs of outflow tract obstruction, preserved ejection fraction with diastolic dysfunction, and mild mitral and tricuspid regurgitation. These common, nonspecific echocardiographic findings, in the clinical context of our patient prompted a cardiac MRI, which showed asymmetric thickening of the left ventricular wall from the proximal to mid septum, without subaortic left ventricular outflow tract obstruction. A cardiac catheterization was unremarkable. Given the findings of ASH, the patient had an implantable cardioverter defibrillator (ICD) placed for primary prevention of sudden cardiac death. Discussion: This case illustrates the need for awareness of rare causes of syncope such as ASH, which cannot be elicited by physical exam or ECG, and have equivocal echocardiographic findings. Cardiac MRI has proven to be a useful tool in the diagnosis of variants of HCM in unexplained syncope with transient evidence of LVH by ECG, without conclusive findings on echocardiography. Identification of these patients is critical, as they can subsequently develop sudden cardiac death, and require placement of an ICD.

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CARBAMAZEPINE-INDUCED DRESS (DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS)

Carbamazepine is an antiepileptic medication used for seizures, mood disorders, and neuropathic pain. The most common serious adverse effects of carbamazepine include hyponatremia secondary to a syndrome of inappropriate antidiuretic hormone secretion, hepatitis, and cytopenias. Rarely, carbamazepine has been associated with severe hypersensitivity reactions such as Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis, and Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS). We report a case of carbamazepine-induced DRESS occurring decades after drug initiation and complicated by an indolent coexisting parasitic infection. The patient is a 66-year-old Costa Rican male with a history of seizure disorder who presented with two days of fever, facial edema, and a pruritic, erythematous rash that started on his
abdomen and spread to his extremities. Home medications included carbamazepine, initiated at age 21, valproic acid, enalapril, and aspirin. Physical exam revealed facial and periorbital edema with a diffuse, blanching, maculopapular rash on the trunk and extremities. Laboratory findings were significant for hyponatremia of 117, AST of 35 and ALT of 32, which increased to 54 and 194 respectively, and eosinophilia of 10%, which increased to 31%. Further studies revealed positive HHV-6 IgG titers, negative HIV, and positive stool culture for Strongyloides stercoralis. During the hospital course, the patient developed fevers up to 103° F, a severe nonproductive cough, and increasing confluence of his rash. CT scan of the chest revealed bilateral effusions and extensive lymphadenopathy. Carbamazepine was held due to hyponatremia and a high clinical suspicion for DRESS based on the constellation of findings of fever, rash, facial edema, lymphadenopathy, hepatitis, HHV-6 seropositivity, and eosinophilia. A skin biopsy was obtained to confirm the diagnosis. Strongyloides, an intestinal parasite common in the tropics, can present similarly with a syndrome of eosinophilia, pulmonary, gastrointestinal, neurologic, and skin manifestations. Glucocorticoid therapy, used in the treatment of DRESS, is contraindicated in active Strongyloides infection as it can precipitate a potentially lethal disseminated hyperinfection, particularly in immunocompromised patients. In our immunocompetent patient from an endemic area, the infection was deemed to be indolent and glucocorticoids were safely initiated along with antihelminthic therapy. The patient’s symptoms abated promptly and skin biopsy showing perivascular inflammation composed of lymphocytes and eosinophils confirmed DRESS. DRESS is a rare allergic reaction associated most commonly with the use of carbamazepine and allopurinol. It typically occurs within weeks of drug initiation; however, in our patient it occurred almost 45 years later. Furthermore, in the setting of concurrent Strongyloides infection, a careful risk-benefit assessment had to be undertaken before initiating steroid therapy. Clinicians should be aware of at-risk populations for Strongyloides infection and initiate appropriate treatment to avoid serious complications. Additionally, skin biopsy is a useful tool to definitely distinguish between these two causes of peripheral eosinophilia, fever, and rash.

Author: Neelam Gidwaney

COLLAGENOUS GASTRITIS: A RARE ENTITY IN GI PATHOLOGY

Patients often present with vague gastrointestinal symptoms and it is important for clinicians to consider collagenous gastritis as a potential underlying pathology. Long-term follow up of patients with CG is warranted to better understand the treatment and complications of this rather novel GI entity. Case: A 68-year-old man presented to the outpatient clinic with complaints of epigastric pain exacerbated by the ingestion of food, bloating, nausea without vomiting, and a 10-lb weight loss over two and one-half months. He denied dysphagia, odynophagia, and changes in his bowel movements. There was an absence of diarrhea, constipation, hematochezia and melena. Physical examination showed no jaundice, no scleral icterus, and mild epigastric tenderness. There was no guarding and no tenderness to palpation. Rectal exam showed guaiac negative brown stool. Laboratory studies showed WBC of 10.7 K/mcL with a normal differential, hemoglobin of 13.9 g/dL, AST of 27 U/L, ALT of 45 U/L, Alkaline phosphatase of 94 U/L, total bilirubin of 0.3mg/dL, amylase of 55 U/L and lipase of 4 U/L. Helicobacter pylori serology (IgG) was positive and celiac serology was negative. The patient underwent an esophagoduodenoscopy, which showed edema and thickening of some of the gastric folds, especially in antrum and distal body of the stomach. No erythema and no ulcers were noted. Biopsies showed active gastritis with thickened collagen bands and intraepithelial lymphocytes consistent with collagenous gastritis. H. pylori was not identified. After four weeks of treatment with Rabebrazole and over-the-counter antacids, his symptoms began to improve. Collagenous gastritis (CG) was first reported by Colletti and Trainer in 1989 and there have only been 40 cases in the literature to date. Collagenous gastritis is described as collagen deposition of more than 10 microns within the lamina propria and is most often associated with collagenous colitis and celiac disease. There are two subsets of patients: one inclusive of children and young adults to the age of 25 and a second group that encompasses everyone older than 25. Most children and young adults present with anemia and abdominal pain whereas older patients present with watery diarrhea as their primary symptom. There are no treatment protocols for CG due to the lack of longitudinal studies. Oral steroids, H-2 blockers, PPIs, and gluten-free diet have been utilized in the treatment of CG with variable symptomatic relief. The only documented complication of CG is glandular atrophy and intestinal metaplasia without evidence of dysplasia.
SUPERINFECTED HYDATID CYST: AN UNUSUAL CAUSE OF CYCLIC FEVERS

