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ASSOCIATES SPRING ABSTRACT

COMPETITION

“THE REST OF THE BEST”

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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.

POSTER PRESENTATIONS

FDG-PET INCIDENTALOMAS- HOW DO WE INTERPRET THEM?

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Objective: 18Fluorodeoxyglucose positron emission tomography (FDG PET) is an essential tool of a modern oncology practice and frequently demonstrates increased activity in the thyroid gland. We present two cases of FDG-PET 'incidentalomas' and discuss their implication.

CASE 1: A 40-year-old woman with history of breast carcinoma diagnosed 6yrs ago, treated with a lumpectomy, lymph node dissection and bilateral oophorectomy was found to have an intense uptake in the neck/thyroid area on a routine surveillance FDG -PET. The uptake was bilateral and diffuse. The patient was asymptomatic except for fatigue. No goiter, nodules or cervical lymphadenopathy was apparent on examination. TSH was 14.2 μ IU/ml (0.35-5.5); thyroid peroxidase antibody was strongly positive at 192.5 IU/ml (0.0-3.9). She was diagnosed with Hashimoto's thyroiditis and primary hypothyroidism and started on levothyroxine replacement. Follow up thyroid sonograms and neck CT scans remain negative for any nodules or lymphadenopathy; she continues to do well. CASE 2: A 64-year-old woman who underwent lumpectomy for breast cancer 1 year ago was found to have focal increased uptake on the right side of the neck on a surveillance FDG-PET. The patient reported palpitations and weight loss of 15 lbs in the last 12 months. Physical examination revealed a goiter with multiple nodules, right greater than left. There was no bruit and no lymph nodes were palpable. A thyroid sonogram confirmed a multi nodular goiter, the largest 3cm nodule being in the right lobe. TSH was 0.59 μ IU/ml (0.35-5.5). Fine needle aspiration of the nodules was consistent with colloid goiter and benign nodule. The patient elected not to have surgical excision. Serial sonograms at 6 months intervals show neither growth nor the development of malignant features and the TSH remains at the lower limit of normal. Conclusion: Thyroidal uptake of FDG on PET is worrisome given up to a 50% probability of malignancy, either primary or secondary. The other 50% are 'incidentalomas' and common etiologies include thyroiditis, Graves' disease, toxic nodule, recent FNA or surgical intervention. Appropriate clinical, radiological and pathological follow up after a positive FDG-PET may avoid unnecessary extensive surgical procedures from being undertaken for benign thyroid lesions.

METHIMAZOLE-INDUCED HEPATOTOXICITY: IS AMIODARONE THE MISSING LINK?

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INTRODUCTION: Methimazole infrequently causes liver dysfunction with toxicity seen in 0.1% up to 6% of patients. In most cases, mild elevations in liver enzymes and bilirubin occur within 2-4 weeks of initiation of methimazole therapy and are associated with cholestasis with minimal cellular damage on liver biopsy. Many patients receiving methimazole are young and are healthy except for thyroid disease. We recently encountered three patients in whom methimazole toxicity occurred during treatment for amiodarone-induced hyperthyroidism raising the question of whether this agent maybe more toxic in older patients with co-morbidities. CASE 1: A 62-year-old man presented with a 20-30 pounds weight loss over several weeks, dysphagia, weakness and malaise. A diagnosis of amiodarone-induced hyperthyroidism was made. Liver function tests (LFTs) were normal. He was treated with prednisone, atenolol and methimazole 30 mg daily. At follow-up he had no new symptoms but abnormal LFTs were noted: ALT 153 IU/L (10-60), AST 88 IU/L (10-42), Alk Phos 82 IU/L (38-126), Direct Bilirubin 2.0 mg/dL (0.0-0.2), and Indirect Bilirubin 2.4 mg/dL (0.2-1.3). Methimazole was discontinued and LFTs normalized within 4 days. CASE 2: A 92-year-old woman with a past history of atrial fibrillation presented with weight loss and poor appetite and was diagnosed with amiodarone-induced hyperthyroidism. LFTs were normal. She was started on metoprolol, prednisone and methimazole 30 mg daily. Approximately 6 weeks later, abnormal LFTS were noted incidentally during an unrelated hospitalization: ALT 95 IU/L, AST 73 IU/L, GGT 450 IU/L, Alk Phos 187 IU/L. Methimazole was stopped and LFTs returned to normal by day #6. CASE 3: A 65-year-old woman with a history of coronary artery bypass grafting and atrial fibrillation presented with weight loss and tremulousness and was diagnosed with amiodarone induced hyperthyroidism. She was started on metoprolol and methimazole 30 mg daily. The patient came to the ER 5 weeks later with poor appetite and syncope and was noted to have elevated LFTs: ALT 277 IU/L, AST 291 IU/L, Alk Phos 273 IU/L. Methimazole was changed to propylthiouracil and LFTs returned to normal in 5 days.

CONCLUSION: Methimazole-induced hepatotoxicity is generally thought to be uncommon. These cases raise the question of whether patients who are older, who have co-morbid conditions or who have received amiodarone may be more susceptible to this adverse reaction. Clinicians should be aware of this possibility and monitor LFTs in these patients in order to facilitate early recognition of this syndrome and prevent worsening of hepatotoxicity.

PACHYDERMOPERIOSTOSIS- AN UNUSUAL CAUSE OF ARTHRITIS

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Case presentation: A 32 year-old Hispanic male was admitted to the trauma service after a motor vehicle accident. Endocrinology consultation was requested for questionable acromegaloid appearance. The patient initially denied any symptoms prior to admission but, on direct questioning, he did state that his hands and feet had gotten bigger over the last 5 years, his shoe size had increased and that he had some pain and stiffness in his hands, but minimal limitation in his activities of daily living. His forehead had also become more prominent. There was no significant past medical, surgical, social or family history. Physical examination revealed a normotensive male with BMI 23 kg/m²; he had coarse facial features with seborrheic dermatitis of his scalp and face. He also had an enlarged, protruding jaw, enlarged hands and feet with skin thickening, and mild clubbing. There was no evidence of congestive heart failure, valvular dysfunction or joint abnormalities. Initial laboratory workup revealed a normal comprehensive metabolic panel, IGF-1 127 ng/ml (106-255); prolactin, testosterone, LH, FSH, TSH, free T4 were all normal. Repeat laboratory evaluation specifically assessing growth hormone overproduction was all within reference ranges as well: IGF-1 122ng/ml (106-255), IGFBP-3 2710ng/ml (2500-5806), GH 0.26ng/ml (0.01-1.0) and Insulin 4μU/ml (3.5-30). MRI of the pituitary to rule out a 'burnt-out' tumor was negative. X-rays of the hand and distal ulna revealed hyperostotic bone dysplasia and cortical thickening (typical for pachydermoperiostosis).

Conclusion: We present a case of pachydermoperiostosis or primary hypertrophic osteoarthropathy, one of the differential diagnoses of acromegaloidism, and often confused with acromegaly. Diagnosis is based on characteristic phenotypical and radiological abnormality and the exclusion of other diseases. This disease is a rare genetic disorder, and can lead to significant disability with musculoskeletal morbidity with advancing polyarthritis. It is more common in African American males; our case is unusual given his Hispanic origin. It is important to recognize this condition as a differential in workup of acromegaly since no hormonal abnormality (growth hormone or otherwise) is associated with pachydermoperiostosis. Treatment is usually conservative involving NSAIDs and treatment of complications of arthritis.

SPORADIC GASTRINOMA RESECTION RESULTING IN CURE OF SCHIZOPHRENIA: A PECULIAR NEUROPSYCHIATRIC RELATIONSHIP

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Introduction: Gastrinomas are neuroendocrine tumors arising from endodermal stem cells in the pancreas or small intestine. The triad of gastric acid hypersecretion, severe peptic ulcer disease and non-beta islet cell pancreatic tumors are known as the Zollinger-Ellison syndrome (ZE). This report documents the resolution of a longstanding and severe psychiatric illness coincident with curative surgery for ZE.

Case Report: A 62 year old woman was admitted multiple times in the past for nausea, vomiting and diarrhea. Her history was significant for chronic paranoid schizophrenia and depression for over 10 years, for which she was on risperidone 2mg daily. Evaluation revealed esophageal and gastric erosions and ulcerations, elevated serum gastrin levels and a positive secretin test. Endoscopic ultrasonography revealed a 1.4 x 1.2 x 1.2cm mass in the pancreatic head. Cytologic analysis of a fine needle aspirate confirmed the presence of a gastrinoma. Metastatic disease was excluded by octreotide scan. The patient underwent an ERCP to stent the CBD prior to surgical removal of the gastrinoma. She was sent home without any psychotropic medication and at one year follow-up remains free of psychiatric symptoms. **Discussion:** Gastrinomas present with symptoms usually associated with PUD and NSAID-induced gastritis, relieved by antisecretory drugs. Approximately 80% of them are sporadic, the remainder being found in association with MEN Type 1. One third of patients found with gastrinoma have metastatic disease and have a poor prognosis in spite of chemotherapy. A few case reports and preliminary uncontrolled studies have demonstrated an interesting link between neuropeptides and psychiatric disorders. "Gut-brain" peptides, like gastrin, secretin and cholecystokinin, may be involved in neurochemical alterations such as modulation of brain dopamine function producing neuroleptic-like effects on dopamine regulated behavior. In rodents, gastrin releasing peptide (GRP) and its receptor (GRPR) are importantly involved in regulating behavioral aspects that might be altered in autism, schizophrenia, depression and dementia. In one particular study, intravenous injections of secretin resulted in substantial improvement of clinical symptoms in a group of patients with treatment refractory schizophrenia.

Conclusion: Further studies are warranted to elucidate the role of neuropeptides in neuropsychological disorders.

ATLANTICARE REGIONAL MEDICAL CENTER

AN UNUSUAL FOREIGN BODY IN HEART

Ali Seifi

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An Unusual Foreign Body In Heart. Ali Seifi,MD. Associate, Zia Salam,MD,FACE, Tome Nascimento,MD,FACC, Howard Axelrod, MD. AtlantiCare Regional Medical Center, Atlantic City, NJ. Needle localized breast biopsy is commonly used to diagnose and remove non-palpable breast lesions. Although a variety of techniques can be used for needle localization, today it is most commonly performed with hook-wires or retractable curved wires. Few reports have specifically addressed the complications associated with needle localized surgical breast biopsy. We present the first case of post-operative cardiac injury, resulting from retained hook-wire, after needle localized breast biopsy. A 34-year-old female presented to the emergency department with progressively worsening intermittent chest pain for several months. The pain was left sided, stabbing and radiating to the back. It was associated with shortness of breath and palpitations. The patient had no known cardiac history. She had no other known cardiac risk factors. Her past medical history was significant for a left breast mass with needle localization and lumpectomy two years prior to admission. Pathology revealed benign fibrocystic change with ductal hyperplasia. On physical exam she was in mild distress. Her vital signs were stable. Jugular venous distension was noted. CBC, electrolytes and cardiac enzymes were within normal limits. EKG revealed sinus rhythm with no evidence of ischemic changes. Echocardiogram was performed, which revealed a large pericardial effusion with the evidence of early tamponade. Also noted was a linear echodensity seen in the left ventricle, which continued through the aortic valve into the aortic root. The linear echodensity was appeared to be traversing through the posterolateral free wall of ventricle into the pericardium. CT scan of the chest was performed. This revealed three metallic foreign bodies within the heart and chest wall. She was taken emergently to the operating room. Approximately one liter of blood was removed from pericardial space proving hemodynamic improvement. A wire was seen protruding through the inferolateral wall of the left ventricle. This was removed by being pulled through the left ventricular wall that measured 80mm. The innominate artery was opened, and a second segment of 30mm wire was visualized and removed. Patient tolerated the procedure well and was transferred to the cardiac ICU hemodynamically stable, and finally discharged to home without any complication. To conclude, the management of foreign bodies in the heart should be discussed on an individual basis. We have to consider that although the complications of needle localized surgical breast biopsy are infrequent, close communication between radiologist and surgeon during and after procedure is critical. A basic tenet of medical care is that still a complete history is the key to diagnosis. The danger of some iatrogenic complications should not be underestimated and physicians should be routinely aware of them.

LADY WINDERMERE SYNDROME

Magdalena Szulc, M.D.

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The designation, Non-Tuberculous Mycobacteria (NTM), encompasses the mycobacterial species other than organisms of the mycobacterium tuberculosis complex and *Mycobacterium leprae*. The NTM are distributed widely in the environment. Most species are less virulent for humans than mycobacterium tuberculosis. Thus, symptomatic infections are often associated with local or generalized defects in host defenses. A 66 year-old female, presented to the Emergency Department with pleuritic chest pain, progressive shortness of breath, and cough productive of scant sputum for several weeks. Review of systems revealed intermittent fever, weight loss, and decreased appetite. She has COPD, secondary to life-long smoking, necrotizing pneumonia five years ago, and deep vein thrombosis with IVC filter placement. She worked in the Respiratory Care Department. She did not have any HIV risk factors and one of her yearly PPD tests was positive, but she refused treatment. Her CT scan of chest without contrast showed multiple non-enlarged mediastinal lymph nodes, right upper lobe composed predominantly of a large air-filled structure with a nodular border, a small amount of dependent fluid, consolidation within the lingual and inferior aspect of the right upper lobe, and multiple pleural-based and parenchymal nodules bilaterally. She had bronchoscopy and washings were negative for malignancy and AFB. Sputum was positive for AFB. She was placed on respiratory isolation and anti-TB medications were begun. A few days later, samples of her sputum culture were positive for *Mycobacterium avium-intracellulare* complex. She improved and was discharged from the hospital with follow up with Infectious Disease and Pulmonology. Asymptomatic infections with NTM are common in humans. Studies with skin tests derived from NTM indicate that 30 to 40% of adults in the northern and southern U.S. have had prior unrecognized or asymptomatic infection with NTM, most often with organisms of the mycobacterium avium complex (MAC). This suggests that possibly we should start screening elderly patients for TB or MAC if they present with shortness of breath and cough, with or without history of lung disease or smoking. Without lung disease and smoking history, our index of suspicion for TB and MAC would be rather low. We have to be aware of radiological findings of NTM including lingual involvement and prominent lymph nodes, especially in non-smokers, which gives a picture of Lady Windermere syndrome. Treatment is a long and multi-drug regimen, although approximately 20% of patients experience treatment failure, relapse, or re-infection. Surgical resection may also be performed for patients with localized disease who are intolerant or unresponsive to multi-drug therapy; however, this is associated with postoperative complications in as many as 20% of patients. In conclusion although we usually look for the most common causes of diseases, we also have to consider the atypical causes of common diseases.

EOSINOPHILIC STORM

Wesam Moustafa Hussein, M.D.

Co-Authors: Ricardo Barzaga MD, William Rodriguez MD, and Francis Salvatore MD

In 1971, George Wells first described a syndrome of recurrent granulomatous dermatitis with eosinophilia. Wells and Smith renamed it eosinophilic cellulites. Eosinophilic cellulitis (Wells syndrome) is an uncommon, rare condition of unknown etiology. Although Wells syndrome is usually sporadic, some familial cases have been reported. This is a 30 year old white male with a history of asthma who was referred because of an erythematous, pruritic lesion on his left thigh. The lesion was an itchy, indurated and erythematous plaque measuring 31x25mm., and multiple small satellite papules were noted. Patient does not recall any history of insect bite or trauma to the area. The patient was prescribed Keflex initially and had some resolution of the surrounding erythema. After a 10 day course of antibiotics the rash reoccurred. However, the lesions were more pruritic this time and more diffuse involving the trunk. He was admitted and was started on IV antibiotics and diphenhydramine. Hepatitis C, RPR and HIV tests were obtained the results were all negative. Patient did not improve on the above therapy. A skin punch biopsy was obtained. The biopsy showed Epidermal spongiosis, subepidermal edema, with characteristic 'flame figures' and dense infiltrates composed mostly of lymphocytes and eosinophilic leukocytes found in the dermis and subcutis. Patient was diagnosed with Well's syndrome. He was started on IV steroids after 4 days the lesions subsided and patient was discharged. Eosinophilic cellulitis (Wells syndrome) is an uncommon condition of unknown etiology. The presentation usually involves a mildly pruritic or tender cellulitis like eruption with typical histologic features characterized by edema, flame figures, and a marked infiltrate of eosinophils in the dermis. The condition can recur and may be preceded by a pruritic papular eruption. Although systemic steroids appear to be the only therapeutic modality of benefit in Wells' syndrome, antihistaminics and dapsone have been found to be useful. Familiarity with this syndrome is critical to institution of the right diagnostic and therapeutic approach.

AN EXTREMELY RARE CASE OF BLEOMYCIN INDUCED ACUTE CORONARY SYNDROME

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Introduction- Cancer patients receiving chemotherapy have an increased risk of developing cardiovascular complications, and the risk is even greater if there is a known history of heart disease. We present a rare case of acute coronary syndrome following first dose of Bleomycin infusion which has not been described previously. Bleomycin is a class of chemotherapy agents that works by inhibiting topoisomerase two. The common side effects of Bleomycin are dose-dependent pulmonary fibrosis and skin changes. There are few case reports of occurrence of acute chest pain during Bleomycin infusion in young patients for which infusion were continued without any sequelae. Currently there is no recommendation for cardiac evaluation before Bleomycin treatment in patients with known cardiac history or significant risk factors. Our patient is a 66-year-old gentleman with past history of quadruple coronary artery bypass graft surgery in 1998, myocardial infarction (MI) in 2003 with subsequent stents placement, type 2 diabetes mellitus and hyperlipidemia. His last cardiac catheterization in 2006 showed patent grafts and stents. He was worked up for his normocytic normochromic anemia which ultimately revealed bone marrow invasion with Hodgkins' lymphoma. One month after diagnosis and following Port-A-Cath placement, chemotherapy with Bleomycin alone was initiated in hematology office. Toward the end of his first Bleomycin infusion, he developed acute substernal chest pain which was initially relieved by sublingual nitroglycerin but recurred later. The infusion was completed and then he was sent to hospital for further evaluation due to his extensive cardiac history. Initial EKG and cardiac enzyme as well as coronary CT angiogram were unremarkable but second set of cardiac enzymes became strongly positive. He was transferred to the coronary care unit with the impression of non-ST elevation myocardial infarction. The next day percutaneous coronary intervention showed a 75% narrowing of the second obtuse marginal branch. He had an uneventful hospital course following successful placement of a drug eluting stent. Conclusion- Although rare, but Bleomycin can potentially induce acute coronary syndrome and MI in patients with significant coronary artery disease. Thorough cardiac evaluation before initiation of treatment as well as vigilant attention to signs and symptoms of acute coronary syndrome during Bleomycin infusion is recommended. The safety of Bleomycin use following acute chest pain in patients with significant cardiac history remains unclear.

DREXEL UNIVERSITY COLLEGE OF MEDICINE, SAINT PETER'S UNIVERSITY HOSPITAL

A CASE OF HYDATID DISEASE OF THE LIVER

Abilash Balmuri, M.D.

Co-Authors: Harshit Khara, M.D. and Kiran Allam, M.D.

INTRODUCTION: Hydatid disease, a parasitic infestation by a tapeworm of the genus *Echinococcus*, is very rare in the United States with an incidence of <1 per million. The recent rise in the number of cases might be attributed to an increase in the number of immigrants from endemic areas.

CASE PRESENTATION: 39 year old Caucasian female, who emigrated from Georgia presented with increasing right upper abdominal discomfort for three months. She was diagnosed with a liver cyst during antenatal testing six years ago, but was lost to follow-up. There was no history of blood transfusions, contact with pet or farm animals. On further questioning, she admitted to having consumed water from an open well as a child. Physical examination was negative for pallor, scleral icterus or other signs of liver failure. Cardio-respiratory examination was normal. Abdominal examination revealed a distended abdomen and an enlarged liver with a smooth edge palpable 7 cm below right costal margin. There was no clinical evidence of ascites or portal hypertension. Laboratory studies showed a normal hemoglobin, with no leucocytosis or eosinophilia. Liver function tests were abnormal and as follows: AST – 269 IU/L; ALT – 197 IU/L; alkaline phosphatase – 1047 IU/L; total bilirubin – 1.1mg/dL. Albumin, and prothrombin time were normal. Abdominal ultrasound showed a large hepatic cyst measuring 14.6 x 11.1 x 13 cm with evidence of intrahepatic biliary dilatation, but no hydatid sand. The patient tested positive for Echinococcal antibody (IgG ELISA). Hepatic cystectomy was done with appropriate precautions to prevent spillage of the cyst fluid. Biopsy showed laminated cyst wall with numerous scolices compatible with *Echinococcus granulosus*. Patient underwent chemotherapy with albendazole.

DISCUSSION: Hydatid disease (echinococcosis) is prevalent in the Mediterranean region and Middle-eastern countries. *Echinococcus granulosus*, commonly called the dog tape worm, has the dog, or other canines as its definitive hosts, while sheep and other farm animals are the intermediary hosts. Humans are accidental hosts acquiring the infection by food and water contaminated by dog feces. Humans ingest the eggs which hatch into oncospheres in the intestine, penetrate the intestinal mucosa to enter the portal circulation and migrate to the liver, lungs, muscles, brain and kidney. As hydatid cysts may be misinterpreted as simple cysts (as in this patient) radiologically, it is always important to rule out echinococcosis by serology, especially in patients from endemic regions, or those with a history of travel to these regions. Spillage of the unsterilized cyst fluid during aspiration or surgery may result in life-threatening anaphylaxis or secondary seeding of daughter cysts leading to recurrence. Treatment options include surgery, and a newer technique called PAIR (puncture, aspiration, injection, and reaspiration). Chemotherapy with antiparasitic drugs (albendazole and mebendazole) serves as an adjunct to either surgery or PAIR.

AN UNCOMMON CAUSE OF ABDOMINAL PAIN ---GASTRO INTESTINAL STROMAL TUMOR

Leena Bodapati, M.D.

Co-Author: Kathleen Toomey, M.D.

AIM - To report a rare case of gastrointestinal stromal tumor (GIST), presenting as vague abdominal pain of one year duration.

Case Presentation--- 79 year old women with a history of multiple myeloma, anemia, diabetes and chronic renal insufficiency presents to the clinic complaining of abdominal pain for one year duration. The pain was located at the epigastrium, intermittent, dull in nature, non-radiating, of mild to moderate intensity and associated with early satiety resulting in a 20 pound weight loss. Patient denies any nausea, vomiting, hematemesis, melena or hematochezia. On abdominal examination she had mild epigastric tenderness but no masses or organomegaly could be appreciated. Her laboratory studies showed a hemoglobin of 9.5 gram/dl. Ultrasonography of abdomen revealed a heterogenous mass with dimensions 18x18x18 cm in left upper quadrant. Subsequent CAT scan of abdomen showed 15 cm mass in left upper quadrant fixed to the peritoneum between stomach, pancreas, spleen and transverse colon and arising from the greater curvature of the stomach. Surgical excision with partial gastrectomy was performed. Histopathological study of specimen showed spindle cells with eosinophilic cytoplasm and high mitotic activity. Immunohistochemical stain of specimen was positive for CD117 leading to the diagnosis of GIST. Patient thereafter was started on imatinib therapy. Discussion--- GISTs are rare but common mesenchymal neoplasms of the gastrointestinal tract. GISTs are most commonly found in the stomach (60-70%) followed by the small intestine (20-30%), the colorectum (10%) and esophagus (<5%). They may also occur as primary tumors outside the gastrointestinal tract, especially in the omentum, mesentery and retroperitoneum. The most common clinical manifestation of symptomatic stromal tumors in gastrointestinal tract is bleeding caused by mucosal ulceration. Patients may present with hematemesis, melena, hematochezia or signs and symptoms caused by anemia due to occult bleeding. Other symptoms are abdominal pain with a palpable mass or the patient may remain asymptomatic. GIST is diagnosed during routine tests like upper endoscopy, laparoscopy, colonoscopy, barium X-rays, ultrasound of abdomen, CAT scans, MRI and PET scans. Tissue sample or biopsy is needed for the diagnosis along with the presence of Kit/CD117 on immunohistochemical stain. Complete surgical excision is the treatment for localized GIST. For in-operable and metastatic tumors imatinib mesylate can be used.

ENDOSCOPIC ULTRASOUND AS A DIAGNOSTIC AND THERAPEUTIC TOOL IN PANCREATIC LESIONS: A STUDY OF 30 CASES

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Co-Authors: Harshit Khara, MD, Sandeep Bhargava, MD and C. S. Pitchumoni, MD

The increasing utilization of CT scan of abdomen detects a number of silent pancreatic lesions- both cysts and masses. Endoscopic ultrasound (EUS) is a recent but extremely useful diagnostic modality which gives high resolution images of pancreas without interference by overlying bowel gas. Also, it has evolved from a simple imaging modality to one that can also be used both for diagnostic and therapeutic procedures. The ability of EUS to identify the pancreatic lesions that are too small to be identified by CT or MRI and to guide a needle both for biopsy or drainage, gives it a clear advantage over other imaging modalities in investigating pancreatic lesions. Material and Methods: We reviewed the charts of 30 patients who underwent EUS at our hospital in last three years for suspected pancreatic mass lesions as seen on CT or MRI. The data was obtained from the endoscopic service records. Findings: A total of 30 patients had EUS evaluation for pancreatic pathology. The study group comprised of 16 males and 14 females with a mean age of 56.9years (range 27-79 years). Main Indications of EUS in these cases were abnormal CT/MRI findings, suggestive of mass/cyst in the pancreas. • 13 out of 30 cases had pancreatic cystic lesions. - 10 out of these cases were completely drained. - 3 cases which could not be drained, 2 had large blood vessels surrounding the cyst and in 1 case the cyst was very large (6cm in diameter) located in tail of pancreas. • 11 out of 30 cases showed solid or complex partial mass lesions. All these cases had fine needle aspirations (FNA) done and sent for cytology. • 5 out of 30 cases were evaluated for recurrent chronic pancreatitis with suspected pancreatic mass on CT/MRI - 4 showed no mass - 1 showed tortuous pancreatic duct with salt and pepper appearance in parenchyma. • 1 out of 30 cases was evaluated for MRI finding of annular pancreas, EUS confirmed this finding with no mass lesion. Conclusions: In spite of availability of EUS at most institutions, it has been not been widely used for evaluation of Pancreatic pathology. In this study we have tried to highlight the unique ability of EUS as imaging modality that allows invasive procedure like FNA of pancreatic lesions and endoscopic pancreatic cyst drainage. The possibility of using EUS for both diagnostic and therapeutic purpose gives it superiority over other imaging modalities like Ultrasound/CT/MRI and MRCP. The disadvantages of EUS are that it needs a great deal of technical expertise, state of the art equipment and the need for IV sedation during the procedure.

USE OR ABUSE OF ABDOMINAL CT SCAN IN THE EMERGENCY DEPARTMENT

Abilash Balmuri, M.D.

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BACKGROUND: Abdominal pain is one of the most frequent reasons for emergency department (ED) visits and upto 40% get a computed tomography (CT) scan. The number of annual CT scans done in the US are an estimated 62 million. There is a perception that a large number of these scans are done without any valid indication. Although studies have evaluated the use of CT scans in specific conditions like appendicitis, renal colic, and abdominal trauma, the utility of CT abdomen in the ED has been seldom studied. **AIM:** To evaluate the clinical utility of abdominal CT done in adult patients presenting to the ED. **METHODS:** This is a retrospective study of abdominal CT scans done in the ED over a month, in a 450 bed hospital. We reviewed the records to determine the clinical context, using the ED physicians' documentation of history, physical examination and initial laboratory values prior to ordering the CT scan. **RESULTS:** Total ED visits: 3545. Total abdominal CT scans: 176. 1) Normal CT scans: 48(27 %). 2) CT scans with nonspecific/inconclusive findings: 39(22%) 3) Total number of CT scans, which were either normal or had inconclusive findings that did not help in the diagnosis: 1+2=87(49%). 4) In the above group 3, the number of CT scans that were justified using clinical and laboratory evidence: 28(32%). CT scans not justified by these criteria: 59(68%) 5) CT scans with abnormal findings: 89(51%) 6) In group 5, CT scans not backed by clinical and laboratory findings = 26(29%). CT scans backed by the above criteria = 63(71%) 7) CT scans with incidental findings = 75(43%). These include, but are not limited to fatty liver, diverticulosis, cysts of adrenal, liver, ovary and pancreas and cholelithiasis. 8) The positive predictive value of having a CT with abnormal findings increases by more than 100% (69%vs 31%), if ordered judiciously after evaluating clinical/laboratory findings. **CONCLUSIONS:** A prudent use of CT abdomen involves a careful assessment of its indication by appropriate clinical and laboratory findings. The increased use (or abuse) of CT abdomen in the ED may be explained by: a) the urgency to promptly arrive at a diagnosis; b) the high volume and nature of patients seen; c) an excessive reliance on imaging studies; d) a reasonable desire to avoid missing critical illness (surgically treatable); and e) fear of litigation. The negative aspects of indiscriminate CT scans are: 1. Increased incidental findings that may lead to anxiety, and a futile path of further diagnostic testing although, a small percentage of treatable lesions may also be identified. 2. Potential radiation hazard. 3. Wasteful expenditure. Reference: Brenner DJ, Hall EJ. Computed Tomography — An Increasing Source of Radiation Exposure. N Engl J Med 357:2277, Nov 2007.

JERSEY CITY MEDICAL CENTER

A CASE OF PRIMARY SPLENIC LYMPHOMA AND AUTOIMMUNE HYPOTHYROIDISM IN A PATIENT WITH HEPATITIS C

Carla Wang-Kocik, MD

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USA Primary splenic lymphoma is rare with a reported incidence of less than 1%. There are several studies of primary splenic lymphomas in patients with Hepatitis C. We describe a patient with history of hepatitis C who presented with severe hypothyroidism. Abdominal mass proved to be primary diffuse large B-cell lymphoma of spleen. A 50 year old man with history of depression and hepatitis C presented with altered mental status, dehydration and hypercalcemia. Thyroid function tests showed primary hypothyroidism with decreased in T3, T4 and elevated TSH as shown [(FT4=0.05 ng/dl (0.58-1.64); FT3=0.0 ng/dl (87.0-178.0); TSH=69.62 IU/ml (0.34-5.60)]. Autoimmune hypothyroidism was diagnosed based on elevated thyroglobulin antibody [6838 IU/mL, (I 0-40)] and elevated thyroid peroxidase antibody [109 IU/mL, normal (0-34)]. An ultrasound and computed tomography of abdomen showed a large splenic tumor. Patient underwent splenectomy and resection of distal pancreas. Histopathology revealed diffuse large B-cell lymphoma of the spleen. Unfortunately, patient developed cardiac arrest during surgery and expired later in the hospital. Association of Hepatitis C (HCV) with autoimmune thyroid disease specifically Hashimotos thyroiditis have been described in the literature. Albert Tran et al. described the association of Hepatitis C and autoimmune thyroid disease with a p value of 0.02 in his study. Another study showed that patients with HCV are more susceptible than patients with HBV to autoimmune thyroid disease. Other studies have found elevated levels of thyroid antibodies in up to 42% of patients with HCV infection. On the other hand, B-cell non-Hodgkin's lymphoma has also been linked to HCV infection. One study linked HCV infection and splenic B-cell lymphomas in seven out of nine patients with splenic lymphoma who were treated with interferon monotherapy. Hermine et al. described regression of splenic lymphoma after treatment of Hepatitis C virus infection. Our patient was unique in having Hepatitis C, autoimmune thyroid disease and splenic lymphoma, which was confirmed by pathology after splenectomy.