Fever of unknown origin (FUO), with more than 200 potential causes, represents a diagnostic challenge for clinicians. Despite the advent of powerful diagnostic tools and continued refinement of criteria, FUO remains a diagnosis of exclusion, with upwards of 20% of cases failing to reach a final diagnosis. We present a particularly challenging case of FUO, with an unusual final diagnosis. A 30-year-old female reported to her outpatient physician with fevers to 103 degrees Fahrenheit and fatigue for two days. She was empirically prescribed ciprofloxacin. Over the next day she noted persistent high fevers and fatigue along with nausea with vomiting and shortness of breath. On arrival to the hospital, she was profoundly hypotensive (BP 81/47) with lactic acidosis (4.5), normal white count (5.6), INR (1.25) and transaminitis (AST 103, ALT 116). HIV and Hepatitis panels were negative. Initial CT abdomen noted a large hypoattenuating lesion at the dome of the liver, measuring 6.2 x 4.5 cm. Her urinalysis and chest radiograph were unremarkable. There was no history of toxin exposure, injection drug use or transfusions. Of interest, her medical history included congenital renal and uterine abnormalities but none related to the hepatobiliary system. A native of New Jersey, she reported traveling to Australia and South America within the past two years. She showed initial clinical improvement with aggressive fluid resuscitation and broad-spectrum antibiotics, but fevers persisted as a cyclic pattern (every 12 hours). Blood cultures grew Streptococcus viridans. A transesophageal echocardiogram did not reveal vegetations. Her transaminitis improved and her fevers began to decrease. After reassurance of her initial concern about contrast imaging given her lone kidney, the patient consented to an MRI with gadolinium where the previously described liver hemangioma had characteristics consistent with a complex, multiloculated abscess. Aspirate of the abscess grew Streptococcus viridans despite negative repeat blood cultures. Given significant travel history and close contact with dogs, an echinococcus antibody screen was obtained and was positive. Our final diagnosis was cyclic fevers due to hydatid cyst from prior echinococcus exposure that was acutely superinfected with Streptococcus viridans. Over the next two weeks, the patient’s cyclic fevers remitted on IV ceftriaxone; she was transferred to another institution for definitive surgical excision of the cyst. Human hydatidosis, caused by the dog-borne echinococcus parasite, is rare in the US but endemic to South America, Africa, and Asia. While typically asymptomatic, it may present through compression of pericystic structures or cause a systemic anaphylactoid reaction. Superinfection of the cyst by other microorganisms is an uncommon but well-described phenomenon that may result in bacteremia and cyclic fevers. In the setting of an appropriate travel history, the clinician may consider this unusual nidus of infection when evaluating patients with FUO.

UMDNJ - SOM (JOANNE KAISER-SMITH, DO)

Author: Amanda Valvano

AN 82 YEAR-OLD VIETNAMESE MAN WITH FEVER, ABDOMINAL PAIN, AND ABNORMAL CT RESULTS: AMEobic LIVER ABSCESS

Clinical scenario: An 82 year old Vietnamese male presented with abdominal pain and fever. He had no medical problems or prior surgeries. Originally from Vietnam, he had not visited in over a decade. A CT showed cecal and liver masses and cholecystitis. The patient was admitted to be evaluated by GI and Surgery. II. Evidence: He had a temperature of 100.9, his vitals were stable, and the physical exam was positive for poor dentition and abdominal tenderness. The appearance of the gallbladder was unchanged when compared to a CT from 2007, and surgical intervention was deferred. To evaluate the cecal mass, GI performed a colonoscopy, which showed ulcerations. Biopsies were negative for malignancy. Fine needle aspiration of the liver showed inflammation, consistent with abscess. A drainage catheter was placed, and the necrotic debris was sent for cytology and cultures, both which were negative. Serum antigen detection for antibodies against E. histolytica came back positive and Q.N. was placed on Paromomycin and Metronidazole. On day twenty status post drainage, an abscessogram was negative for fluid or fistula formation and the drainage catheter was removed. On day twenty-one, Q.N. was discharged on oral antibiotics. III. Reason for presentation: Extraintestinal amebiasis can present like metastatic colon cancer. A colonoscopy is warranted and may show either an ulcerated mucosa resembling inflammatory bowel disease or a carcinoma-like lesion in the cecum,

Author: Jonathan Foster

A RARE CASE OF METASTATIC COLORECTAL CARCINOMA

Colorectal cancer is the fourth most commonly diagnosed cancer in the world, and is more commonly diagnosed in developed countries. Colorectal cancer usually metastasizes to the lymph nodes, liver, lung, bone, and brain. Most cases of testicular metastasis are incidental findings on autopsy. Cases can present with testicular complaints after or before the diagnosis of colorectal cancer. 52 year old male complains of testicular pain and swelling. The patient has a history of colorectal cancer treated with FOLFOX neoadjuvant chemotherapy and a subtotal colectomy with low-anterior resection. Ultrasound of the right testicle is done in the emergency room, which showed a solid nodular mass measuring approximately 2.7 x 2.1 x 1.6cm. The process appeared to be extratesticular at that time. The patient underwent a radical orchectomy. Histologic analysis from the right radical orchectomy demonstrated metastatic colorectal adenocarcinoma of the tunica as well as proximal and mid-portions of the spermatic cord. Immunoperoxidase stains were performed and showed tumor cells staining positive for CDX2 and Cytokeratin 20, which is most consistent with metastatic adenocarcinoma of colorectal origin. Literature reviews have shown that colorectal cancer with metastasis to the testes is rare and survival ranges from 6 to 12 months. In a 2010 literature review, there have been 33 documented cases of testicular metastasis from rectal or colonic carcinoma. Testicular metastasis is usually evidence of advanced disease. Various routes of spread of metastatic tumors to the testes have been suggested. These include arterial embolization, retrograde venous spread, retrograde lymphatic spread, direct spread along the vas deferens to the epididymis, and transperitoneal seeding through a patent tunica vaginalis, which is suspected in this case. The most frequent site of colorectal cancer to metastasize to the testis is the sigmoid colon, followed by the cecum and then rectum. This type of metastasis is important for clinicians to consider in the setting of patients with testicular complaints who have a history of colorectal carcinoma. 1.Bryan NP, Jackson A, Raftery AT. (1997) Carcinoma of the sigmoid colon presenting as a scrotal swelling. Postgrad Med J 73:47-48. 2.Hatoum, H, Abi Saad, G, Shamseddine, A. (2011) Metastasis of colorectal carcinoma to the testes: clinical presentation and possible pathways. International Journal Clinical Oncology 16:203-209. 3.Tiltman, AJ (1979) Metastatic tumours in the testis. Histopathology 3:31-37. 4.Almagro UA (1988) Metastatic tumors involving testis. Urology 32:357-360. 5.Jubelirer SJ (1986) Metastatic colonic carcinoma to the testes: case report and review of the literature. Journal of Surgical Oncology 32:22-24