ANTI-GLOMERULAR BASEMENT MEMBRANE GLOMERULONEPHRITIS: EARLY DIAGNOSIS IS CRITICAL

Austine Mengnjo, MD

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Acute glomerulonephritis due to anti-glomerular basement membrane antibody disease is rare, with a US prevalence rate of less than 1/1,000,000. It is characterized by circulating antibodies directed against antigen intrinsic to the glomerular basement membrane (GBM), thereby resulting in acute or rapidly progressive glomerulonephritis (RPGN) typically associated with crescent formation. We present a case of anti-GBM glomerulonephritis who rapidly progressed to end stage renal failure (ESRD). A 66 year-old African American female with past medical history of hypertension, recently diagnosed congestive heart failure (CHF) presented with shortness of breath, leg swelling, weakness, and decreasing urine output for one week. She denied hemoptysis. Two weeks before admission, she was treated for CHF her serum creatinine level that time was 1.38mg/dL, and ejection fraction, 35%. She was discharged on lisinopril 5mg and furosemide 20mg. Pertinent physical examination showed: pulse rate of 78/min; blood pressure of 173/87 mmHg; and temperature of 99.7 Fahrenheit. There were scant basilar crackles and an S3 gallop and a 2+ ankle edema. Pertinent laboratory findings were: hemoglobin of 10.8g/dL, sodium of 133mEq/L; potassium of 6.6mmol/L; chloride of 105mmol/L, bicarbonate of 16mmol/L, and B-type natriuretic peptide of 2000pg/mL. Blood urea nitrogen (BUN) was 69mg/dL, and creatinine was 11.15mg/dL. An arterial blood gas revealed non-anion gap metabolic acidosis. Urinalysis was significant for 2+ proteins, 7-10 dysmorphic erythrocytes/high power field, and fine granular casts. She was admitted into the intensive care unit and emergency hemodialysis was started. Further laboratory work-up revealed normal titers of anti-nuclear antibody, anti-double stranded DNA, cytoplasmic-staining antineutrophil cytoplasmic antibodies (c-ANCA), and perinuclear-staining antineutrophil cytoplasmic antibodies (p-ANCA). Serum complement levels were normal. The anti-glomerular basement membrane antibody titer was high (1:128). Renal biopsy revealed diffuse acute crescentic glomerulonephritis with linear glomerular basement membrane IgG staining. Plasmapheresis and immunosuppressive therapy with intravenous methylprednisone and cyclophosphamide were instituted. Renal function did not improve after three weeks of therapy and she was placed on permanent dialysis. Anti-GBM antibody disease is one of the three major forms of RPGN. Although some patients present with relatively mild renal insufficiency, this disorder is typically associated with severe renal injury that, if untreated, progresses quickly to ESRD. An important determinant of the response to therapy and long-term prognosis is early diagnosis. There is a direct correlation between the initial plasma creatinine concentration and the percent of glomeruli with crescents. Avoidance of maintenance dialysis is rare in patients who require dialysis within 72 hours of presentation, particularly in those who have crescents involving all glomeruli. Prevention of end-stage renal disease can usually be achieved in less severe cases, although some do progress. The proportion of preserved glomeruli may be the best determinant of prognosis.

HYPERNATRAEMIA CORRECTED BY INTENSE DIURESIS IN CRITICALLY ILL

Shikha Sharma

Co-Authors: Jyoti Matta and Abdul Ameer

"Background Hyponatremia is commonly recognized electrolyte abnormality in critically ill patients associated with dehydration. We report a case of hypernatremia with generalized anasarca and hypervolemia. Case A 72-year-old Polish female was hospitalized with generalized weakness and worsening dyspnea. Her medical history was significant for ischemic cardiomyopathy, and hypertension. On examination, she weighed 93 kg and had crackles at left lung base, +1 bilateral pitting pedal edema. Laboratory data showed elevated creatinine (1.38), BUN (36) and hyponatremia (Na 128). Chest X-ray showed bilateral pulmonary congestion and cardiomegaly. On Day 1, patient developed cardiac arrest with monomorphic ventricular tachycardia that was cardioverted and she was resuscitated. Because of sepsis and associated hypotension, she was given approximately 4 liters of isotonic fluid over next 24 hours. Her serum sodium increased to 138 with weight gain and progression of edema. During next 14 days the patient remained critically ill, intubated and received a variety of nasogastric and intravenous fluids. Her weight had increased to 103 kg and sodium to 153. She had distended neck veins and anasarca. Mean pulmonary capillary wedge pressure was 20 mm of Hg, RA pressure was 9, RV pressure was 80/6 mm of Hg and pulmonary artery pressure was 72/30. She had metabolic alkalosis (pH 7.52 and bicarbonate 38, chloride 109). We managed her with aggressive natriuresis induced by different classes of diuretics along with hydration with intravenous dextrose water and intermittent free water bolus. Urine Osmolality increased from 352 to 604 mosm/kg and urine sodium increased from 5 to 70 mmol/L in 72 hrs with combination of metolazone, furosemide, acetazolamide, spironolactone. In addition to decline in weight from 103 to 96 kg with net negative free water clearance, serum sodium also declined from 153 to 145 mmol/L within 48 hours. However, etiology of hypernatremia in this case remains unknown in presence of normal serum aldosterone, ADH and renin levels but various factors including medications, nasogastric feeding, decompensated heart failure, thyroid disease, recent acute tubular necrosis might have contributed to this complex paradoxical electrolyte imbalance.

Discussion: Hypernatremia is characterized by either absolute (hypovolemic) or relative (euvolemic) free water deficit. Therefore, treatment of these disorders involves replacement of free water deficit with hypotonic intravenous fluids. On the contrary, hypervolemic hypernatremia is caused by increase in total exchangeable sodium and potassium ion in excess of the increment in total body water, resulting in relative free water deficit. Treatment of these patients is challenging, as they are Hypervolemic. They have relative free water deficit as a result of positive sodium balance and positive water balance. And treatment in these cases should be targeted at both the correction of hypernatremia and the attainment of a negative free water balance as we achieved in our case.

IDIOPATHIC PORTAL HYPERTENSION, NEPHROLITHIASIS AND PANCYTOPENIA: SHOULD IT BE RECOGNIZED AS SYNDROME IN SOME VARIANT OF CELIAC DISEASE?

Manish Sharma, MD

Co-Authors: Matt Chua, MD, Anh Thu Tran, MD, and Joykumar Patel, MD

Idiopathic portal hypertension (IPH) is diagnosed in patients with clinical symptoms of portal hypertension, patent portal vein, and the absence of morphological signs of liver cirrhosis. We describe a case of idiopathic portal hypertension in a young patient with nephrolithiasis and possible celiac disease. A 33 year-old Indian male with history of recurrent bilateral nephrolithiasis for one year was admitted to our hospital for acute renal colic pain. He denied any fever, weight loss, skin rashes, joint pain, nausea/vomiting or diarrhea. Past medical history was significant of thrombocytopenia for 15 years with non-specific bone marrow aspirate and biopsies. Past surgical history revealed an appendectomy with pathology of chronic granulomatous disease. Family history was unremarkable. Physical examination was notably for splenomegaly without any stigmata of chronic liver cirrhosis. The rest of physical examination was unremarkable. Complete blood count showed hemoglobin of 10.4 g/dL, hematocrit of 31.7%, white blood cell count of 3,300/uL with normal differential, and platelet of 56,000/uL. Iron, ferritin and vitamin B12 levels were low. Liver function test and coagulable profile were within normal limits. Ultrasonography (US) of abdomen confirmed a splenomegaly of 19cm in dimension with normal caliber portal vein. Computed tomography (CT) of abdomen without contrast showed bilateral kidney stones without obstruction. Both US and CT of abdomen did not reveal liver disease. Previous work-up found grade III esophageal varices which were banded and a chronic non-specific inflammation of the colon. Further work-up were negative human immunodeficiency virus, hepatitis B and C. Serology tests were negative. Due to unexplained pancytopenia, splenomegaly, portal hypertension without liver disease and recurrent nephrolithiasis, a malabsorptive disorder was suspected. Serum IgG tissue-transglutaminase by ELISA was positive. He was given intravenous iron and subcutaneous vitamin B12 to correct his anemia. A gluten free diet will be tried after a small bowel biopsy to confirm celiac sprue disease. Unfortunately, he was lost to follow up. Although our patient does not meet the criteria to diagnosis of celiac disease, his positive IgG tissue-transglutaminase, iron and vitamin B12 deficiency anemia and bilateral nephrolithiasis highly suggest celiac sprue disease. A small bowel biopsy and gluten free diet may help to confirm this condition. Splenomegaly, pancytopenia and moderate esophageal varices are clinical symptoms of IPH without liver cirrhosis. A splenectomy and/or a splenorenal shunt are the main treatment of IPH after histopathological of liver biopsy can exclude liver cirrhosis. To our knowledge, this is the second case of idiopathic portal hypertension associated with celiac disease. It should be recognized a variant of celiac disease in a patient with IPH, nephrolithiasis and pancytopenia.

INFECTIVE ENDOCARDITIS COMPLICATED BY MYOCARDIAL INFARCTION (MI), VENTRICULAR FIBRILLATION AND CARDIAC ARREST

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Infective Endocarditis (IE) is known to cause systemic complications including microembolization of coronary vessels. However, a fatal transmural left heart vessel blockage is still a rare and dreaded complication of IE. We present a case in which a patient suffered from MI the day after getting her affected valve repaired. A 49 year old female with history of diabetes and hypertension presented with severe respiratory failure and sepsis. Echocardiogram revealed 41 x 10 mm vegetation attached to the posterior leaflet of native Mitral Valve (MV), associated with severe mitral regurgitation and pulmonary hypertension. Cardiac catheterization showed a lesion in Left Anterior Descending (LAD) artery. Patient was taken for open heart surgery and successfully received MV replacement and coronary artery bypass surgery for LAD blockage. Post-operative course was uneventful for one day. The next day, patient had ST segment elevation in electrocardiogram in LAD distribution, followed by ventricular fibrillation and cardiac arrest. Patient was successfully resuscitated but developed a new left bundle branch block, Mobitz type II 2:1 block and atrial flutter. Patient could not be taken for emergent cardiac catheterization due to her condition; only medical management was carried out. Blood culture grew ESBL-negative *Klebsiella pneumoniae* and antibiotics were appropriately adjusted. Patient's condition improved in the next ten days. Our case highlights the rare possibility of developing fatal embolic acute coronary syndrome due to severe IE, even after valve repair. Such complications have sparsely been described in case reports. However, in most of those cases, virulent staphylococci aureus has been the culprit. In our case, *Klebsiella pneumoniae* was identified as the etiology. Additionally, the challenge in such cases is in the decision to anticoagulate the patient if emergent angiogram and angioplasty is not an option. Unfortunately, studies have not been conclusive in this matter.

ISOLATED RENAL ASPERGILLOMA IN AIDS: A CASE REPORT

Matt Chua, MD

Co-Authors: Carlos Martinez, MD, and Anh Tran, MD

Aspergilloma is well described in the lungs, but very rarely in the kidneys. Involvement of the kidneys usually spreads from disseminated disease. We describe a case of isolated renal aspergilloma in an AIDS patient. A 52 year-old intravenous drug abusing African American male with AIDS diagnosed since 1997 (recent CD4 of 20 cells/UL, non compliant with HAART therapy), presented with 3 day history of bilateral upper quadrant pain radiating to the back associated with loss of appetite and generalized weakness. Physical examination revealed tenderness in the epigastric, right upper quadrant and left costo-vertebral angle. Pertinent laboratory results showed white blood cell count of 3,300/UL with absolute neutrophil count more than 2,000/UL; hemoglobin of 9.6g/dL and hematocrit of 28.6. Urinalysis showed more than 10 leukocytes per high power field, and large leukocyte esterase but no cast. Urine culture did not grow any bacteria. Computed tomography scan (CT) and ultrasound of abdomen found a mass with dimension of 4.0 cm by 6.0 cm in the lower pole of the left kidney. CT of the chest was normal. Patient was empirically started on antibiotics. Clinically patient did not respond to antibiotics and still complained of abdominal pain. The patient underwent CT guided renal biopsy of the mass. Histopathology revealed acute and chronic pyelonephritis with necrosis and invasive fungal hyphae showing septation with dichotomous branching, confirmed Aspergillus infection. Patient then was started on caspofungin acetate daily, but he initially refused surgical intervention. He was continued on intravenous caspofungin in inpatient rehabilitation center. However, persistent symptoms led him to be hospitalized again. He underwent nephrectomy of the left kidney, and was discharged on oral voriconazole. Unfortunately, his condition deteriorated over the next few months and the patient subsequently died. Isolated renal aspergilloma without dissemination is a rare complication in AIDS patients especially without significant neutropenia. Its fatal outcome may present an advanced stage of AIDS. In 2008 according to A.W. Oosten et al., there have been 19 cases of renal aspergilloma; among these cases 15 were unilateral. Most aspergillus infections have occurred in the setting of multiple opportunistic infections and are usually diagnosed at autopsy. Despite current therapy of nephrectomy with antifungal, renal aspergilloma still carries a bad prognosis and median survival time is 3 months.

LEFT VENTRICULAR NON-COMPACTION ASSOCIATED WITH POLYCYSTIC KIDNEY DISEASE

Anh Thu T. Tran, MD

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Left ventricular non-compaction is a rare congenital cardiomyopathy of unknown etiology characterized by prominent trabeculations, deep intertrabecular recesses and combined systolic and diastolic dysfunction. We report a case of left ventricular non-compaction associated with polycystic kidney disease (PKD). Coexistence of these two conditions might help us understand the theory of arrested embryonic development as the pathogenesis of left ventricular non-compaction versus a possible genetic mutation. A 66-year-old man presented with progressive shortness of breath. Patient mentioned one-year history of exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea and edema of the lower extremities. On examination, he was noted to have jugular venous distension and bibasilar crackles. The electrocardiogram showed sinus rhythm with a left axis, right bundle branch block and mild left ventricular hypertrophy. Laboratory studies revealed a brain natriuretic peptide (BNP) of 500 pg/mL. Chest x-ray showed pulmonary vascular congestion. Transthoracic echocardiogram revealed severe left ventricular systolic dysfunction with an ejection fraction of 25%; the left ventricular chamber was not dilated. Marked thickening of the left ventricular trabeculae, particularly in the apex and free wall of the left ventricle were identified. The prominent trabeculae and deep intertrabecular recesses that communicate with the ventricular cavity are the hallmarks of isolated left ventricular non-compaction syndrome. These features were seen on the patient's echocardiogram. Ultrasonography and computed tomography of the abdomen and pelvis showed multiple cysts in both kidneys. The patient was treated for heart failure and anticoagulation to decrease the risk of systemic embolism. He has been doing well since discharge. Left ventricular non-compaction is a cardiomyopathy characterized by deep trabeculations in the ventricular wall, which has recesses communicating with ventricular lumen. The syndrome is rare. Echocardiography is a simple and useful diagnostic procedure to diagnose left ventricular non-compaction. Polycystic kidney disease is characterized by formation of multiple cysts in the kidneys, liver, and less frequently, in the pancreas. Cardiovascular abnormalities including hypertension, mitral valve prolapse, and intracranial aneurysms are all associated with PKD. However, polycystic kidney disease and isolated ventricular non-compaction have not previously been correlated. To the best of our knowledge, this is the third reported case of left ventricular non-compaction associated with polycystic kidney disease. Because the genetic pathogenesis of non-compacted myocardium and polycystic kidney has not been fully clarified, it is uncertain if these two different hereditary diseases are etiologically related or not. Their coexistence in a patient like ours may help us understand the etiopathogenesis of these two congenital anomalies. Recognizing this syndrome by echocardiography may help to manage patients by traditional heart failure therapy in addition of anticoagulation to prevent systemic embolism. Our patient is unique to have both isolated- left ventricular non-compaction associated with polycystic kidney disease.

MYASTHENIA GRAVIS ASSOCIATED WITH NON-HODGKIN'S LYMPHOMA: COULD IT BE A PARANEOPLASTIC SYNDROME?

Carla Wang-Kocik, M.D

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USA Myasthenia gravis is a B-cell-mediated autoimmune neuromuscular disorder characterized by weakness and fatigability of skeletal muscles. We report a patient presenting with myasthenia gravis and found to have B-cell non-Hodgkin's lymphoma. Although their association is understandable with regard to the physiopathology, myasthenia gravis has less frequently been associated with lymphoma. A 63-year-old Hispanic woman with no significant past medical history presented with four month history of voice changes, choking sensation, and shortness of breath. The symptoms fluctuated but were progressive. One month prior to admission, she developed dysphagia and could not open her left eye. She had weight loss of 30 pounds in three months; generalized weakness, but denied any fever or night sweats. Family history of breast cancer in her mother was recorded. Physical examination revealed an anxious looking female with left ptosis; dysphonia; diplopia; no facial droop; motor and sensation intact; no lymphadenopathy felt; a large hard abdominal mass was found around the epigastric and umbilical areas. Myasthenia gravis was suspected and confirmed by positive edrophonium test and immunologically high titer acetylcholine receptor antibody. Electrophoresis of serum proteins showed a monoclonal increase in gamma globulin rate associated with a monoclonal lambda peak of IgM. Computed tomography (CT) of the chest did not find any mediastinal masses. CT of abdomen and pelvis showed a large lobulated heterogeneously enhancing retroperitoneal mass measuring at least 20 cm. Biopsy of abdominal mass revealed a non-Hodgkin lymphoma classified as follicular lymphoma grade I expressing CD10, CD19, and CD20. The patient responded well to pyridostigmine and prednisone. She was started on cyclophosphamide, doxorubicin, vincristine, dexamethasone and rituximab (CHOP+R). Her clinical status improved and she was continued on chemotherapy and pyridostigmine as outpatient. The immunologic coexistence of acquired myasthenia gravis and non-Hodgkin's lymphoma is rarely recognized. The association of these two diseases may have been the result of an underlying immunological disorder and an immune response may suggest that myasthenia gravis could be a paraneoplastic process. Anti-B cell biological agent such as rituximab is currently a part of the standard regimen (CHOP) for non-Hodgkin's lymphoma and recently being studied for its role in treatment of autoimmune neuromuscular disease. A response to rituximab may consider a potential treatment option for immune-mediated diseases. This case demonstrates the rarity of an autoimmune disease and a malignancy."

PANCREATIC MASS – UNUSUAL PRESENTATION OF LYMPHOMA

Matt Chua, MD

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Primary Pancreatic Lymphoma (PPL) is a rare but potentially curable pancreatic malignancy. Pathological diagnosis of all pancreatic masses is very important, since treatment and prognosis of lymphoma are different from the more common adenocarcinoma. A 48 year-old Hispanic male with no significant past medical history and a negative human immunodeficiency virus (HIV) presented with 3 week history of right upper quadrant (RUQ) and epigastric pain radiating to the back. Patient had developed jaundice, tea colored urine and acholic stools, also reported 15 pound weight loss, fatigue, and generalized pruritus. On physical examination, patient was icteric, had an enlarged liver, tenderness in the epigastrium and RUQ regions. Pertinent laboratory exam showed AST 382 IU/L, ALT 748 IU/L, alkaline phosphatase 525 IU/L, total protein 6.4 gm/dL, albumin 2.4 gm/dl, total bilirubin 9.2 mg/dl, direct bilirubin 6.4 mg/dl, lipase 594 IU/L, amylase 150 U/L. Ultrasonography and computed tomography of abdomen showed a large cystic mass in the head of the pancreas. Magnetic resonance cholangiopancreatography (MRCP) found dilatation of the intra-hepatic and extra-hepatic bile ducts, as well as mild dilatation of the main pancreatic duct. Endoscopic retrograde cholangiopancreatography (ERCP) could not be completed because of an infiltrating mass obliterating the ampulla. Based on these findings, a pancreatic malignant tumor was strongly suspected. Subsequently, patient underwent exploratory laparotomy with double bypass for imminent obstruction, and tissue biopsy. Histological examination revealed diffuse large B-cell non-Hodgkin lymphoma of the pancreatic mass with flow cytometry positive for CD 20 and CD 43. Patient was referred to a cancer institution for combination of chemotherapy and radiation therapy of pancreatic non-Hodgkin lymphoma. Primary pancreatic lymphoma is an extremely rare tumor, representing fewer than 2% of extranodal malignant lymphomas and 0.5% of all pancreatic masses. Diagnosis is established only after histopathology. Treatment usually consists of a combination of chemotherapy and radiation therapy, with 5 year survival rate of more than 50% as compare to less than 5% in pancreatic adenocarcinoma.

PROTEIN-LOSING ENTEROPATHY IN AUTOIMMUNE DISEASE ASSOCIATED WITH A POSITIVE CANCER ANTIGEN

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We report a case of severe hypoalbuminemia associated with protein losing enteropathy (PLE) as a result of systemic lupus erythematosus (SLE). The unusual aspect of this case is the association of SLE with the cancer antigen, CA-125. This marker of malignancy has been found before in lupus and may be a reflection of PLE or the autoimmune process. A 35-year-old previously healthy woman presented with a 2-week history of dyspnea, periobital edema, 20-pound weight gain, and increased abdominal and bilateral lower extremity edema. Her family and gynecologic history were not significant. A review of systems was negative for cutaneous rash, myalgias, Raynaud's phenomenon, dry eyes, diarrhea or urinary symptoms. Physical examination revealed dullness to percussion and decreased breath sounds in the right lower lung field; abdomen was distended, nontender, with a palpable fluid wave and shifting dullness. There was no hepatosplenomegaly or other stigmata of chronic liver disease. There was 4+ pitting edema over the abdomen, pre-sacral area, and lower extremities. Other than striae over the abdomen there were no other skin lesions. Pertinent serology tests showed positive antinuclear antibody (ANA) at 737 U/mL, positive SSA/anti-Ro and SSB/anti-La at 537 U/mL and 737 U/mL, respectively; C3 was low at 76 mg/dL and C4 was normal at 19 mg/dL. Thyroid function tests were within normal limits. Ultrasonography and computed tomography of abdomen revealed massive ascites without any masses or organomegaly. Serum CA-125 was elevated at 2913.7 U/mL. Total serum protein was 4.0gm/dL and albumin was 1.40 gm/dL. Urine, liver and heart test results did not account for the hypoproteinemia. A 24 hour alpha-1-antitrypsin clearance was elevated at 729 mL/24hours which was highly suggestive of protein losing enteropathy. Random biopsies taken from the stomach, duodenum, and colon revealed nonspecific inflammation. The patient was started on 40 mg of prednisone and 400 mg of hydroxychloroquine orally per day with diuretics. A month after discharge, she developed shortness of breath, gained 10-pounds, was readmitted to the hospital; and albumin level was 0.8 g/dl. All drugs were discontinued and the patient was started on azathioprine 100 mg per day. She is now maintained on azathioprine and her last CA-125 was 53.8 U/ml. This patient barely met The American College of Rheumatology's 1996 revised criteria for the classification of SLE. However, the findings of high ANA, SS-A/SS-B, and low complement C3 along with her clinical picture suggest an autoimmune disease like SLE. The elevated CA-125 without evidence of malignancy may reflect inherent bowel pathology which is possibly due to the deposition of immune complexes in the bowel wall. Her clinical improvement with continued immunosuppression and the lowered CA125 levels suggest that this finding is a reflection of underlying autoimmune disease.

RHABDOMYOLYSIS, ACUTE TUBULAR NECROSIS AND DISORDERS OF CALCIUM AND PHOSPHATE METABOLISM: UNUSUAL COMPLICATIONS OF LEGIONNAIRES' DISEASE.

Austine Mengnjo, M.D

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Rhabdomyolysis with myoglobinuria is an uncommon complication of Legionnaires' disease. We describe rhabdomyolysis and acute tubular necrosis following Legionella pneumophila sepsis accompanied by hypocalcemia initially, then hypercalcemia during resolution of rhabdomyolysis. A 54 year-old female, was brought by her brother to emergency room for altered mental status. Her family reported that she has been having fever, chills, cough with greenish sputum, nausea, vomiting and diarrhea for 4 days. On the day of admission, she was found to be very drowsy, responding incoherently, and breathing hard. She had no history of seizures, prolonged immobilization, heavy exercise, alcohol intake, or trauma. She had just returned from Alabama after a six week vacation. On admission, her temperature was 100.8.Fahrenheit, pulse rate of 128 beats per minute, respiratory rate of 20 breaths per minute, and blood pressure of 117/89 mm Hg. Physical examination was notable for tachycardia, tachypnea and crackles at right base. Chest X-ray revealed right middle and lower lobe infiltrates. Pertinent laboratory findings included a white blood cell count of 16,500 cells/ μ L with a left shift, serum sodium of 128mEq/L, potassium of 6.3 mEq/L; phosphate of 11.9 mg/dL; and corrected serum calcium of 6.22 mg/dL. Blood urea nitrogen was 60 mg/dL, creatinine was 10.80 mg/dL and creatine kinase was 12,100 U/L. Serum parathyroid hormone was 136ng/L(10–65 ng/L). Liver function tests were normal. Urine microscopy revealed muddy brown casts consistent with acute tubular necrosis. Urine legionella antigen test was positive. The patient was admitted into the intensive care unit and she was started on intravenous fluids, antibiotics, and hemodialysis. She clinically improved, hemodialysis was discontinued, and she was transferred to regular floor. On 15th hospital day, she developed sudden abdominal pain and confusion. Laboratory examination revealed serum calcium of 16 mg/dL and phosphorus level 8.7 mg/dL. Serum PTH and 1,25 dihydroxyvitamin D levels were suppressed. She was started on isotonic saline, pamidronate and calcitonin with no improvement, eventually she was placed back on hemodialysis. She responded well and subsequently hemodialysis was stopped. The patient was discharged with complete resolution of her metabolic derangement. Rhabdomyolysis with associated acute tubular necrosis is a rare complication of Legionnaires' disease associated with 40% mortality. Alterations in calcium metabolism often complicate rhabdomyolysis-induced acute renal failure. During the recovery phase, serum calcium levels return to normal and may rebound to significantly elevated levels due to the release of calcium from injured muscle, mild secondary hyperparathyroidism from the acute renal failure, and an unexplained increase in calcitriol (1,25-dihydroxyvitamin D). This disorder is generally self-limited and therapy is conservative. As this case illustrates, hypercalcemia in the resolution phase of rhabdomyolysis can be potentially life-threatening and unresponsive to conservative management, thus requiring rapid and aggressive intervention.

SPONTANEOUS RUPTURE OF ESOPHAGUS: A RARE CATASTROPHIC PHENOMENON

Kaushik MD, MPH

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Spontaneous esophageal rupture, also called Boerhaave's syndrome is a rarely encountered event in clinical practice. Barrett ulcer has been implicated as one of the causes of spontaneous perforation of esophagus. We describe a case of spontaneous esophageal rupture in a known case of Barrett esophagus. A 61 year old Caucasian male was brought to the hospital because of vomiting and confusion for one day. Past medical history was significant for severe anemia, Barrett esophagus, and heavy alcohol use. On the day of admission, patient was found to be tachycardic, hypotensive, hypoxic and tachypneic. Physical examination showed decreased air entry on the left side of the chest. A preliminary diagnosis of septic shock was made and intravenous antibiotics were started. Chest X-ray showed bilateral pleural effusion, left more than right. Chest tube was placed in the left side and a brown-gray pleural fluid was drained. Pleural fluid analysis was remarkable for very high lactate dehydrogenase (LDH), leukocytosis, low glucose and high amylase. Rupture of the esophagus was suspected. Computed tomography of the chest showed small posterior, inferior mediastinal air collection adjacent to the esophagus and thickening of the esophageal wall consistent with possible microperforation of the esophagus. Water-soluble esophagogram confirmed the tear of the distal esophagus. Patient was immediately taken to the operating room. Two foci of rupture in the distal esophagus were identified; the largest one being around 2 cm. Distal esophagectomy was performed. Biopsy of the surgical specimen revealed Barrett's esophagus showing extensive intestinal metaplasia. After the surgery, patient improved transiently, but died two months later due to multiorgan failure and sepsis. Boerhaave's syndrome, first described in 1724, is a very uncommon condition making it a challenging diagnosis. A high clinical suspicion is needed in a patient with history of esophagitis or alcohol abuse presenting with vomiting and signs of tachypnea, dyspnea, cyanosis, fever and shock. Early diagnosis and emergent surgery is imperative to ensure favorable prognosis as delayed surgery is associated with a high mortality of 50%. In addition, associated sepsis as seen in our patient further diminishes the chances of recovery.

TWO RARE INTRACRANIAL COMPLICATIONS OF ACUTE BACTERIAL SINUSITIS COEXISTING IN ONE PATIENT

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Subdural empyema and pneumocephalus are infrequent complications of sinusitis. They have been individually described in case reports. However, their simultaneous presence in an adult patient is very rare. We present a patient whose initial diagnosis was acute sinusitis, but further work up revealed both subdural empyema and pneumocephalus. A 33 year old Indian male was brought to the hospital because of altered mental status. Two weeks prior to admission, he complained of runny nose, facial pain and headache. He was prescribed an oral antibiotic by his primary care physician for acute bacterial sinusitis. However, his condition worsened and he developed high grade fever, photophobia and vomiting. On the day of admission, physical examination was remarkable for fever, tachycardia and confusion. Laboratory examination showed leukocytosis. Computed tomography (CT scan) of the head showed pansinusitis with a small pneumocephalus in the left frontal area. Lumbar puncture was performed to rule out meningitis. Cerebrospinal fluid analysis depicted a parameningeal phenomenon with high protein, normal glucose, pleocytosis with high polymorphonuclear cells. Broad spectrum antibiotic coverage was started and an emergent endoscopic sinus surgery was performed. Despite the sinus surgery, the patient's clinical condition remained unchanged. Magnetic resonance imaging of the brain was promptly done that disclosed a collection of fluid in the left subdural space, consistent with the diagnosis of subdural empyema. Neurosurgery was immediately consulted but no surgical intervention was carried out as the size of the collection was relatively small. In the course of the hospital stay, his condition improved. Repeat CT scan of the head demonstrated a decrease in the size of subdural empyema and resolution of pneumocephalus. Sinus fluid culture was positive for *Streptococcus pneumoniae* and antibiotics were appropriately adjusted. Eventually, he was discharged without any neurological sequelae. Presence of both subdural empyema and pneumocephalus in our patient signifies a breach in the sinuses and subdural space. This occurs either through valveless emissary veins or by direct extension via an eroded skull. Subdural empyema is a progressive and potentially fatal condition. It should be treated with broad spectrum antibiotics and neurosurgical drainage should also be considered. It is also important to recognize that pneumocephalus could be an early and/or the only sign of an intracranial involvement of acute bacterial sinusitis.

JERSEY SHORE UNIVERSITY MEDICAL CENTER

ACUTE AORTITIS AND MYCOTIC ANEURYSM COMPLICATING BACTEREMIC HAEMOPHILUS INFLUENZAE PNEUMONIA

Nishant Patel, M.D.

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Introduction: Infection of the aortic wall is rare in the antibiotic era. The vast majority of these few cases involve the abdominal aorta and are associated with *Staphylococcus* sp. or *Salmonella* sp. and present in an indolent fashion. We report a unique patient in whom bacteremic *Haemophilus influenzae* pneumonia lead to acute thoracic aortitis and mycotic aneurysm.

Case Report: A 78 years old woman with well controlled hypertension was admitted for persistent flu-like symptoms, weakness, cough and fever to 102°F. CXR showed an infiltrate and laboratory evaluation revealed WBC 12,600/ μ L with 29% band forms. Blood cultures were obtained; ceftriaxone and azithromycin were administered. *H. influenzae* was isolated and antibiotics were changed to ampicillin-sulbactam. Three days into the admission the patient developed severe, sharp, pleuritic, substernal chest pain that was sudden in onset and radiated to the back and left arm. Chest CT and CT Angiography showed bilateral infiltrates and thickening and irregularity of the entire thoracic aortic wall suggestive of aortitis but no aortic dissection. PET scan was likewise consistent with aortitis. Antibiotics and supportive care were continued. Follow-up CT two weeks later showed dilatation of the distal thoracic aorta (3.8 x 4.5cm). The patient's symptoms resolved and, in consultation with the cardiothoracic service, it was decided to defer surgical intervention. Antibiotics were switched to ceftriaxone 2gm daily and the patient was discharged with close follow-up. One month later the aneurysm had increased in size to 6 x 5.3 cm and the patient underwent urgent Dacron graft placement of the thoracic aorta.

Discussion: The term mycotic aneurysm was coined by Osler in 1885 when he described a mushroom-shaped aneurysm complicating subacute bacterial endocarditis. Such infections develop either when a new aneurysm is produced by infection of the arterial wall (as in our case) or when a preexisting aneurysm becomes secondarily infected. They may develop in any vessel but are very rare in the thoracic aorta. In all, fewer than 10 cases of aortitis or mycotic aneurysm due to *H. influenzae* have been reported in the past 50 years. The case described here also offers a glimpse into the dynamic period between infection and aneurysm formation.

ACUTE HYPOCALCEMIA CAUSING MYOCARDIAL INFARCTION AND LV THROMBUS: AN UNUSUAL AND REVERSIBLE CONDITION

Nishant Patel, M.D.