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ACUTE MUSHROOM TOXICITY

A 65 year old Caucasian male presents with a one day history of multiple episodes of bilious emesis and non-bloody diarrhea with symptoms progressing to total body cramps, jaw pain, and expressive aphasia. Past medical history includes Non-Hodgkin’s Lymphoma and Prostate cancer. Social history includes tobacco use greater than 70 pack year and 2 drinks per day for the last 2-3 years. Pertinent physical exam findings include mild scleral icterus and diffuse tenderness to palpation of the abdomen. Initial labs showed total bilirubin of 4.5, direct bilirubin of 2.6, alkaline phosphatase of 128, AST of 4442, ALT of 7870, total protein of 6.4, albumin of 3.7, lactate of 5.5, and coagulation indices showed PT of 39.1, PTT of 69.1, and INR of 5.8. These labs
indicate a combination of synthetic liver dysfunction, hepatocellular and cholestatic damage. After further inquiry of social history, it was found that patient was a mushroom forager and ate five mushrooms that resembled Amanita Phylloides earlier on the day of admission. During the hospitalization, patient became encephalopathic requiring intubation for airway protection. The patient was initially started on Penicillin G and intravenous Silybum marianum but his confusion persisted along with elevations in his liver function tests. Eventually this patient required a liver transplantation with subsequent improvement in laboratory tests and mental status. Amanita poisoning presents with four various clinical stages: 1) the incubation stage occurs between 6-12 hours after ingestion; 2) the gastrointestinal stage can occur up to 24 hours post ingestion characterized by abdominal pain, nausea, vomiting, and diarrhea; 3) the cytotoxic stage is characterized by clinical improvement after 24-48 hours post ingestion followed by a worsening of renal or liver function; 4) the final phase can begin suddenly with coagulopathy, hepatic encephalopathy and lead to fulminant hepatic failure. Milk thistle (Silybum marianum) has been investigated for use as a supportive treatment for liver damage from Amanita phalloides poisoning. Silymarin, the active ingredient, undergoes enterohepatic recirculation leading to higher concentrations in liver cells and inhibits the binding of toxins of the mushroom to hepatocytes. The cytoprotective effects of Silymarin include acting as an antioxidant and free radical scavenger. Additionally, Silymarin can enter the nucleus and act on RNA polymerase leading to increased ribosomal formation and thus increased protein synthesis. This specific action leads to repair of damaged hepatocytes and restoration of normal liver function. This case report presents a rare cause of acute liver toxicity. The differential diagnosis of markedly elevated transaminases above 1000 IU/L includes acute viral hepatitis, shock liver and drug/toxin induced liver injury. It is extremely important to obtain a complete history including going into depth of the social history as it can provide clues to the etiology of liver disease.

UMDNJ NEW JERSEY MEDICAL SCHOOL PROGRAM (NEIL KOTHARI, MD)

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ADJACENT SEGMENT DISEASE FOLLOWING THORACIC PEDICLE SCREW FIXATION

INTRODUCTION: Pedicle screw fixation is a widely utilized technique employed for conditions ranging from spine deformities to fractures. The use of pedicle screws, as opposed to hooks, in spine deformity surgeries has demonstrated lower complication rates, better three-dimensional correction, better pull-out strength, and shorter fusion lengths. Traditionally, pedicle screws were used in the lumbar spine; however, pedicle screws are being used with increasing frequency in the thoracic spine as a more favorable alternative to hooks. While safety concerns, such as the incidence of adjacent segment disease (ASD) following cervical and lumbar fusions (30% incidence of ASD) have been reported, these issues have yet to be thoroughly addressed for the thoracic spine. METHODS: A retrospective review of a prospectively maintained database was performed to determine the incidence of complications for 123 consecutive patients (76 males and 47 females, mean age – 39 years), who underwent thoracic pedicle screw fixations over 13 years. Inclusion criteria were: adults with thoracic pedicle fixation. Children, pregnant or lactating women, and prisoners were excluded from the study. By comparing the preoperative and postoperative radiographic imaging, the occurrence of ASD and disease within the surgical construct was determined. RESULTS: Definitive radiographic fusion was detected in 115 (93.5%) patients. Seven incidences of instrumentation failure and eight lucencies surrounding the screws were observed. In total, 1135 thoracic pedicle screws and 233 rods were placed. Fifteen patients (12%) developed ASD and thirteen (11%) experienced perioperative complications. Mean duration of follow-up was 50 months. CONCLUSION: Radiographic evaluation showed the use of pedicle screws for thoracic fixation to be an effective stabilization modality. Compared to previously published studies, long-term outcomes analysis demonstrated a lower incidence of adjacent segment disease.
RADIOGRAPHICALLY OCCULT PRIMARY RENAL CELL CARCINOMA PRESENTING WITHIN THE OPERATIVE SITE OF A THORACIC SPINE MENINGIOMA

BACKGROUND AND IMPORTANCE: Local tumor recurrence following surgical resection is a well-documented phenomenon. However, metastasis to an operative site from a different primary tumor is quite rare. Even more unusual is the discovery of a metastatic tumor with no detectable primary lesion. CLINICAL PRESENTATION: A 54-year-old man initially presented with decreased sensory and motor function in the lower extremities. He underwent laminectomies of T3 to T5 with a gross total removal of an intradural, extramedullary meningioma. The postoperative course was uneventful and he regained full neurological function. After a 3-year period, the patient developed progressive upper thoracic pain and lower extremity paresthesias. Imaging studies showed an epidural mass at the T2 to T4 levels, extending into the neural foramina bilaterally, as well as what appeared to be blastic involvement of the T2 to T4 vertebrae. A contrast enhanced computed tomography (CT) scan of the chest, abdomen, and pelvis was negative as was a prostate-specific antigen test and urinalysis. Emergent revision laminectomies of T2 to T5 levels were performed with a subtotal resection, yielding a fibrous, nonvascular mass. Neuropathology was consistent with metastatic renal cell carcinoma. After 6 months, the patient re-experienced symptoms of pain and paresthesias. Tissue resection revealed tumor cells morphologically and immunophenotypically similar to those obtained from the prior surgery. Cytogenetic analysis confirmed the presence of metastatic renal cell carcinoma in the epidural region. CONCLUSION: A novel case of an epidural metastatic renal cell carcinoma of unknown primary in the same operative bed of a previously resected intradural, extramedullary meningioma of the thoracic spine is reported. A recommended therapeutic strategy consisting of radiation therapy for local control was initiated.

QUALITY OF PATIENT EDUCATION MATERIALS FROM MEDICAL SPECIALTIES

Objective: The Internet is rapidly becoming a major source of health information. The increasing use of online healthcare-oriented materials as first-line resources necessitate they are written at a reading level the average American can understand. The average American adult has approximately a seventh grade reading level. As such, the American Medical Association and National Institutes of Health recommend that patient education material be written between a 4th and 6th grade level. The authors aim to assess the quality of patient education resources from each medical specialty and provide guidelines for revision in line the national recommendations. Methods: Online patient education materials from each medical specialty were downloaded in January 2012. Resources from the 16 specialties were examined including: Anesthesiology, Dermatology, Diagnostic Radiology, Emergency Medicine, Family Medicine, General Surgery, Internal Medicine, Neurological Surgery, Neurology, Obstetrics and Gynecology, Ophthalmology, Otolaryngology, Orthopedic Surgery, Physical Medicine and Rehabilitation, Plastic Surgery, and Psychiatry. Quality was assessed via readability analyses of each article were conducted using Readability Studio Professional Edition Version 2012.1. Patient education material was assessed using the Coleman-Liau Index, FORCAST formula, SMOG Grading, the New Dale-Chall readability formula, Flesch Reading Ease, Flesch-Kincaid Grade Level, Fry Graphical Analysis, Gunning-Fog Index, the New Fog Count, and the Raygor Readability Estimate. Results: Online patient education material from all specialties was found to be written at a level well above the recommended 6th grade level, as assessed using each of the ten scales. Furthermore, all of the online education material was found to be written well above the 7th to 8th grade level, which is the average reading ability of an American adult. Conclusion: The readability of patient education resources from professional medical websites exceeds the average reading ability of an American adult. This disconnect may impact patient understanding of such resources contributing to poor health outcomes and inefficient healthcare spending. As such, website revisions may be warranted to increase the level of readability and quality of these patient resources to effectively reach a broader patient population. In addition, other modalities of patient resources such as multimedia may be a viable synergist or alternative to printed material.
INVESTIGATING THE CHALLENGES OF IMPLEMENTING INTERVENTIONS DESIGNED TO DECREASE PATIENT NON-ATTENDANCE AT A STUDENT RUN FREE HEALTH CARE CLINIC

Efficient allocation of scarce resources is a critical element in the successful operation of a medical practice. This is especially important when considering the management of a free, community-based primary health care clinic which caters to a population of mostly uninsured patients. One such clinic is the Student Family Health Care Center (SFHCC) at UMDNJ - New Jersey Medical School. High rates of last-minute appointment cancellations and no-shows are significant problems at this and other free clinics. Missed appointments not only compromise the effectiveness of primary care preventative measures and the management of chronic health conditions, but also dampen the extent of benefits the clinic can provide to the patients and community served. Missed appointments in the primary care setting disrupt the patient-clinician relationship and reduce the opportunity for other patients to receive timely and appropriate care. Due to the limited number of exam rooms available per clinic session, no-shows represent a gross under-utilization of resources. Three interventions were designed to decrease patient non-attendance, however there were significant barriers encountered on implementing these strategies. Providing child care, giving patients an additional reminder call one week in advance, and allowing online schedule access each had their own set of difficulties. Originally this study was designed to compare the patient attendance rates before and after implementation of the three interventions. Both pre and post intervention online chart review were each collected for 10 clinic sessions between May 31 to August 16th, 2012. Outcome of visits (attended, no show, or rescheduled) were collected along with other demographic variables. Childcare, patient reminder calls one week in advance, and patient email reminders one week in advance/online schedule access were conducted and assessed. The effectiveness of the interventions was measured in patient attendance rates. We also examined the influence of the following covariates: sex, primary language, age, visit type, and insurance status. Statistical analysis of the proportions of attendance rates were calculated. Although differences were observed, the study highlighted major barriers in the implementation of these interventions. There were several unforeseen challenges associated with addressing the major barriers patients face in attending their appointments which need to be considered before developing an effective approach to decrease patient non-attendance. The barriers encountered were low patient participation, lack of resources to implement the designed interventions, liability of providing childcare, limitations in the EMR, and inherent resistance to change. Future studies should be designed to provide quality health care to community residents by maximizing the utilization of available resources. These studies must include strategies to address the aforementioned barriers in order to create a sustainable program which improves access to care, especially in underserved populations.

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OVERLAPPING SYNDROMES: NOT ALL TREATMENTS ARE CREATED EQUAL

Rheumatologic diseases can present with a variety of puzzling symptoms. Despite initial diagnosis of a distinguished autoimmune disease, a high degree of suspicion should be maintained for overlapping syndromes in patients presenting with rapidly progressive multiorgan disease. 60-year-old caucasian woman with a history of hypertension, COPD, and rheumatoid arthritis with medication non-compliance, presented to her primary care physician with an erythematous rash and petechiae on her legs for one week. Initial work up by her primary doctor revealed acute kidney injury with a creatinine of 3.3 and positive ANA titer. Three days later, upon initial presentation to the ER, she had a creatinine of 7.9 along with frank hematuria and decreased urine output. Further investigation revealed a 20 pound weight loss and fatigue over the preceding six months. Physical examination revealed a malnourished woman with bitemporal wasting, diffuse muscle atrophy, ulnar deviation of bilateral MCP’s with paronychial inflammation, along with a non-blanching, lace-like erythematous rash on bilateral lower legs. Admission labs showed positive anti-ds DNA (436.6, ELISA), positive ANA (1:320), low complements (C3 37, C4 8.2), suggesting SLE, which was confirmed with Class III lupus nephritis on renal biopsy. Treatment was initiated with high-dose methylprednisolone, mycophenolate mofetil induction along with hemodialysis. Despite this treatment, the patient worsened by developing frank hemoptysis resulting in hypoxemic respiratory failure and subsequent intubation. Falling hematocrit, new alveolar infiltrates on chest
radiograph in addition to hemosiderin laden macrophages on bronchoalveolar lavage confirmed suspicion for diffuse alveolar hemorrhage. Surprisingly, further serologic testing showed a strongly positive P-ANCA (1:640) and Anti-MPO (1:60) titers suggesting a possible overlapping microscopic polyangiitis vasculitis along with SLE. At this point, cyclophosphamide and plasmapheresis were initiated. Unfortunately, the patient’s hospital course continued to worsen including development of sepsis and stroke, before she passed away. Diffuse alveolar hemorrhage is a catastrophic, infrequent pleuropulmonary manifestation of SLE. A true overlapping syndrome with SLE and ANCA-associated vasculitis is a rare phenomenon with atypical presentation, which in our case included a rapidly progressive glomerulonephritis and diffuse alveolar hemorrhage (1). Even though the literature suggests the equivalent role of mycophenolate mofetil and cyclophosphamide in SLE nephritis, there are no recommendations for the treatment of overlapping syndromes (2). Given the rapidly progressive nature of this disease, the initial use of cyclophosphamide instead of mycophenolate mofetil may have lead to a better outcome. Patients with overlapping syndromes are a distinct population requiring augmented treatment and further study. 


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ARE CLINICIANS FOLLOWING THE ACG OR ACR GUIDELINES WHEN SUSPECTING ACUTE PANCREATITIS