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Introduction: Chronic hypocalcemia has been reported as a cause of reversible congestive heart failure in both the pediatric and adult populations. Such an association has not been seen with acute hypocalcemia. We report a case of non-ST-elevation myocardial infarction (NSTEMI) and severe 'reversible' myocardial dysfunction with left ventricular thrombus formation in the setting of acute hypocalcemia due to acute surgical hypoparathyroidism. **Case:** An 82-year-old white female was referred to the ER by her primary care physician because a serum calcium was noted to be 5.1mg/dl (8.5-10.5). She had a history of hypertension, dyslipidemia and recently diagnosed follicular thyroid cancer for which she underwent completion thyroidectomy 10 days prior to the presentation. She complained of fatigue and tingling and numbness of both arms and legs, but denied any cramps, chest pain, focal weakness or other complaints. On exam, she had normal vital signs, positive Chvostek's and Trousseau's signs and a benign cardiovascular examination. Serum calcium 5.5mg/dl (8.5-10.5), albumin 3.1g/dl (3.5-5), creatinine 1.3mg/dl (0.6-1.4), phosphorus 7.6mg/dl (2.5-4.6), magnesium 1.9mg/dl (1.3-2.5), and an intact PTH level <1 pg/ml (14-72) with concomitant 25 (OH) vitamin D 18ng/ml (30-80). Electrocardiogram showed normal sinus rhythm with prolonged QT interval (490ms) and no significant ST-T wave changes. She was admitted to ICU and received IV calcium. Over the next few hours, she developed shortness of breath. On exam, raised JVP and bilateral rales were noted. CXR showed mild congestion. Electrocardiogram was unchanged. Laboratory workup revealed BNP-1722pg/ml (0-100), CPK-349IU/L (22-232) and peak troponin-I 10.7ng/ml (<0.78). She was treated with aspirin, carvedilol, lasix and IV heparin. 2D-Echocardiogram revealed akinesis of anterior wall and apex with left ventricle thrombus and ejection fraction of 20%. Left heart catheterization revealed noncritical coronary artery disease. Over the next few days she continued to improve and was discharged in stable condition on coumadin, calcitriol, calcium, sevelamer, carvedilol, aspirin, levothyroxine and valsartan. Follow up echocardiogram after 2 months showed normal left ventricular function and resolution of apical thrombus.

Discussion: Calcium plays a critical role in excitation-contraction coupling and hypocalcemia can lead to depressed LV function and increased myocardial oxygen demands. Hypocalcemia also causes coronary vasospasm, which is likely to be the etiology of NSTEMI in our case. Our case is unique given her age, the lack of typical ST segment changes and the severity of LV dysfunction with thrombus formation, which makes it the first reported case associated with hypocalcemia. Restoration of LV function after correction of hypocalcemia demonstrates that even advanced forms of myocardial dysfunction are potentially reversible.

COMPLICATION OF BAG-MOUTH VENTILATION IN A PATIENT WITH TRACHEOSTOMY DURING PULSELESS ARREST

Wajahat Khn, M.D.

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Introduction: The American Heart Association, in its publication on Advanced Cardiovascular Life Support (ACLS), provides a structured algorithm for the approach to pulseless cardiac arrest. We report a case of Pulseless Electrical Activity (PEA) due to tension pneumothorax further complicated by adherence to ACLS protocol.

Case report: A 76 year old female with a history of COPD, CHF, recurrent pneumonias and multiple intubations secondary to respiratory failure presented with dyspnea. The patient was treated for pneumonia, but developed respiratory failure requiring endotracheal intubation and mechanical ventilation. Day #14 a tracheostomy was placed. On Day #19, after the trach was suctioned, she developed spontaneous subcutaneous emphysema in the neck with rapid progression to face, shoulders, and chest. She became bradycardic and pulseless, ACLS protocol was initiated and bag-mouth ventilation was started because it was suspected that the tracheostomy may have contributed to the subcutaneous emphysema. There was substantial airflow resistance to ventilation. The subcutaneous emphysema became massive and inflated the patient's entire face, neck, and chest wall, where the skin was elevated several centimeters. A decompression needle was inserted on the right side, eventually followed by a right sided chest tube. During the course of the code her rhythm changed from PEA to ventricular fibrillation, and eventually asystole. After 15 minutes, life support was discontinued at the request of the family. Approximately five minutes later, an organized rhythm was noted on the defibrillator leads. Palpation of the carotid pulse confirmed return to spontaneous circulation. A stat chest X-ray showed bilateral pneumothoraces. A chest tube was placed on the left side. Although the patient was stabilized, she expired several hours later. **Discussion:** ACLS algorithms provide a structured approach to a pulseless arrest. However, once PEA was identified, the subcutaneous air should have moved tension pneumothorax to the top of the differential, and the bag ventilation should not have been so aggressive. The needle thoracostomy was placed quickly, but the typically aggressive ventilation (to address the possibility that PEA was due to acidosis) limited its efficacy. Until life support was terminated and the air had 5 minutes to escape, the massive subcutaneous emphysema prevented the leads from detecting her rhythm and her pulse from being palpated. This case illustrates a situation where adherence to ACLS protocol, specifically aggressive bag-mouth ventilation, interfered with the ability to assess an unstable patient. The difficult point is that even in a dire emergency we need to take time to examine the patient in order to prioritize our differential diagnoses and before we initiate a clinical algorithm.

DEVASTATING CONSEQUENCES OF NOT PERFORMING UNIVERSAL HIV TESTING

Ditina Ghetia, M.D.

Co-Authors: Anil Aleti MD, Elliot Frank MD, and Nancy Gornish MD

Introduction: With the advent of HAART, opportunistic infections are seen less commonly in patients without obvious immunocompromise. We recently managed a patient in whom Cryptococcal meningoencephalitis was the presenting and terminal event for a previously healthy man without known HIV risk factors. **Case Presentation:** A 63 year old previously healthy married man was brought to the emergency room for evaluation of a three week history of persistent headache, nausea, vomiting, and intermittent hearing loss as well as progressive altered mental status for two days prior to admission. He denied photophobia or neck rigidity. CT, MRI and MRA of the head performed two weeks earlier had been negative. There was no history of intravenous drug use, transfusion or high risk sexual activity. Physical exam was normal. A lumbar puncture yielded clear fluid with 1 WBC, no RBCs, a glucose of 26 mg/dL and a protein of 121mg/dL. Encapsulated yeast was identified on an India Ink preparation and the CSF Cryptococcal antigen was positive with a titer 1:256. Antibodies to HIV were detected and the CD4 count was 14cells/μL. He was started on Amphotericin B and gradually became more alert and complained less of a headache. On hospital day three, patient was found to have increasing lethargy. CT scan of the head was negative. Therapeutic lumbar puncture was attempted but unsuccessful. Within hours, the patient's pupils became fixed and dilated, repeat CT scan revealed global cerebral edema and MRV confirmed complete thrombosis of the sagittal and transverse sinuses. The patient was pronounced dead several hours later. **Discussion:** This case emphasizes the fact that a significant reservoir of undiagnosed HIV infection still exists in the United States and the changing epidemiology of the HIV epidemic to an older demographic, two factors that reinforce universal HIV screening initiatives.

EPSTEIN-BARR VIRUS TESTING: ANTIBODY VS. MONOSPOT AT JSUMC: CLEAR ROOM FOR IMPROVEMENT AND COST SAVING

Purviben Shah, M.D.

Co-Authors: Michael P. Carson, MD and Elliot Frank, MD

Background: The Monospot (Heterophile Agglutination assay or Infectious Mono (IM) Slide Test) will detect the heterophile antibody (HA) in 85-90% of acute IM cases with a specificity of 98-100%. Other Epstein Barr antibody panels have longer turn around times and may not offer additional diagnostic information. We will report which EBV assays were ordered, and at what cost.

Methods: A review of Test Ordering Pattern reports provided to Jersey Shore University Medical Center by our reference lab for the period February 2003-January 2004, inclusive; and 2007 data from tests ordered through our lab. No patient identifiers were provided. **Results:** (Test/number sent to the reference lab/Cost per assay/Total Cost) 2004 Monospot: 2/\$11/\$22. 2004 EBV Antibody Panel II: 749/\$27/\$20,223. In 2007 there were 522 Monospots ordered, and 490 antibody panels. The difference in the assay costs is \$16. If Monospots were ordered instead of the EBV Antibody panel the cost savings would have been \$11,864 in 2004, and \$7,480 in 2007. **Conclusion:** As the Monospot is an "in house" test, the two sent out in 2004 were likely an error. The turn around time for Monospot is 1 hour when ordered STAT and 24 hours for the EBV antibody panel, therefore changing this practice would provide more efficient care as well. While fewer EBV panels were sent out in 2007 vs. 2004, the potential exists to modify test-ordering patterns to continue to decrease use of non-heterophile tests when IM is suspected. The potential cost savings for the institution can best be realized for tests ordered on inpatients. This preliminary analysis of data for a quality assurance project will be followed by a chart review to determine the proportion of tests performed on inpatients, and the number of patients for whom both tests were ordered, after which a program could be developed to educate the medical/pediatric staff.

EXTENSIVE PULMONARY EMBOLI PRESENTING AS SHORTNESS OF BREATH FOR TWO MONTHS IN A HEALTHY YOUNG MALE

Wajahat Khan, M.D.

Co-Authors: Neena Penagaluru, MD., Dana Tarina, MD., and Julianne Pupa

Background: Pulmonary Embolism (PE) accounts for up to three-hundred-thousand hospital admissions each year, and is the third leading cause of death in the United States. Classically, patients present with shortness of breath, chest pain, and tachycardia. However, symptoms can vary greatly and making a diagnosis can be exceedingly difficult. Autopsy literature reveals approximately 60% of patients who died in a hospital setting had a PE; and the diagnosis had been missed in up to 70% of cases.

Case Presentation: A 42 year old male with no significant medical history presented with shortness of breath (SOB) for approximately 2 months. He had previously been extensively evaluated for SOB at another facility, and the patient was being treated for a presumptive fungal pulmonary infection. Prior diagnostic studies included a chest x-ray which revealed bilateral infiltrates and a non-contrast CT examination of the chest which confirmed the x-ray findings. Previous work-up also included a bronchoscopy, bronchoalveolar lavage, and numerous blood tests, including HIV serology – all of which were unrevealing. On the current admission, the patient was not in respiratory distress. His vitals were stable and oxygen saturation was 95% on room air. A chest x-ray revealed a right sided pleural effusion and right sided infiltrates. The left lung appeared clear. A contrast enhanced CT of the chest revealed pulmonary emboli extending from the main pulmonary arteries to the lower lobes bilaterally, with extensive involvement of the left upper lobe arteries. Treatment was initiated with intravenous heparin and the patient was subsequently discharged on oral warfarin.

Discussion: Pulmonary embolism is often a fatal disease process. Making a diagnosis can be exceedingly difficult. This unique case illustrates the diversity with which PE can present. It also demonstrates the variability of radiographic findings and emphasizes that symptoms and physical exam findings can be unreliable.

**FREON GAS INHALATION LEADING TO CHEMICAL PNEUMONITIS BACKGROUND:
CASES OF CARDIAC ARRHYTHMIAS HAVE BEEN REPORTED FROM FREON GAS EXPOSURE BUT INHALATION
OF FREON GAS LEADING TO CHEMICAL PNEUMONITIS HAS NOT BEEN REPORTED**

Swaleha Mahpara, M.D.

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CASE: A 45 y/o male with a past medical history of coronary artery disease and diabetes mellitus presented after inadvertent inhalation of Freon gas while servicing his freezer. He complained of lightheadedness, shortness of breath, wheezing, chest tightness and cough productive of yellow sputum. Lung examination revealed coarse crackles bilaterally. His oxygen saturation was 86% on room air with normal blood pressure, pulse and temperature. Chest X-ray showed left lower lobe opacity. CT Chest showed extensive airspace disease in the lower lobes and a ground glass pattern in the upper lobes. The patient was admitted to telemetry for close monitoring of arrhythmias. Intravenous Solumedrol 60 mg every 8 hours was started for possible chemical pneumonitis. Clinical improvement was seen on day # 3. His oxygen saturation gradually improved and once ambulatory oxygen saturation was stable around 95%, he was discharged on oral prednisone. Follow up in a month showed almost complete resolution of his symptoms. **DISCUSSION:** Freon gas is one of several chlorofluorocarbons which were considered non-toxic but are now considered a danger to the ozone layer. Freon exposure in large quantities from air-conditioners has been known to cause arrhythmias in predisposed patients and asphyxiation if oxygen levels are low. Our case is unique in that our patient developed chemical pneumonitis with severe airway damage which responded to steroid treatment.

FULMINANT SEPTIC SHOCK SECONDRY TO COMMUNITY ACQUIRED MRSA (CA-MRSA)

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Background: Although predominantly associated with skin and soft tissue infection, CA-MRSA is not well recognized as a cause of community acquired sepsis. We report a case of a healthy adult with overwhelming sepsis during Influenza season.

Case: A 50 year-old male was brought to emergency room with fever of 104^oF, lethargy, nausea and vomiting preceded by a few days of flu-like illness. Because of neck pain, stiffness and a change in mental status, lumbar puncture was performed. He was started empirically on Ceftriaxone, Vancomycin, and Acyclovir. CSF analysis did not show any evidence of meningitis. Laboratory studies included a WBC of 9,000/mm³ with 36 bands and troponin of 6.55. The chest x-ray, EKG and 2D ECHO were normal. Eight hours after admission, the patient deteriorated with an interval development of pulmonary infiltrates. By twelve hours he progressed to a pulseless ventricular tachycardia and ultimately expired after multiple futile resuscitative attempts. Autopsy results revealed necrotizing pneumonia and multiple microabscesses in his brain and heart. MRSA grew in blood cultures and from autopsy specimens. Isolates were confirmed to be Pantone-Valentine Leukocidin positive, USA 300 strain consistent with CA-MRSA.

Discussion: CA-MRSA with PVL gene expression is highly virulent and can be associated with fulminant sepsis and end organ damage despite appropriate antibiotics. Cardiac microabscesses damaged the myocardium as reflected in positive cardiac enzymes and resulted in fatal arrhythmia in the absence of hypoxia. This fulminant sepsis could have been preceded by Influenza, unfortunately it was not confirmed prior to patient's demise.

GOODPASTURE'S DISEASE COMPLICATED BY MULTIPLE CAVITARY PULMONARY LESIONS: A RARE MALADY

Syed Nabil Babar, M.D.

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Introduction: Goodpasture's disease is a rare autoimmune hypersensitivity type 2 reaction, characterized by deposition of antibodies against the glomerular basement membrane resulting in glomerulonephritis and often leading to renal failure. **Case report:** A 62 year old male was admitted with fever and chills for 10 days associated with darkening of the urine. He denied chest pain, nausea, vomiting, rash, diarrhea or dysuria. Past medical history was significant for chronic interstitial lung disease. Vital signs were stable except for a temperature of 100°F and physical exam was unremarkable. Labs revealed BUN 113mg/dL and Creatinine 8.6 mg/dL and high titres of anti-GBM IgG antibody were detected. The patient was started on hemodialysis, steroids and plasmapheresis. Renal biopsy disclosed diffuse necrotizing segmental glomerulonephritis consistent with anti-GBM nephritis, following which cyclophosphamide was initiated. The patient was discharged on thrice weekly dialysis. Two months later, he presented with SOB and chest pain. He was found to be pancytopenic. Multiple cavitary mass lesions and consolidations involving both lungs were seen on Chest CT. He developed hemoptysis and underwent bronchoscopy which showed a alveolar hemorrhage. Cultures of blood and bronchial aspirate grew *Pseudomonas aeruginosa*. Cardiac arrest characterized by pulseless electrical activity and associated with a massive left sided pneumothorax ensued. Resuscitation was successful but the prognosis was deemed dismal. Comfort care was provided and he passed away soon thereafter.

Discussion: Goodpasture's disease represents 1-2% of all cases of RPGN in the US and the incidence in Europe is 1 in 2 million. It occurs primarily between ages 20 to 30 and again between 50 to 65 years. 60 to 80% of patients present with renal and pulmonary involvement while fewer than 10% have lung disease alone. Risk factors include cigarette smoking, exposure to inhaled hydrocarbons, viral infections and genetic predisposition. Serological workup with anti-GBM antibodies may be useful but kidney biopsy is often the fastest way to secure the diagnosis and gain information about the extent of the disease and likely effect of treatment, which involves plasmapheresis to remove the circulating antibodies and immunosuppression to stop their further production. Prognosis correlates with the degree of renal involvement at the time of presentation.

Conclusion: A high degree of suspicion and an aggressive diagnostic approach is necessary to allow early treatment of Goodpasture's syndrome since diagnosis is often delayed due to the vagueness and swift progression of the disease.

GUIDELINES FOR SURGERY IN ASYMPTOMATIC PRIMARY HYPERPARATHYROIDISM: TIME FOR AN OVERHAUL?

Dayanand Makey, M.D.

Co-Authors: Taral Jobanputra, MD, Adrian Scaunasu, MD, Muhammad S Huq, MD, Alexander Shifrin, MD, and Sunil Asnani, M.D.