Purpose: Acute pancreatitis is an acute or recurrent inflammatory process of pancreas. Clinical symptoms of pancreatitis include sudden onset of epigastric pain, pain radiating to the back, nausea/vomiting and fever. Clinical symptoms, elevation of pancreatic enzymes and imaging of pancreas are major evidence to diagnose pancreatitis. However, imaging is not always required to make the diagnosis of acute pancreatitis. Imaging with CT Scan or Ultrasound can help grade the severity of the pancreatitis and sometimes provide a cause. This study is to determine whether healthcare providers are using guidelines suggested by American College of Radiology (ACR) or American College of Gastroenterology (ACG) to make the most appropriate imaging decisions for diagnosis and evaluation of acute pancreatitis. Method: This is a retrospective cohort study of a group of 409 patients that were diagnosed with acute pancreatitis hospitalized in Hackensack University Medical Center, NJ, from January 2010 to January 2011. Fifty patients were randomly selected from this group and their most recent admission for pancreatitis was studied. Each workup was checked with both ACG and ACR guidelines for noncompliance. Results: 58% of our cohort is noncompliant with ACG guidelines. Of those who were noncompliant, 57% had a CT Scan or abdominal ultrasound, which are unnecessary imaging or inappropriate workup without an elevated lipase according to ACG guidelines. However, with the same cohort, only 10% is noncompliant with ACR guidelines. Conclusion: There is a higher overall noncompliance with ACG guidelines than ACR guidelines. It’s possible because ACG has a strict algorithm on whether or when to order an imaging test. According to ACG guidelines, while abdominal pain is consistent with acute pancreatitis and there is an elevated amylase or lipase > 3 times of upper limit of normal, imaging is unnecessary to diagnose acute pancreatitis. Discussion: ACR describes in its guidelines that 2 of 3: abdominal pain, lipase x3 upper limit of normal, or CT Imaging can establish the diagnosis of pancreatitis, and that CT imagery can establish severity and predict clinical outcomes, thereby justifying its more liberal use depending on clinical presentation. That may explain the noncompliance of 10% with ACR guidelines. However, with proper establishment of diagnosis, there is no difference in disease management or outcome. The unnecessary ordering of CT Scans, as indicated by the ACG guidelines exposes the patient to unnecessary radiation, and increases the overall cost of health-care. This study will help bring awareness in clinicians regarding ordering imaging studies appropriately, and help define a safe and cost-effective approach for the delivery of health care.
Gastrointestinal Leiomyomas are extremely rare benign mesenchymal tumors of the GI smooth muscle, originating from the muscularis mucosa or submucosa. These tumors are difficult to differentiate from gastrointestinal stromal tumors. Previously published studies of leiomyomas are actually GIST tumors, now diagnosed by immunostains, such as c-Kit (CD117) and CD34. A 65 year old Caucasian woman with history of Diabetes Mellitus Type II, HTN, and a recent diagnosis of anemia of undetermined etiology presented with a chief complaint of persistent nausea and vomiting for six days. Her abdomen was distended, diffusely tender, and a firm non-nodular mass with smooth borders was palpable in the left upper quadrant extending down into the left and right lower quadrants. CT scan of Abdomen/Pelvis showed a large solid heterogeneous mass. Endoscopic ultrasound showed a submucosal mass arising from the duodenum. A core needle biopsy of the mass showed a spindle cell tumor strongly positive for smooth muscle markers actin, myosin, desmin, and negative for c-Kit, CD 34 and S-100. Final diagnosis was intestinal leiomyoma of uncertain malignant potential. The tumor was deemed nonresectable due to its invasion of the superior mesenteric vessels. After improvement of small bowel obstruction and anemia patient was discharged to sub-acute rehabilitation facility, and outpatient follow-up with medical and surgical oncology was provided for further management. This case illustrates the importance of c-Kit immunostaining in differentiating leiomyoma/leiomyosarcoma from gastrointestinal stromal tumors. GIST tumors arising from the small intestine can be managed with Gleevec followed by surgical resection, while surgery is the only definite therapy for gastrointestinal leiomyomas. The prognosis of gastrointestinal leiomyomas is directly related to increased size, metastasis, histologic atypia, anatomic irregularity and mitotic rate.

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ARE WE FOLLOWING THE NJ GUIDELINES FOR ADVANCE DIRECTIVES IN THE INTERNAL MEDICINE OUTPATIENT OFFICE?

An advance directive is a document that allows patients to state their wishes for medical treatment in the event that they are unable to do so for themselves. Patients and physicians are in agreement that the patient’s autonomy should be preserved, when patients cannot speak for themselves; however, few patients have completed an advanced directive, and fewer have shared it with their physician. Medical ethicists and expert physicians advocate that the ideal scenario for advance directive discussions is a physician-initiated discussion in the outpatient setting when the patient is healthy, rather than from a non-physician during an acute medical illness. According to the State of New Jersey Department of Health and Senior Services (Article IV), the rights and responsibilities of the healthcare professional is to make an affirmative inquiry of the patient, his family or other about the existence of an advance directive; it is the responsibility of the attending physician to make a note of it in the patient’s records. In addition, the NJ guidelines suggest that it is the responsibility of the institution to adopt these policies at the time of admission and when appropriate. To evaluate whether the NJ guidelines for advance directives are being followed at our outpatient client clinic, we performed a quality assurance research assessment. Using the electronic medical record (EPIC), patients’ demographics including age, gender, primary disease, religion and primary language were recorded. Lastly, whether or not a patient has an advanced directive was noted. After reviewing 102 patient charts from the UMD care medicine clinic, it was found that 99 patients answered "NO", 3 patients answered "YES" and 1 patient was not asked whether or not they had an advance directive. The most striking part of our study was that the overwhelming majority of patients did not have any form of an advanced directive, regardless of race, gender, education level or economic class. More than 95% of the patients did not have an advance directive at the time they were asked; however, compliance with the guidelines was 98%. Patients with an advance directive tended to be older, suggesting that these patients may be more aware of the need for this. Surprisingly, primary disease was not linked to whether patients had an advance directive, although the reasons for this are unclear. Literature review suggests that while most patients desire advance directive discussions, they believe it should
be initiated by the physician in the outpatient setting while the patient is healthy. Ideally, discussing the advance directive should be an on-going part of the health maintenance and education.

Author: Vinnidhy Dave

**RENALE VEIN THROMBOSIS WITH AN IVC FILTER IN A HORSESHOE KIDNEY**

Renal vein thrombosis (RVT) is a rare finding outside of patients with nephrotic syndrome or malignancy. This group consists of about 10%. IVC filters have commonly been shown to cause thrombus formation. It is seen in Here we discuss a patient with horseshoe kidney who presented with extension of thrombus from an IVC filter into the iliac and renal vein. This is a 46 year old man with a medical history of sarcoidosis, deep vein thrombosis s/p IVC filter 2 years prior, alcohol and tobacco abuse who presented with one day history of abdominal pain that was characterized as sharp, constant, 10/10 in pain scale, non-radiating with no exacerbating or alleviating factors. Abdominal exam was remarkable for suprapubic tenderness to light palpation, a mass felt on deep palpation in the right lower quadrant, no rebound tenderness, and no CVA tenderness. Lab findings demonstrated Leukocytosis of 35K with 7% bands and 75% neutrophils, creatinine of 1.9 from 1.0 three months prior, and urinalysis of specific gravity 1.019, pH8, protein 300, glucose 70, RBC >182, and WBC 3. A CT scan of the abdomen and pelvis was preformed without contrast which showed extensive peritoneal inflammatory stranding involving abdomen and pelvis around a horseshoe kidney. The patient was empirically started on piperacillin/tazobactam and given aggressive IV hydration as his BP was 70/43. Eight hours later patient had minimal urine output and creatinine had worsened to 2.7. As his urinalysis did not appear infectious in etiology, renal vein duplex was preformed but was inconclusive for abnormal anatomy. A visceral duplex showed right external iliac femoral vein thrombus and suspicion for RVT in right renal vein. An emergent venogram by interventional radiology (IR) showed thrombus within IVC filter extending back into the iliac vein, and tPA infusion was started at 1mg/hr. With worsening kidney function he required dialysis. On day six of admission IR placed a catheter into the renal vein showing complete occlusion, tPA was started. The following day thrombectomy with removal of all the clot extending from renal vein to the IVC filter was accomplished. Patient was then bridged to warfarin prior to discharge and had recovery of kidney function. Patients with a horseshoe kidney will not present in a similar manner to typical patients. Our patient had suprapubic tenderness with mass felt in RLQ and hematuria. He presented with acute renal failure which normally is only seen in bilateral RVT. Thrombolytic therapy with or without catheter thrombectomy is recommended for the treatement of RVT followed with six to twelve months of anticoagulation.

Author: Gaurav Gandhi

**SALMONELLA ENDOCARDITIS FROM PACEMAKER LEADS**

Endocarditis related to pacemaker lead infection is rare but lethal complication of permanent pacing. A mortality rate as high as 66% has been reported when infection of a pacemaker system is left completely untreated. Patient is a 61 year old Caucasian man with a history of aortic stenosis status post prosthetic valve replacement and pacemaker placement secondary to complete AV block presented complaining of cyclical fevers, chills, and night sweats every 12 hours. Patient was admitted to general medical floor for suspected sepsis, source unknown. He was started on intravenous hydration and broad spectrum antibiotics. Blood culture were growing gram negative rods identified as Salmonella. Peripheral smear was normal with no toxic granules, schistocytes, and no platelet clumping. Cardiology was consulted for possible endocarditis and TTE/TEE showed no evidence of vegetation on valves but a thrombus coated RA/RV lead wires were noted. CT chest/abdomen/pelvis showed no focus of infection or evidence of aortic aneurysm. Colonoscopy and WBC scan also did not show a focus of infection. Dental was consulted for patient’s poor dentition and all nonviable teeth were extracted. Patient was initially tried on antibiotic therapy to clear his bacteremia but failed medical therapy after two weeks on antibiotics. Since blood cultures continued to show GNR Salmonella, arrangements were made for removal of pacemaker and lead wires. Post extraction, 4/4 cultures were positive for Salmonella Dublin from lead wires. The incidence of cardiac device infections is low, with population-based studies showing rates below 1 percent. Staphyloccoccus species account for up to 89% of device-related endocarditis with Salmonella being a very rare cause, less than 2%. Nontyphoidal salmonella is primarily an enteric organism but it can progress to development of infection at any site. In particular, the organisms are presumed to home to existing endovascular atherosclerotic sites in older adults. In the absence of evidence of
device-related endocarditis, such patients may be treated with antibiotics for bacteremia without explantation of device and then observed for relapse. Subsequent unexplained relapse suggests device infection and a need to extract the system.

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DIABETES MANIFESTO: EVALUATING ADA GUIDELINES FOR THE MANAGEMENT OF TYPE 2 DIABETES IN AN URBAN MEDICINE RESIDENT CLINIC

Introduction  The American Diabetes Association(ADA) releases guidelines for the diagnosis, prevention, and management of diabetes. These guidelines include risk factor monitoring, diabetes control, and management of end-organ complications. In December 2009, a revolutionary book, The Checklist Manifesto by Dr. Atul Gawande, demonstrated how a well-designed step-by-step process can improve outcomes of professionals with complex responsibilities. Our project sought to measure the degree to which our urban resident clinic follows ADA guidelines in managing diabetics, with the intent to create a template checklist to improve adherence. Methods This project was reviewed by the UMDNJ IRB prior to initiation. A retrospective chart review of 97 patient visits in an urban residents’ internal medicine clinic between January and December 2011 was conducted. Patients with established type 2 diabetes were selected using a random numbers generator. Resident progress notes were evaluated for documentation of pre-selected ADA outpatient guidelines. The measured outcome was the proportion of times each guideline was documented in the notes. Results Blood pressure and finger-stick glucose measurements had 100%(95%CI 96-100) adherence. HbA1c measured within six months:85%(95%CI 76-90), home glucose log reviewed:68%(95% CI 46-85), lipid measurement within one year: 56%(95%CI 46-65), ophthalmology referral within one year:53%(95%CI 43-62), and foot exam or podiatry referral within one year:53%(95%CI 43-62). Our areas with the least compliance (<50%) included nutrition counseling or referral:41%(95%CI 33-52), exercise counseling:35%(95%CI 26-45) and urine microalbumin:34%(95%CI 25-45). Discussion In our analysis, adherence to the above guidelines was suboptimal in most categories. Possible explanations include lack of knowledge of the guidelines, competing demands of care, and incomplete documentation. Our results are comparable to analyses conducted at other resident clinics. While it has already been established that a multifaceted professional intervention is most effective in improving diabetes management, we wanted to investigate an area of quality improvement for residents. The use of electronic medical records (EMR) offers the opportunity to reinforce adherence to guidelines, as computer generated prompts can assist residents in providing comprehensive care. Previously attempted interventions at other institutions include take-home diabetes records, birthday reminders and registry-generated audits linked to the EMR. Within the last few years, checklists have emerged in many medical fields as an effective quality improvement measure. Checklists have been successfully implemented in the operating room, intensive care units as well in the outpatient setting. Our goal, using the Checklist Manifesto as a framework, is to create a simple, easy-to-use template for our residents. Each month we will reassess our ADA guideline compliance by selecting random patient visits and reviewing the documented charts. We hope that post-interventional analyses will demonstrate improved internal medicine resident compliance in outpatient diabetes management and will lead to the establishment of checklists for other chronic conditions.

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QUALITY OF WARFARIN BASED ANTICOAGULATION AT THE EAST ORANGE VETERANS AFFAIRS MEDICAL CENTER

Quality of warfarin based anticoagulation at the East Orange Veterans Affairs Medical Center. Lata Cherath MD, PhD; Arpita Gandhi MD; Lauren Parker MD; Luvkarnjit Singh MD; Susana Tapia MD; Aleksey Tentler MD. Department of Medicine, Veterans Affairs at East Orange New Jersey Medical School, UMDNJ Newark Introduction: Atrial fibrillation (AF) is the most common arrhythmia and it accounts for 1/3 of hospital admissions for arrhythmias. AF is associated with a 7-fold increase in risk of ischemic stroke compared to the general population. In our investigation, we used the time in therapeutic range (TTR) to assess the quality of warfarin management at the East Orange, NJ VA Medical Center (EOVA). Methods: 100 patients with
nonvalvular Afib were randomly selected from a database at EOVA. 72 of 100 patients were chosen excluding those with CHADS2 scores of 0 or 1 and were not on warfarin, or, were determined by their physician to have a contraindication to anticoagulation; or, had dual care and warfarin management was not at EOVA. We recorded the last 5 INR values of each of these patients and calculated the percent time in therapeutic range (TTR). We defined therapeutic range as an INR of 1.8-3.5.1. The mean TTR was then calculated. If a patient had 3 out of 5 INR values (60%) in the therapeutic range, they were considered to be at goal. Results: Our analysis showed that 65 of the 72 (90%) patients were at goal (95% CI = 0.81-0.95). Of these 65 patients – 17 patients were in TTR 60% of the time, 25 patients were in TTR 80% of the time and 23 patients were in TTR 100% of the time. The mean TTR for the entire sample was 76%. All the INR values collected over 5 visits for 72 patients were analyzed; they were found to have a mean INR of 2.47, median INR of 2.36, and standard deviation ± 0.752. Conclusion: Our data shows that after exclusion of those patients deemed “unsuitable” for warfarin therapy, 100% of our patient population at the EOVA received appropriate anticoagulation therapy based on their CHADS2 score. The meta-analysis presented by Baker et al1, showed the mean TTR in anticoagulation clinics was 66% vs. 57% for community-based care provided by physicians. Our data at the EOVA is superior to that presented by the meta-analysis above. This data can be used to profile the quality of outpatient oral anticoagulation in a large, integrated health system and the measures adopted at EOVA can serve as the basis for quality measurement and quality improvement efforts. References: 1. Baker et al; Meta-analysis to assess the quality of warfarin control in atrial fibrillation patients in the United States. J Manag Care Pharm. 2009 Apr;15(3):244-52.