Introduction: We present a case of successful, uncomplicated parathyroidectomy of a right inferior parathyroid adenoma in an 82 year old with primary hyperparathyroidism (PHPT) with clinical improvement and suggest that improving surgical technique may warrant deemphasizing age in assessing surgical appropriateness in PHPT. **Case Report:** An 82 year old female with a history of hypertension, gout, nephrolithiasis, chronic kidney disease-stage IV, anemia, osteoporosis, and a recent history of recurrent deep vein thrombosis with 1 episode of pulmonary embolism was evaluated for hypercalcemia. Serum calcium was 10.1 mg/dl (8.5-10.5), serum creatinine was 1.5 mg/dl (0.8-1.4), and intact parathyroid hormone (PTH) level was 159 pg/ml (10-60). She was not on calcium or vitamin D replacement. A parathyroid sestamibi scan showed accumulation in the right inferior parathyroid gland. A diagnosis of PHPT was made. In view of her deteriorating renal function and history of nephrolithiasis, minimally invasive parathyroidectomy was undertaken. Intra-operative PTH level was monitored and was decreased by >50% decrease suggesting curative excision. Pathological diagnosis of the excised tissue was a benign adenoma of the parathyroid gland with cystic components. The patient tolerated the procedure well, with no peri-operative complications, and was sent home the same day. No post-op paresthesias or change in voice was reported; calcium supplementation was not required. At the last follow-up, 4 months after surgery, patient remained eucalcemic.

Discussion: Patients over 80 years were once cast as unacceptable candidates for surgical intervention in a number of diseases. The natural course of PHPT is one of exacerbation and progression of symptoms; therefore, delaying, deferring or rationalizing against surgery will worsen the symptom complex due to PHPT. With the advent of minimally invasive parathyroidectomy, even the oldest adults are candidates for definitive treatment of PHPT. Smaller incision, miniscule blood loss, shorter operative time (10-20 min), avoidance of endotracheal intubation, limited pain and limited hospital stay all support cautious but aggressive approach in elderly patients with PHPT. The 2002 consensus guidelines from NIH suggest age < 50 year as a surgical criterion. Clearly, it is time to revisit these guidelines.

H. PYLORI ANTIBODY ORDERING PATTERNS: OPPORTUNITY TO IMPROVE PRACTICE PATTERNS

Sakshi Pawa, M.D.

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Background: The utility of the IgM assay to diagnose *Helicobacter pylori* infection has not been established, and IgA is only present for the first 60 days after an acute infection. When ordering blood tests, serum IgG is the best test to assist clinicians in establishing a diagnosis of this infection in the proper patients. Clinicians should order tests on inpatients when the results can dictate a change to the short term plan, be reliable in that clinical setting, be cost effective, and be in line with clinical guidelines. This project will determine the frequency at which serological tests for *H. pylori* were ordered and at what cost. **Methods:** A review of Test Ordering Pattern reports provided to our laboratory director by our reference lab for the period February 2003-January 2004, inclusive. No patient identifiers were provided. **Results:** (Test/number/cost per assay/total cost) IgA/120/\$19.50/\$2,340. IgM/159/\$19.50/\$3,100. IgG/6/\$19.50/\$117. **Conclusion:** Assuming no patient had more than one test, it seems the opportunity exists to shift clinicians away from IgA and IgM testing to the recommended IgG. This will not decrease costs, but could lead to testing in line with current data. A query of the Data Warehouse and chart review are necessary to determine the proportion of tests performed on inpatients and the best approach to educate the medical staff.

HENOCH SCHOLEIN PURPURA MIMICKING SEPSIS AND MULTIORGAN FAILURE

Vandana Palan, M.D.

Co-Authors: Kathleen Casey, MD, Sayed Nabil, MD, Nishant Patel MD, and Sushil Mehandru, MD

Introduction: Henoch Schlonlein Purpura (HSP) is a systemic vasculitis of small vessels characterized by palpable purpura, arthritis, bowel angina and renal failure. HSP is relatively common in children (15 cases/100,000) but very rare in adults (15 cases/ 1,000,000). We recently encountered a patient who presented with what appeared to be intra-abdominal sepsis and multi-organ failure in whom a diagnosis of HSP was later confirmed. Case presentation: A 78 year old man with hypertension, diabetes, coronary artery disease and rheumatoid arthritis presented with epigastric abdominal pain radiating to his back, jaundice, fever, acute renal failure and progressive hypotension. A week after his presentation, he developed a purpuric rash from scalp to feet. Many lesions had necrotic centers. The hemoglobin was 10.7g/dl, WBC 7.9K/uL, platelets 192K/uL, ESR 60mm/hr, creatinine 3.3 mg/dl, blood urea nitrogen 47mg/dl, alkaline phosphatase 404 IU/l, AST 1036 IU/l, ALT 1022 IU/l, direct bilirubin 2.8 mg/dl, amylase 400 U/l, lipase 416 U/l, GGT 60 IU/l. Urine analysis showed large blood, granular casts and > 300mg/dl protein. Blood and urine cultures were negative. Serology for hepatitis A, B and C, EBV, CMV and HSV; ANA, SSA, SSB, RNP, C3, C4, antiphospholipid antibodies and immunoglobulin G, M, and A were negative or normal. Abdominal ultrasound, HIDA scan and MRI revealed no evidence of cholecystitis or biliary obstruction. CT showed thickening of the cecum with stranding consistent with cecal inflammatory disease. A colonoscopy revealed edema of the ascending colon and cecum, possibly secondary to ischemia. An endoscopy revealed multiple superficial ulcers in the stomach which were negative for IgA on immunoflorescent stains. The patient's general condition gradually improved with resolution of rash and abdominal pain and normalization of LFTs. However renal function deteriorated requiring dialysis. A renal biopsy demonstrated focal endocapillary proliferative glomerulonephritis with IgA immunoflorescence consistent with HSP. Plasmapheresis was begun but he continued to require hemodialysis. The patient died of an unrelated medical condition on day ten of plasmapheresis.

Discussion: The exact pathophysiology of HSP is poorly understood but it is thought that abnormal glycosylation of oligosaccharides leads to deposition of immune complexes composed predominantly of IgA1 in the vessel wall. These immune complexes recruit polymorphonuclear leukocytes and cause leukocytoclastic vasculitis. Our patient's presentation initially suggested intra-abdominal sepsis however no inciting infectious process was identified. It is rare for HSP to present with multiple organ failure and sepsis syndrome. It was only after that the rash appeared and the renal failure persisted that HSP was suspected and confirmed with renal biopsy.

HIGH ON ROLAIDS- MODERN MILK-ALKALI SYNDROME

Dayanand Makey, M.D.

Co-Authors: Wajahat Khan, MD, Ravikanth Vidyula, MD, Neena Penagaluru, MD, Kathleen Casey, MD, and Sunil Asnani

Introduction: Fewer than 50 cases of milk-alkali syndrome have been reported in the last 15 years since the advent of newer anti-ulcer therapies. We report a patient with obsessive compulsive disorder (OCD) in whom milk-alkali syndrome developed.

Case Report: A 39-year-old Caucasian man, with OCD and HIV presented to the ER with syncope. He reported poor appetite, weight loss, fatigue, epigastric discomfort and diarrhea due to anxiety, and performing repetitive actions due to his OCD. Careful elicitation of the medication history revealed that he had ingested Extra-Strength Multi-Symptom Rolaids, amounting to 16 gm of calcium carbonate per day, for the past few weeks. Examination was unremarkable. Admitting labs revealed: Calcium 16.6 mg/dl (8.5-10.5), phosphorus 2.0 mg/dl (2.5-4.5), CO₂ 43 mmol/l (24-31), BUN 50 mg/dl (5-25), creatinine 3.8 mg/dl (.5-1.4), magnesium 3.5 mg/dl (1.3-2.5), potassium 3.1 mmol/l (3.5-5.3), chloride 88 mmol/l (96-110), and albumin 4.5 g/dl (3.5-5). Absolute CD4 count and HIV, 3 months earlier, were 550 cells/ μ l and 510 copies/ml, respectively. Chest X-ray and CT of the head were normal. Normal saline was infused at 200 cc per hour. PTH was 2.7pg/ml (14-72); PTH-rP <1.5pmol/l (0-4); 25-OH-vitamin D 30ng/ml (30-80); 1,25-(OH)₂-vitamin D 28pg/ml (15-75); and serum protein electrophoresis (SPEP) was normal. He was placed on pantoprazole 40mg twice daily. Renal function gradually improved and one week after stopping the antacids, his creatinine was 1.2mg/dl, CO₂ 24 mmol/l, and corrected calcium 8.6mg/dl. He was advised against taking calcium containing antacids and to follow-up with his psychiatrist for control of anxiety and OCD.

Discussion: The modern milk-alkali syndrome, though rare, is seen in patients who over medicate for dyspepsia or osteoporosis and is characterized by hypercalcemia, hypermagnesemia, and metabolic alkalosis. Hypokalemia and hypophosphatemia occur despite acute renal insufficiency. In our case it is highly likely that OCD contributed to the ingestion of excess calcium. Restoration of normal renal function depends on the duration of hypercalcemia with acute cases having a better prognosis. Inability to suppress calcitriol and impaired calcium excretion increase susceptibility to the development of this syndrome and likely play a pathophysiological role.

HYPOGLYCEMIA IN PREGNANCY TWO YEARS AFTER GASTRIC BYPASS SURGERY

Vivienne Policarpio, M.D.

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Introduction: Insulinoma is the most common cause of hyperinsulinemic hypoglycemia in adults. Persistent hyperinsulinemic hypoglycemia due to nesidioblastosis is a well-recognized but infrequent disorder in infancy; however adult-onset nesidioblastosis in the form of noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS) is an entity even more rare. Only one other case of symptomatic hypoglycemia complicating pregnancy following gastric bypass surgery has been previously documented. Case Report: A 33-year-old Caucasian female was evaluated for dizziness which began during the eighth week of pregnancy. Two years earlier she had undergone gastric bypass surgery for obesity. One year later she began to have episodic lightheadedness for which she did not seek medical attention and, one year after that, dizziness occurred daily and was associated with palpitations, shortness of breath, diaphoresis, and occasionally tremors and confusion. Episodes occurred many times a day, usually approximately 30 minutes after eating a meal, and especially with food rich in carbohydrates. Symptoms were transiently relieved by food intake so that patient had significant weight gain during her pregnancy. She started to monitor her blood sugar. During one episode, blood sugar was 61 mg/dL. 30 minutes after food intake, repeat blood sugar was 165 but subsequently dropped to 51. Patient was advised small frequent meals low in carbohydrates. Hypoglycemic episodes were to be treated with a small amount of carbohydrates to be followed by food richer in protein and fat to prevent recurrent hypoglycemia. The patient returned to the office and labs were obtained during an episode: When blood sugar was 50, cortisol was 18.3 ug/dL, growth hormone 2.5 ng/mL, insulin 6.6 uIU/mL, and C-peptide 2.7 ng/mL,. Both insulin and C-peptide were deemed abnormally elevated in the face of hypoglycemia when levels should have been suppressed. Screening for sulfonylureas was not done as the medication history was felt to be reliable. Further evaluation was limited due to the patient's pregnant state. 72-hour fast, imaging studies, and selective arterial calcium stimulation test were not done to prevent harm to the fetus. Such diagnostic work-up is planned after delivery. However, in light of the patient's symptoms and available results, patient's postprandial hypoglycemia is most likely due to noninsulinoma nonpancreatogenous hypoglycemia. Discussion: Functional hyperinsulinism or reactive hypoglycemia may cause neuroglycopenic symptoms compatible with low blood glucose levels that occur after meals. Several causes of true postprandial hypoglycemia include ackee fruit poisoning, factitious hypoglycemia, galactosemia, gin and tonic hypoglycemia, hereditary fructose intolerance, insulin autoimmune hypoglycemia, insulinoma, pancreatic transplantation, and noninsulin pancreatogenous hypoglycemia syndrome. Patients with NIPHS experience postprandial hypoglycemia and have nesidioblastosis with islet cell hypertrophy likely due to the extreme resistance of pancreatic beta cells to insulin present in morbidly obese patients.

INCREASED RISK OF DEEP VENOUS THROMBOSIS WITH ULCERATIVE COLITIS: A CASE FOR VIGILANCE

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Introduction: We present a case of occult lower extremity deep vein thrombosis (DVT) fortuitously diagnosed in a patient with ulcerative colitis (UC). **Case:** A female UC patient in her 30's, on an oral contraceptive for 10 years, presented with increased stool frequency and urgency, bloody bowel movements, weight loss and weakness. Colonoscopy 3-years before showed ulcerative pancolitis. **EXAM:** Afebrile. Abdomen: normal bowel sounds, nondistended, soft, mild left lower quadrant tenderness, rebound absent. Hemoglobin 7.9 gm/dL, MCV 83 microL3, RDW 20.8%. She was admitted, the contraceptive was stopped, intravenous fluids and corticosteroids were initiated. Stool studies for bacteria, parasites and Clostridium difficile toxin were negative. Hospital day #2: colonoscopy showed pancolitis. Hospital day #4: the attending physician ordered a D-dimer and fibrinogen as part of an evaluation to exclude additional causes of the anemia. A covering resident was called with the D-dimer result of 2284 ng/ml (normal <230). The bedside evaluation revealed no symptoms or findings suggestive of DVT, but a Doppler/ultrasound was ordered. Hospital day #5: the ultrasound revealed extensive left leg DVT from the common femoral to the popliteal vein and incompletely occlusive right DVT in the common femoral vein. Heparin was begun. On hospital day #6 she had a syncopal event and was hypotensive due to a severe GI bleed. She responded to resuscitation with IV fluids and 2 units PRBC, stabilized and eventually had an IVC filter placed. Hypercoagulable work up was negative. Her UC responded to the steroids and she was discharged on oral prednisone and iron.

Discussion: DVT and pulmonary embolism (PE) are infrequent, but significant extraintestinal complications of inflammatory bowel disease. The incidence of DVT in a 1964 case series of 624 UC patients was 4.6%, the 10 PE's occurred during acute disease flares, and the rate was not different during the pre- and post-steroid eras. However a recent population based study found the incidence of DVT with UC to be 30/10,000 person-years and 20/10,000 person-years for PE. While the risk was increased compared to the non-UC population, it is lower than the 1964 estimate. The prevalences of the Prothrombin gene and Factor V Leiden mutations were not increased in UC patients with DVT. It is postulated that the systemic inflammatory response contributes to the risk of vascular complications, including arterial. This case demonstrates the need to suspect this extraintestinal complication and how it came to be diagnosed in the absence of symptoms or exam findings. Rational use of testing is important, but so is a high clinical suspicion for a notoriously subtle disease.

INPATIENT CRP TESTING ADDS LITTLE CLINICAL VALUE

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Background: Serum C-reactive protein (CRP) is a non-specific test associated with inflammation, cancer, and cardiac events (exactly when is not clear), but the clinical utility outside of rheumatology is uncertain. Generally, tests obtained on inpatients should dictate a change to the short term plan and be in line with clinical guidelines. A quality assurance project revealed that 2980 CRP assays were ordered through our lab in 2007; each cost \$26 (assay and technician time) for a total expenditure of \$77,480. Since virtually all hospital reimbursement is based on case rates or daily rates, such testing represents pure cost. This preliminary analysis will identify the diagnoses most often associated with inpatient CRP testing and compare the length of stay (LOS) and hospital charges for those who had CRP tests obtained (cases) to a cohort of patients with similar diagnoses who did not (controls). Methods: Our data warehouse was queried to identify adult inpatients that had CRP assays in 2007 (cases) and they were compared to all adult inpatient admissions 2006-2007 with similar admitting diagnosis codes (controls). Common disease codes associated with the test were identified, and within these groups mean LOS and mean hospital charges were determined. As LOS and charges were not normally distributed, the Wilcoxon Rank-Sum (Mann-Whitney) test was used to compare the mean values.

Results: 2,980 CRP assays were ordered; 1070 of these were in patients >18 years of age. 13% had "unspecified chest pain", 50% were in men. The mean LOS was longer for abdominal pain cases (8.6+7.5 days, n=49) than controls (5.8+5.70 days, n=602) $p=0.007$; for chest pain cases (5.9+17, n=145) vs. controls (3.5+3.6, n=2137), $p<0.0003$; for the total case cohort (7.9+11.4, n=1070) vs. control cohort (4.8+5.4, n=23,844), $p<0.0001$, but not for those admitted with "pneumonia" or "fever". A left heart cath was performed on 28% of chest pain cases (n=41, LOS 5.4 days) vs 20% (n=429 LOS 4.3 days), but LOS was not significantly longer. The LOS for all inpatients who had a cath, regardless of diagnosis, was 4.2 days suggesting a difference between the entire cohort and our chest pain patients. Mean total charges were significantly increased only in the chest pain group ($p=0.0006$).