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RELATIONSHIP OF NEUTROPHIL-TO-LYMPHOCYTE RATIO AND SERUM ALBUMIN LEVELS WITH C-REACTIVE PROTEIN IN HEMODIALYSIS PATIENTS: RESULTS FROM AN INTERNATIONAL STUDY

Background: Neutrophil-to-lymphocyte ratio (NLR), defined as the neutrophil count divided by lymphocyte count, is an inexpensive and readily available parameter which may serve as a surrogate for inflammation markers, such as C-reactive protein (CRP). The aim of this study was to determine the utility of NLR to predict elevated CRP levels in hemodialysis (HD) patients. We postulated that: (1) higher NLR and low albumin levels are independently associated with higher CRP levels, and (2) combination, of high NLR and low albumin levels further improves the prediction of high CRP levels in HD patients. Methods: We studied a sample of 27,857 HD patients from Fresenius Medical Care (FMC) clinics in 17 European countries. In all patients routine contemporaneous measurements of neutrophil and lymphocyte counts, serum albumin and CRP levels were available. In the majority of patients albumin was measured using the bromocresol green (BCG) method; in those where the bromocresol purple (BCP) method was used, the results were converted to BCG. In our analysis NLR and albumin levels were the independent and CRP levels the dependent variable. Logistic regression was used to determine the relationship of trichotomized NLR (<2.5, 2.5-5, >5.0) and albumin levels (<3.1, 3.1-4.0 and >4.0 g/dL) with elevated CRP levels (>5.0 mg/L) as the outcome variable. This categorization was based on clinical cut-offs reported in the literature. Results: Mean (±SD) age was 63.5 ± 15.2 years, 58.3% of the patients were males. Dialysis vintage was 53.0 ± 59.4 months (median 33.2 months, interquartile range 11.3-73.7 months). The mean albumin, absolute neutrophil count, absolute lymphocyte count and median (interquartile range) CRP were 3.9±0.5 g/dL, 4.3±2.1 x 10^9 cells/L, 1.63±.87 x 10^9 cells/L, and 6.3 (0.10-160) mg/L, respectively. We found a statistically significant positive correlation between NLR and CRP (r=0.23, P<0.0001). After adjusting for age, gender, dialysis vintage, we found that NLR > 5.0 versus <2.5 (adjusted OR: 1.84, 95% CI: 1.63-2.08; P<0.0001) was associated with CRP levels > 5.0 mg/L. Similarly, albumin <3.1 g/dL versus >4.0 g/dL (adjusted OR: 3.39, 95% CI: 2.99-3.85; P<0.0001) was associated with high CRP levels. There was no strong association between absolute neutrophil and lymphocyte counts and serum CRP levels as compared to NLR. Furthermore, stepwise increase in the odds ratio for CRP > 5.0 mg/L was seen with the combination of high NLR and low albumin levels. Conclusions: Our results may add to diagnostic abilities in settings where CRP is not measured routinely in HD patients. NLR is easy to integrate into daily practice and may be used as a marker of systemic inflammation. Further studies should evaluate the prognostic significance of serial NLR measurements in HD patients.
ABSOLUTE VS. RELATIVE CHANGE IN SERUM CREATININE: INFLUENCE OF BASELINE RENAL FUNCTION ON DIAGNOSIS AND STAGING OF AKI

Background: Several diagnostic criteria including the AKIN, RIFLE and Waikar classification systems have been proposed for diagnosing and staging acute kidney injury (AKI) based on absolute and relative changes in serum creatinine (sCr). The aim of the study was to evaluate the performance of different classification systems to diagnose and stage AKI in critically ill patients. Additionally, we evaluated whether the time to diagnose AKI based on absolute and relative changes in sCr is influenced by the level of underlying renal function. Methods: We assessed the incidence, time to diagnosis and severity of AKI in 717 patients admitted to surgical intensive care unit (SICU) and medical intensive care unit (MICU) at UCSD between June 2006 and Dec 2008. We compared the AKI incidence and staging based on: AKIN (Crit Care; 2007), RIFLE (Crit Care; 2004) and Waikar (JASN; 2009) classification systems. We used the ICU admission sCr as the reference. Results: The incidence of AKI ranged from 22.5% by Waikar, 28.3% by AKIN criteria, to 34.7% by RIFLE criteria. The median time to reach AKI diagnosis in the above three criteria was 19.0 hrs (12.0-35.0), 24.3 hrs (13.4-39.8) and 32.1 hrs (17.1-61.5), respectively; P=0.20. Forty-two and 23 patients diagnosed by AKIN classification were misclassified as not having AKI by Waikar and RIFLE criteria, respectively. The Waikar misclassification was due to the shortened time interval available whereas the RIFLE misclassified patients with small increases in sCr. The overall mortality rate was 15.3% in AKI patients versus 4.7% in non AKI. Patients misclassified by Waikar and RIFLE had mortality rate of 11.9% and 13%, respectively. Conclusion: Diagnostic and staging criteria for AKI vary in sensitivity for identifying AKI. The RIFLE classification identify more patients with AKI as it includes GFR as a criterion and computes the changes over the 7 day period.; however it is less sensitive to detect small changes in kidney function. The Waikar classification misclassifies patients with slow development of AKI. Absolute changes in sCr are not influenced by baseline kidney function and occur earlier than relative change in sCr. Further studies are required to define the influence of the underlying renal function on time to diagnosis and progression in severity of AKI.