Conclusion: CRP is ordered frequently at considerable cost and was not associated with a difference in LOS for pneumonia and fever patients suggesting that it did not offer any assistance to the treating physician. Although CRP testing was associated with increased LOS for patients with abdominal pain and chest pain it is doubtful that this information altered management in these groups either. The increased LOS and charges for chest pain patients was not completely explained by having a cardiac cath. A chart review is necessary to identify clinical characteristics or confounders such as abnormal test results that could account for the increased LOS and costs for chest and abdominal pain patients, and whether CRP levels in any way influenced management decisions.

IS THERE A ROLE FOR STEROIDS IN GROUP B STREPTOCOCCAL MENINGITIS?

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Introduction: Group B streptococcal (GBS) meningitis occurs in 2-4% of invasive GBS bacteremia in adults. Current guidelines recommend the use of adjunctive dexamethasone in adults with meningitis only when *Streptococcus pneumoniae* is the suspected pathogen. We report our observations in two adults with GBS meningitis, one who received steroids and one who did not. Case 1: A 57-year old previously healthy woman presented in May 2008 with altered mental status associated with vague abdominal pain, low grade fevers and headache for one week. Examination was remarkable only for lethargy and a cluster of petechial lesions on the medial aspect of her left arm. LABS: WBC 5,600/ μ L, platelets 62,000 / μ L AST 169 IU/L, ALT 167 IU/L. CSF analysis: glucose 5 mg/dL, total protein 760 mg/dL, WBC 92/ μ L (neutrophils 65%, bands 29%, lymphocytes 4%, monocytes 2%), RBC 201/ μ L, Gram stain-negative. CXR and CT of the head and abdomen were negative. Ceftriaxone, vancomycin, ampicillin and doxycycline were administered. CSF and blood cultures grew GBS within 12 hours. Progressive cerebral edema led to herniation and death within 48 hours. Case 2: A 77- year old woman with history of pneumococcal meningitis, chronic otitis media, hypertension and penicillin allergy presented with fever, chills, headache, earache, photophobia, nausea and vomiting. She was lethargic and had nuchal rigidity. LABS: WBC 19,600 / μ L. CSF analysis: glucose 7 mg/dL, white cell count 1,750 / μ L (neutrophils 96%, bands 1%, lymphocytes 2%, monocytes 2%), protein 447 mg/dL, Gram stain negative. CT showed opacification of the right middle ear and mastoid air cells. Treatment was initiated with trimethoprim-sulfamethoxazole, chloramphenicol and vancomycin. Given the high suspicion of pneumococcal meningitis, she also received dexamethasone at the initiation of antibiotic therapy. Blood cultures grew GBS. Vancomycin was continued and other antibiotics were discontinued. The patient completely recovered without neurological sequelae.

Discussion: Current guidelines advocate the use of adjunctive steroids only for meningitis due to *S. pneumoniae*. Our experience with these two cases certainly invites speculation about the potential utility of corticosteroids in adults with meningitis caused by other pathogens, especially GBS.

LIFE THREATENING COMPLICATION OF LOW FAT DIET: PRESCRIBERS BEWARE

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Introduction: Vitamin D deficiency is one of the most common mineral/vitamin deficiencies worldwide. For patients with multiple co-morbid conditions, dietary manipulation may have profound implications.. We present an interesting case of severe life threatening metabolic disturbances precipitated by the initiation of a low fat diet after the diagnosis of coronary artery disease. Case: A 64-year-old man presented to the ER with generalized weakness. He had been feeling progressively unwell for about 2-3 days to the point that he was unable to stand up. He reported tingling and numbness in his feet but denied any spasms, diarrhea, or constipation. His past history included type 2 DM and colon cancer with a right hemicolectomy 4 years ago. Two months earlier coronary artery disease was diagnosed at which time a stent was deployed and he began a low cholesterol, low fat diet. The examination was normal except for a positive Chvostek's sign. His initial labs revealed: serum calcium 5.5mg/dl (8.5-10.5), albumin 2.9gm/dl (3.5-5), creatinine 1.0mg/dl (0.5-1.4), phosphorous 1.9mg/dl (2.5-4.6) and magnesium 0.9mg/dl (1.3-2.5). Immediately prior to his new diet regimen, calcium levels had been consistently between 7.8-8.1mg/dl (8.5-10.5). Treatment with IV magnesium sulfate and IV calcium gluconate was initiated in the ER followed by oral replacement. Vitamin D (25-hydroxy) was <4ng/ml (30-80) and PTH was elevated to 329pg/ml (14-72). All symptoms quickly resolved and he was discharged on vitamin D 50,000IU once a week, calcium carbonate 750mg twice a day and magnesium oxide 400mg twice daily. He was also counseled regarding the importance of moderation in diet and required nutrition supplements. Discussion: Vitamin D is found in many dietary sources including fish, egg yolk, fortified milk, red meat and cod liver oil. The sun also contributes significantly to the daily production of vitamin D, and studies suggest that Vitamin D levels are lowest at the end of winter. Our patient likely had longstanding, borderline vitamin D deficiency which was exacerbated by a "heart-healthy" diet that virtually eliminated vitamin D rich foods from his diet. It is thus critical to identify 'at risk' populations before making dietary recommendations to avoid precipitating such life threatening events. Nutritional consultation and, in some cases, dietary supplementation may decrease the likelihood of complications.

MARANTIC ENDOCARDITIS OF A PROSTHETIC MITRAL VALVE IN A PATIENT WITH PRIMARY ANTIPHOSPHOLIPID SYNDROME

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Introduction: Marantic endocarditis, or non-bacterial thrombotic endocarditis, is most commonly seen on native cardiac valves in patients with malignancy, systemic lupus erythematosus (SLE), and hypercoagulable states such as antiphospholipid syndrome (APS). Marantic endocarditis involving prosthetic valves has rarely been reported. We report a case of prosthetic valve marantic endocarditis occurring three months after insertion of a mechanical mitral valve.

Case Report: A 51 year-old Caucasian male with a history of treated hepatitis C, hypertension, and diabetes presented to his primary care physician with mild confusion and forgetfulness. He was subsequently hospitalized with new-onset seizures secondary to multiple cerebral infarcts. An echocardiogram showed vegetations on his native mitral valve, with significant mitral regurgitation. He ultimately underwent mitral valve replacement with an OX-2 prosthesis. Blood cultures were persistently negative, but culture of the native valve grew *Enterococcus faecalis* in the broth only. The patient was treated with ampicillin and gentamicin for six weeks for presumed infective endocarditis and transferred to a rehabilitation facility. Approximately two months later, he was hospitalized with congestive heart failure. A transesophageal echocardiogram (TEE) at this time revealed an echogenic, non-mobile mass surrounding the mechanical mitral valve, pulmonary hypertension and significant tricuspid regurgitation. An emergent repeat mitral valve replacement was performed. On gross pathologic examination of the explanted prosthetic mitral valve, thin fibrinous deposits were noted on the rim of the mitral valve prosthesis. All cultures were sterile. Histopathology was consistent with the diagnosis of marantic endocarditis. Further workup revealed an anticardiolipin IgG antibody level >120 units and anti-β₂-glycoprotein 1 IgG level >150 units supporting a presumptive diagnosis of APS. Extensive workup for underlying connective tissue disease and malignancy was unrevealing. The patient was diagnosed with primary APS, given his infarcts and serologies; this was the presumed etiology of his marantic endocarditis.

Discussion: APS is associated with venous or arterial thrombosis, or pregnancy morbidity in the presence of one or more specific antiphospholipid antibodies. Such antibodies include a lupus anticoagulant, anticardiolipin antibodies and anti-β₂-glycoprotein 1 antibodies. Primary APS occurs in the absence of underlying connective tissue disease. Valvular thickening and marantic endocarditis are the most common cardiac manifestation of APS. The mitral valve is most commonly affected. Recently, Khan et al reported a case of bioprosthetic valve marantic endocarditis in a patient with lung cancer. However, to our knowledge, prosthetic valve marantic endocarditis associated with primary APS has not previously been reported.

METASTATIC BREAST CARCINOMA INVOLVING THE THYROID GLAND

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Introduction: Secondary involvement of the thyroid gland from a remote primary malignancy is uncommon. We discuss here a case of metastatic disease involving the thyroid gland. **Case Report:** A 68-year-old woman with a history of breast cancer diagnosed 3 years ago, treated with lumpectomy and adjuvant chemotherapy and then lost to follow up, was admitted with an enlarging left axillary mass. On exam, she was found to have a 6 cm thyroid nodule, which, the patient acknowledged, had been growing over the last several years. FNA revealed markedly atypical cells with increased N/C ratio; papillary clusters were also seen. While metastatic disease could not be excluded, total thyroidectomy was undertaken with a presumptive diagnosis of papillary thyroid carcinoma. Final pathology revealed poorly differentiated adenocarcinoma with extensive necrosis. Immunostaining was CK7+ and CK20-; BRST, ER, PR, Her2/neu, CEA and thyroglobulin were all negative and the tumor was deemed to be a metastatic lesion to the thyroid with a breast primary. She was started on radiation therapy to the left axilla; adjuvant chemotherapy is planned. **Discussion:** Metastases to the thyroid are rare despite its rich blood supply. The distinction of a metastatic lesion from a primary thyroid malignancy is important because the treatment may vary significantly. Usually the appearance of a new thyroid nodule or an increase in size of a pre-existing thyroid lesion, in a patient with a known malignancy suggests a metastatic lesion rather than a primary thyroid cancer. FNA with immunostaining will often clarify the diagnosis. Unfortunately, immunostaining was not helpful in our patient's FNA and she underwent thyroidectomy. A pre-operative diagnosis of metastatic breast cancer might have changed the treatment paradigm. While surgical resection of an isolated metastatic lesion may result in prolonged disease free survival, most patients have multiple metastases by the time thyroid metastases are discovered, conferring a poor prognosis.

MI OR NOT MI? : ELEVATED CARDIAC TROPONIN I AFTER COMPETING IN A TRIATHLON?

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Introduction: Cardiac troponin I is typically elevated in patients with acute myocardial infarction. However, elevations in troponin I can occur with other clinical conditions and may complicate differential diagnosis and lead to unnecessary diagnostic testing and inappropriate therapy. **Case Report:** A 54 year old female, non-smoker, with a history of well controlled hypertension and hyperlipidemia (10 year cardiovascular risk based on Framingham guidelines was 2%, total risk of 4%), presented to the emergency room with severe substernal non-radiating chest pain beginning 12 hours after completing the New York Triathlon. The patient had no other coronary risk factors and denied nausea, diaphoresis, dyspnea, palpitations or other associated symptoms. The chest discomfort resolved one hour after receiving two sublingual nitroglycerine tablets in the emergency room. Electrocardiogram was unremarkable except for sinus bradycardia. There was no evidence of myocardial ischemia or infarction. Initial laboratory studies were: total creatine kinase (CK) of 194 U/L (reference level 22-232); CKMB of 14.1U/L (reference level 0-5), and troponin I of 4.44 mug/l (reference level:<0.78). The patient was admitted to the coronary care unit with a presumptive diagnosis of acute myocardial infarction. Peak troponin I was 6.38 mug/l five hours after the admission and nine hours after it declined to 5.12mug/l. An echocardiogram was unremarkable. Coronary angiography and left ventriculography revealed no obstructive coronary lesions, coronary spasm or regional wall motion abnormalities. The patient was discharged with a diagnosis of acute myocardial infarction. She has remained free of any cardiac complaints and has continued to participate in triathlons.

Discussion: Elevated troponin I levels are a key element in the diagnosis of acute myocardial infarction. However, abnormally elevated troponin levels may also be physiological and/or pathological in association with coronary vasospasm, congestive heart failure, myocarditis, pulmonary embolism, cardiac infiltrative disorders, renal insufficiency and/or sepsis in the absence of myocardial infarction. Serum troponin levels are commonly elevated in athletes after strenuous exercise. Therefore in patients with low probability of coronary ischemia or injury, increased troponin levels may not indicate myocardial infarction and may divert attention from other causes.

Conclusion: Clinicians observing elevated troponin I elevations in athletes soon after participating in competitive endurance exercise should be aware that elevated troponin I levels alone, in the absence of typical ECG changes, symptoms of coronary insufficiency or imaging evidence of loss of viable myocardium, are not sufficient for the diagnosis of myocardial infarction.

MYCOBACTERIAL COLITIS PRESENTING WITH MASSIVE RECTAL BLEEDING

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And Michael Carson, MD

Introduction: We describe a patient with massive rectal bleeding from colonic tuberculosis. **Case report:** A 62 year old hemodialysis (HD) patient living in the USA for 17 years, with frequent trips to her native Mexico, presented with three episodes of symptomatic massive painless bleeding per rectum. Eight months prior to this a CT scan of the chest revealed diffuse centrilobular nodules without cavitations and a pleural effusion. After 2 negative PPDs, bronchoalveolar lavage and pleural fluid analysis were negative for AFB culture or stain. A PET ordered to evaluate the lung lesions revealed normal lungs, but increased uptake in the colon. A colonoscopy was scheduled but she missed the appointment. The test was rescheduled, but she presented to the hospital before the planned date. During the current hospitalization a colonoscopy revealed multiple ulcers consistent with tuberculosis (TB) or sarcoidosis, and the biopsies revealed active granulomatous colitis and few AFB on Kinyoun stain. PPD was again negative. HIV serology was negative. Repeat chest CT revealed worsening lung nodules. Wedge resection of the lung revealed granulomatous pneumonitis, focal areas of caseation necrosis and presence of acid fast bacilli. Quadruple therapy with isoniazid, pyrazinamide, rifampicin and ethambutol. Six weeks after discharge she presented with difficulty seeing and hallucinations. Ophthalmological exam and MRI of the brain were normal. The clinical diagnosis was psychosis secondary to the antituberculous medications, which were subsequently held. Unfortunately her confusion continued, she developed pneumonia with respiratory failure, bacteremia, and eventually had a cardiac arrest. Because of anoxic brain injury from the arrest the family decided to withdraw care.

Discussion: Abdominal TB has been reported in renal transplant, peritoneal dialysis patients, and patients who immigrated from East Asia, but TB colitis is not common in the United States or among HD patients. This case demonstrates the need to remain diligent and recognize the resurgence of tuberculosis secondary to increased immigration, more immunosuppressive therapy and AIDS. Including intestinal TB in the differential will prompt biopsy and early diagnosis and treatment for those at risk. "

MYXEDEMA COMA COMPLICATED BY CARDIAC ARREST AND RESPIRATORY FAILURE

Adrian Scaunasu

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INTRODUCTION: Myxedema coma is a rare medical emergency, usually the result of a precipitating event, in a patient with longstanding hypothyroidism. Myxedema coma is a misnomer because most patients exhibit neither the nonpitting edema known as myxedema, nor coma. The principal manifestation of myxedema coma is a deterioration of the patient's mental status. While the actual prevalence of myxedema coma is unknown, its lethal nature demands recognition.

CASE: An 86-year-old Caucasian female with a history of atrial fibrillation and hypothyroidism presented with confusion and decreased short term memory. Her speech was noted to have increased in volume but slowed in rate. She had not seen a doctor in 4 years and had not been taking her thyroid medication for at least that long. On admission the patient was bradycardic, hypotensive and hypothermic: pulse 54 bpm, BP 90/55 mmHg, Temperature 91°F. PE revealed a lethargic but arousable woman, thin build in no respiratory distress. Patient had dull facies with periorbital puffiness, cool and dry skin. Thyroid gland was not palpable and relaxation phase of deep tendon reflexes was prolonged. Laboratory: Na 128 mmol/dL, K 3.1 mmol/dL, Chloride 100 mmol/dL, Bicarb 29 mmol/dL, Glucose 101 mg/dL, BUN 36 mg/dL, Creatinine 1.6 mg/dL, Free T4 0.17 mcg/dL, TSH 108.68 mIU/L, CPK 1192 U/L, Troponin-I 0.03 mcg/L and Hb 11.2 g/dL. Hydrocortisone 75 mg every six hours along with a 300 mcg bolus of intravenous (IV) levothyroxine and fluids were begun. The patient's mental status rapidly deteriorated and cardiopulmonary arrest, characterized by pulseless electrical activity, ensued. The patient was successfully resuscitated and transferred to the ICU on mechanical ventilation. Evaluation for pulmonary embolus, myocardial infarction and sepsis was negative. IV Synthroid 100 mcg daily was continued. The patient slowly improved, was weaned from the ventilator and was transferred to a subacute rehabilitation unit in improved condition.

DISCUSSION: This case highlights the gravity of severe hypothyroidism and the rapidity with which patients may deteriorate once they are myxedematous. Factors associated with a poor prognosis include advanced age, bradycardia and persistent hypothermia. The most important elements in the treatment of myxedema coma are early recognition, thyroid hormone replacement, hydrocortisone and supportive care. While myxedema coma carries a significant mortality rate even with appropriate treatment, an early diagnosis is essential for favorable outcome.