Author: Arpita Gandhi

A 54 YEARS OLD MAN WITH RAPIDLY ENLARGING MASSES AT HEAD AND NECK

Introduction: Multiple Myeloma (MM) is a plasma cell disorder that accounts for approximately 1% of neoplastic diseases and 13% of hematologic cancers. The median age of diagnosis is approximately 70 years with 37% of patients < 65 years, 26% are between 65 – 74 years and 37% of patients are > 75 years of age. Plasmacytomas are clonal proliferations of plasma cells that have a localized osseous or extraosseous growth pattern, with potential to progress to multiple myeloma. Case Presentation: This is a 54 year-old Hispanic man with a medical history significant for hypertension and prior stroke who presented with head and neck masses. He was in his usual state of health when he noticed these growths that increased in size over 3 weeks. These masses were painless and without superficial wounds or discharge. Other associated symptoms included decreased appetite for 1 week and 10 lb weight loss over 3 weeks. Physical exam was significant for a 5x6 cm mass on the Right Temporal area and a 6x7 cm mass arising from supraclavicular area on the left anterolateral neck area with both masses being firm, fixed, nodular, non-tender and non-erythematous in nature. Initial labs were significant for microcytic anemia, thrombocytopenia, renal failure with Cr of 22.6, and elevated calcium of 10.3. Imaging revealed 2 distinct masses, a soft tissue mass (6 x 6.8cm) at the right temporal bone and zygoma with involvement of the zygomatic arch and superolateral aspect of the orbit and another large, destructive soft tissue mass (7.3 x 7.3 cm) arising from proximal left clavicle. Deformities in multiple ribs and multiple lytic lesions within vertebral bodies were also noted. Skeletal survey showed lytic lesions diffusely, including the skull. Serum Protein Electrophoresis was consistent with a monoclonal gammapathy, Immunofixation that showed IgG paraprotein with kappa type and urine was positive for Bence Jones protein. Fine Needle biopsy of these masses showed plasmacytoma and Bone Marrow biopsy showed plasmacytosis of 32% by flow cytometry. The final diagnosis was made as Stage IIIB Multiple Myeloma, Ig G type with kappa light chain restriction in the presence of two plasma cytomas. Patient was immediately started on chemotherapy with VCD (Bortezomib, Cyclophosphamide and Dexamethasone) and within weeks of treatment initiation, patient died after being admitted to intensive care unit for respiratory failure secondary to septic shock. Discussion: The presence of Plasmacytoma is one of the major criteria for the diagnosis of
Multiple Myeloma. Here we have a case with rapidly enlarging extramedullary plasmacytomas along with anemia, hypercalcemia, renal failure and lytic lesions for the diagnosis of Multiple Myeloma.

Author: Arpita Gandhi

**NEED OF IMPROVED SURVIVORSHIP CARE FOR BREAST CANCER SURVIVORS:**

A case of recurrent metastatic breast cancer  Arpita Gandhi MD Department of Medicine, New Jersey Medical School, UMDNJ Newark  Introduction: Breast cancer is the second most common cause of cancer death, after lung cancer, among women in the U.S. Several trials have demonstrated the utility of adjuvant hormonal therapy that results in long term survival of breast cancer patients with hormone receptor-positive disease. The number of breast cancer survivors is increasing and the survivorship care in clinical practice becomes critical for prevention and recurrence of the disease.  Case Presentation: This is a 56 year old post menopausal Hispanic woman with past medical history of breast cancer – DCIS, high grade (2008), s/p chemotherapy and right mastectomy with sentinel node dissection who presented to East Orange VA with recurrent cough, shortness of breath and temporal headaches refractory to medical management. Patient denied associated fevers, chills, nausea, vomiting, hemoptysis, weakness or visual disturbances. In brief, patient came to Eova about 2 months ago to establish primary care and was started on oral anti-hypertensives. Prior to this visit, patient was lost in follow-up by medical and surgical oncology in the community and reported she abruptly discontinued letrozole treatment &#8805; 1 year. Patient denied side effects from letrozole use and reported “She felt well and thought she could stop the medication”. Prior to this admission, patient received a mammogram with normal findings. Physical exam was normal except for well healed right mastectomy site and point tenderness at T2-T5 vertebrae. Initial labs showed hypercalcemia, hyperphosphatemia and elevated Alkaline Phosphatase. Further workup showed, PET Scan with a large calvarial lesion within the left frontal lobe, Chest CT with ill defined small nodular, opacities throughout both lungs and MRI of spine with T4 epidural lesion. Transbronchial biopsy showed metastatic cancer with breast as the primary origin and tissue was ER+/PR+, HER-2/neu negative. Patient received radiation treatment to skull and spine and is currently placed on combination regimen of everolimus and exemestane as this is a progressing disease refractory to letrozole.  Discussion: It is critical to the transition from active treatment to survivorship care with the continued use of adjuvant hormonal therapy because it significantly improves the long-term survival outcomes of breast cancer patients with hormone receptor-positive disease. Lack of adherence to adjuvant hormonal therapy among breast cancer survivors is likely multifactorial as discussed in a meta-analysis by Murphy et al 2012. This requires improved guidelines for practitioners and patients to increase drug adherence and prevent disease recurrence as seen in this case.

OTHER

Author: Magalie Bruneus

**QUALITY IMPROVEMENT AND INFECTION CONTROL ANALYSIS: UNDERSTANDING THE BARRIERS TO REDUCTION OF TRANSMISSION OF CLOSTRIDIUM DIFFICILE.**

Prevention of hospital acquired infections through use of evidence based practices is an important patient safety goal promoted by the Joint Commission. Increases in incidence, severity, and risk of recurrence for Clostridium difficile infection (CDI) have been observed, and may be due to emergence of an epidemic strain known as BI/NAP1/027 or NAP1. Fidaxomicin, a newly approved antibiotic, has been shown to have non-inferior clinical cure rates compared with vancomycin, with lower recurrence rates and higher global cure rates in non-NAP1 strains. Despite active infection control practices, rates of infections at the hospital were noted to increase during a recent three month period, causing concern for increased risk of nosocomial transmission. The goals of this project were to: 1) review current infection control practices using quality improvement assessment tools and 2) assess the current epidemiological data on recent cases of CDI and identify any association with the NAP1 strain. Assessment of the environment of care and infection control practices related to prevention of CDI transmission was performed using information obtained from surveys of hospital personnel and process maps were created from observations. Using data from the past 6 months, cases of CDI were analyzed with NAP1 strain typing determined by PCR. Patients were counted as a new case or recurrent...
case one time each month. Recurrent cases were defined as patients who had CD diarrhea that occurred more than a month after treatment completion from a prior episode. From the RCA and process mapping, multiple operational barriers were identified, demonstrating the complexity of effective control of CDI. 96 cases of CDI were identified. Monthly CDI cases varied from 8 to 22, with the highest number of cases (21 and 22, respectively), identified as an outbreak period. Each month, 33% of cases were recurrent and this fraction did not vary significantly during the outbreak period. Overall 51% of cases involved the NAP1 strain, and NAP1 positivity did not significantly change during the outbreak period. The NAP1 strain was less prevalent in recurrent cases (44%) than in new cases (56%). However, this difference was not statistically significant. The process of recognizing CDI and instituting appropriate infection control measures is complex and requires a multifaceted approach involving all hospital personnel. During the two month outbreak, there was no difference in the total number of new versus recurrent cases and there was no change in the prevalence of the NAP1 strain. However the period of observation was limited. It is still unclear how different treatment options affect rates of treatment response and relapse and how these may be affected by the NAP1 strain. However, epidemiological assessment of CDI with molecular analysis may help to improve characterization of cases to design a better treatment approach.