NATURAL HISTORY OF UNTREATED PULMONARY EMBOLISM IN A PATIENT WITH SEVERE MITRAL INSUFFICIENCY

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Background: Pulmonary Embolism (PE) is the third leading cause of death in the United States. The majority of deaths occur in patients in whom PE is not diagnosed or treated. We report a case of probable untreated PE in which the diagnosis was initially missed and no anticoagulation initiated. Two months after the first symptoms occurred, extensive PE was discovered.

Case presentation: A 42 year old male, nonsmoker, with a history of mitral valve prolapse presented after a recent syncopal episode. Two months prior to admission to our hospital he developed marked dyspnea, non-productive cough and hemoptysis. Upon admission to another hospital chest x-ray and non-contrast CT findings showed a right upper lobe infiltrate. He was treated with antibiotics for community acquired pneumonia with no improvement. After extensive negative infectious disease workup, including, blood cultures, serologic testing, HIV testing and bronchoalveolar lavage, the patient was empirically treated with Bactrim and Itraconazole. Upon admission to our hospital for evaluation of syncope and continued dyspnea and cough, vital signs were normal and physical exam was unremarkable except rales in the right posterior lung field and a 4/6 apical holosystolic murmur. EKG demonstrated QT prolongation (QTc 504 ms) which resolved with discontinuation of Itraconazole. Chest x-ray revealed a right-sided pleural effusion and right lower lobe infiltrates. A contrast enhanced chest CT showed bilateral pulmonary emboli extending from the main pulmonary arteries to the lower lobes, with lesser involvement of the upper lobe arteries. No peripheral deep venous thrombi were found. A transthoracic echocardiogram revealed severe mitral regurgitation, possible ruptured chordae tendinae, and preserved LV function. After extensive evaluation, no specific cause for syncope was detected. The patient was started on Warfarin. Outpatient cardiac catheterization was scheduled.

Discussion: Pulmonary embolism is often fatal. It has been estimated that approximately 11% of patients with acute PE die within 1 hour. However, in patients who survive for at least 1 hour, in only 29% of patients is the diagnosis established and treatment instituted. Patients with PE who do not die acutely often present with nonspecific or ill-defined symptoms and the diagnosis is often not made or delayed significantly. A large amount of residual thrombus or clot burden may be found in the lung or other peripheral venous sites and lead to recurrent PE. Ten percent of patients in whom PE was missed and no anticoagulant therapy administered may have a recurrence. This case points out the diagnostic challenges of PE and is an example of the natural history of untreated PE.

NEUROPATHY AND MYELOPATHY DUE TO COPPER DEFICIENCY FOLLOWING GASTRIC BYPASS SURGERY

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Introduction: Copper is an essential trace metal and is a component of key metalloenzymes involved in central nervous system function. Acquired copper deficiency is rare and typically presents with hematological findings of anemia and neutropenia; neurological manifestations secondary to hypocupremia are rarer. We present a case of copper deficiency induced neuropathy and myelopathy following gastric bypass surgery. **Case Report:** A 37 year old Caucasian female with a history of obesity, asthma and pancreatitis lost 100 lbs in 6 months after gastric bypass surgery. She presented with dizziness, confusion, lower extremity paraesthesias, and imbalance, difficulty going up and down the stairs and lifting objects. Neurological examination revealed increased lower limb tone with decreased strength and symmetric brisk reflexes. Distal sensory impairment was profound with absent pain, touch, vibration and position sense. Gait was spastic ataxic. Laboratory results revealed a copper level of 0.68 ug/ml (0.75-1.45), ceruloplasmin 24.8 mg/dl (22.9-43.1), hemoglobin 11.3 g/dl (12-15.5), neutrophils 27% (42-75) and vitamin D (25-OH) < 4ng/ml (30-80). She was started on oral copper replacement titrated to 3 gm daily and high dose ergocalciferol. Later, TPN was initiated for maintenance of weight and to reverse the vitamin and essential elemental deficiencies. Her gait improved significantly along with her muscle strength; her spasticity and her sensory loss are resolving.

Discussion: Copper absorption occurs mainly in the stomach and proximal duodenum. As seen in our case, acquired copper deficiency can develop after gastrectomy or gastric bypass surgery and can lead to peripheral neuropathy and myelopathy. Improvement or reversal of neurologic manifestations after administration of copper supplements supports this diagnosis. Early identification and treatment may prevent progression and facilitate reversal of neurologic dysfunction.

NON B-CELL LYMPHOMA IN AN AIDS PATIENT: AN UNCOMMON EVENT

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Introduction: Only 3% of lymphomas in HIV patients are of T-Cell origin. Anaplastic large T-cell lymphoma (ALCL) comprises 2% of all Non Hodgkin's lymphomas. It is most common in children and young adults. The etiology is unclear, and is rarely seen in HIV patients. The presence of a protein called Anaplastic Lymphoma Kinase (ALK) is associated with a better prognosis compared to the ALK negative subtype. We report a rare case of Anaplastic large T-cell lymphoma, ALK negative, in a young AIDS patient with extranodal involvement. **Case Report:** A 33 year old male with a past medical history of HIV/AIDS and CD4 count of 85 presented with a three week history of progressive severe low back pain, weakness of both legs, diffuse abdominal pain and intermittent subjective fever. On physical examination there was diffuse abdominal tenderness without guarding or rigidity. Tenderness was also elicited on midthoracic and lumbar vertebrae. Abdominal CT revealed celiac trunk and retroperitoneal lymphadenopathy. MRI of the lumbar and thoracic spine ruled out cord compression but revealed an infiltrative marrow process and a large lytic lesion on L4 vertebra. Later, a CT guided biopsy of retroperitoneal lymph node confirmed anaplastic large T-cell lymphoma of ALK negative subtype. He was started on Hyper-CVAD (Cyclophosphamide, Mesna, Vincristine, Doxorubicin, Dexamethasone) chemotherapy regimen. **Discussion:** ALCL primarily involves lymph nodes and skin. The first line of treatment is CHOP (Cyclophosphamide, Adriamycin, Vincristine and prednisone), with Hyper CVAD reserved for more aggressive disease and radiation can be considered for bulky disease. Even with the advancements in the treatment, ALCL-ALK negative subtype has a poor prognosis with overall 5 yr survival rate of 30-50%, but HIV specific data has been limited to case reports. While less common, one must consider T-Cell lymphomas in the differential of an AIDS patient who presents with adenopathy or evidence of metastatic cancer. Research is required with a focus on etiology and effective chemotherapy for this lymphoma subtype considering its aggressive nature.

NORMOCALCEMIC CALCIPHYLAXIS: A NEED TO REEXAMINE THE PATHOGENESIS OF UREMIC ARTERIOLOPATHY

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Introduction: Calciphylaxis is a rare but serious disorder of vascular calcification that leads to ischemia and necrosis of skin and soft tissue. Elevated levels of parathyroid hormone (PTH) and a high calcium-phosphorous product were initially thought to be pivotal in the pathogenesis of this syndrome. The case described herein demonstrates that such laboratory abnormalities are not invariably present in calciphylaxis.

Case: A 70-year-old woman with ESRD on hemodialysis for 5 years presented with extremely tender skin lesions, distributed all over the lower abdomen and lower extremities. The lesions were 2-5 cm extremely tender ischemic/necrotic ulcers with eschars. Laboratory evaluation showed corrected calcium of 9.7 mg/dl (8.5-10.5), phosphate 2.7 mg/dl (2.5-4.6) and PTH 54.5 pg/ml (14-72). She had been on prophylaxis against hypercalcemia and secondary hyperparathyroidism with paricalcitol and cinacalcet. A clinical diagnosis of calciphylaxis was made and was confirmed by punch biopsy of the skin. She was treated with high dose sodium thiosulfate. **Discussion:** Calciphylaxis, also known as calcific uremic arteriolopathy, occurs in about 1-4% of ESRD patients. The pathophysiology of calciphylaxis is poorly understood. Putative mechanisms of pathogenesis include abnormalities in coagulation, defects in inhibitors of mineralization (Fetuin-A and Matrix Gla protein) and an increased calcium-phosphate product (hypercalcemia, hyperphosphatemia and secondary hyperparathyroidism). Associated trigger factors include weight loss, malnutrition, infusion of iron dextran, use of vitamin D and calcium based phosphate binders and insulin use in patients with diabetes mellitus. All these are very common in patients with ESRD; obesity and diabetes are other common co-morbidities. Our patient was interesting due to her lean habitus, normoglycemia and normal calcium, phosphate and PTH levels, a scenario that has been reported with increasing frequency of late and which raises the possibility that other unknown abnormalities of mineralization could also be involved in the development of calciphylaxis in ESRD patients

PARANEOPLASTIC DERMATOSES: A CASE OF RARE COEXISTENCE

Ditina Ghetia, M.D.

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Introduction: Paraneoplastic dermatoses comprise a heterogeneous group of non-inherited skin conditions that herald internal malignancy. Acanthosis nigricans presents as velvety hyperpigmented plaques in intertriginous areas. The sign of Leser-Trélat refers to the explosive onset of multiple pruritic seborrheic keratoses, often with an inflammatory base. Both of these paraneoplastic dermatoses have been associated with gastrointestinal malignancies but rarely coexist. We report a patient in whom the unusual simultaneous coexistence of these two entities was the presenting manifestation of gastric adenocarcinoma.

Case Report: A 46-year-old Mexican man presented to the ER with intermitted episodes of vomiting over the last 2 months. He also reported weight loss and darkening of skin along with a scaly skin rash over the forehead and scalp for the last few weeks. He denied any other past medical history. On examination, he looked malnourished. Skin folds were hyperpigmented with a thick, velvety, leathery consistency suggestive of acanthosis nigricans. He also had evidence of extensive seborrheic keratoses on the forehead and scalp. A 4x5 cm rubbery, mobile, and non-tender left supraclavicular lymph node was palpable. The CBC and metabolic panel were normal. Alkaline phosphatase was 270 IU/L (38-126), LDH 212 IU/L (91-200), GGT 149 IU/L (7-64), CEA 14.7 ng/ml (0-2.5) and CA19-9 was 77 U/ml (0-37). Endoscopic evaluation revealed a large fungating circumferential mass arising in the distal gastric body and extending into the antrum up to the area of the pylorus as well as a nodular mass in transverse colon. Pathology was consistent with poorly differentiated gastric adenocarcinoma from both masses. CT of abdomen and pelvis revealed multiple ring enhancing lesions within the liver and retroperitoneal lymphadenopathy. Treatment options were discussed; however, the patient declined further medical treatment and wanted to return to his homeland.

Conclusion: Paraneoplastic manifestations of gastric adenocarcinoma are rarely seen at initial presentation. Rarer yet is to find acanthosis nigricans and Leser-Trélat at the same time. The expression of cytokines and other tumor growth factors from the primary malignancy are likely responsible for the abrupt appearance of these dermatoses. Since they may be the presenting sign of an occult cancer, cognizance of their features and clinical implications are of considerable importance. Individuals with these syndromes should have a thorough workup for an associated malignancy.

PET SCANNING IN THYROID CANCER: PUTTING A NEWER DIAGNOSTIC MODALITY IN CONTEXT

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Introduction: 18 Fluorodeoxyglucose positron emission tomography (FDG PET) is useful in localizing recurrent or metastatic disease of thyroid origin. We report a case of multifocal papillary thyroid cancer female with negative FDG PET despite metastatic disease.

Case Report: A 58 year old woman presented with a thyroid nodule that had been present for six months. She denied radiation exposure. Physical examination and thyroid ultrasound confirmed a 1.5 cm hypo-echoic thyroid nodule in the left thyroid lobe. TSH was 2.7 uIU/ml (0.35-5.5). Fine needle aspiration (FNA) was suspicious for malignancy. The patient underwent total thyroidectomy; histopathology revealed multifocal papillary thyroid cancer. She was treated with 102 mCi 131I iodine. Whole body scan (WBS) revealed expected uptake in the thyroid bed with no evidence of metastases. One year later, thyroglobulin level was < 0.5 IU/ml (<2.0) and WBS was again negative, suggesting remission. However, a sonogram done concurrently revealed a 5 mm hypoechoic nodule just medial to the left carotid artery. FNA revealed it to be a lymph node with atypia, suspicious for thyroid papillary histology. FDG PET revealed no activity in the cervical lymph node chain and no other evidence of metastatic activity. The patient underwent a modified radical neck dissection. Four lymph nodes (out of 11) showed metastatic papillary carcinoma compatible with primary thyroid cancer. The patient did not receive additional adjuvant treatment and continues to do well.

Discussion: Differentiated thyroid carcinomas (DTC), especially papillary type, are the most common thyroid cancers. DTC run a relatively indolent course with high cure rate and, despite 20-30% recurrence, the estimated 10 year survival is 80-90%. Some thyroid cancers, like that in our patient, do not have or lose the ability to organify iodine, and/or dedifferentiate and hence may have negative WBS and thyroglobulin levels, modalities typically used for surveillance in thyroid cancer survivors. Sonograms and FDG PET scans are particularly important in these patients. FDG PET scans not only help in tumor localization, but may also predict the biological aggressiveness of the tissue. A negative PET scan suggests a rather benign and indolent course and a negative preoperative PET scan is an independent positive prognostic factor for survival. Our patient had a negative stimulated thyroglobulin, negative WBS and a negative FDG PET scan yet was found to have multiple lymph nodes that were positive for metastatic cancer. It is likely that this is not a recurrent disease, rather a residual from the first surgery, and the lack of activity on the PET scan bodes well for the patient.

PITUITARY MACROADENOMA PRESENTING AS HYPONATREMIA

Swaleha Mahpara

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Background: Pituitary macroadenomas in females typically manifest as hormone deficiencies but a nonfunctioning adenoma presenting as hyponatremia in middle aged females is unusual. **Case:** A 56 year old female with past medical history of bipolar disorder presented with a two week history of headache, fatigue, low grade fever, chills, and vomiting. Admission labs: sodium 119 mmol/L, serum osmolality 246, TSH 0.268 IU/mL (low), Free T4 0.53ng/dL (low), FSH/LH/GH all low; GH: normal. Cortisol (mcg/dL): 2 am: 2.4, 8 am 17, post-cosyntropin: 24.7. Prolactin was 43.4 (NL<25). Thyroid hormone supplementation was started. The urine sodium of 53 and urine osmolality of 283 supported a diagnosis of syndrome of inappropriate Anti-Diuretic Hormone secretion (SIADH) and excluded psychogenic polydipsia. Fluid restriction was initiated. Brain MRI revealed a 1.1 x 1.2 x 1.4 cm pituitary macroadenoma with minimal mass effect on the optic chiasm. The sodium improved but she became volume depleted after 3 days, so demeclocycline was prescribed resulting in slight improvements in her sodium and symptoms and she was discharged. She returned to the hospital volume depleted with a urinary tract infection. At this time the demeclocycline was stopped, hydrocortisone was prescribed (50 mg morning/25mg evening) resulting in a significant and sustained improvement in her sodium and symptoms. The final diagnosis was a non-functioning pituitary adenoma causing relative secondary adrenal insufficiency and hyponatremia. Surgery was planned. **Discussion:** The prevalence of pituitary adenomas is 10-20% in autopsy and MRI studies but only 2-8 /100,000 persons per year present with symptoms attributed to pituitary tumors. In secondary hypoadrenalism the serum sodium is generally preserved by aldosterone production. We present an unusual case of pituitary adenoma presenting as hyponatremia. The differential diagnosis included SIADH from compression of the posterior pituitary or the pituitary stalk, or relative adrenal insufficiency. Schmidt's syndrome, the combination of autoimmune hypothyroidism and Addison's disease, can have severely low sodium levels. It is possible that the combination of deficiencies lead to Schmidt's physiology, but for a different reason. While the cosyntropin stimulation test was negative, her response to hydrocortisone strongly suggests that the relative levels of cortisol were too low. Pituitary macroadenoma should be considered in the differential diagnosis of hyponatremia mimicking SIADH in patients with subclinical panhypopituitarism.

POST PARTUM SEIZURES: DIAGNOSING ECLAMPSIA WITHOUT A MRI

Bhavikaben Babaria, M.D.

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BACKGROUND: Thirty-three percent of preeclampsia occurs postpartum: 79% of these are early (<48 hours after delivery) vs. late (>48 hours). Of women with late onset eclampsia, only 22% have been previously diagnosed with preeclampsia. Both syndromes are associated with capillary leak that can be seen on brain MRI. Historically there have been only two MRI patterns of reversible cerebral vasculopathy: Posterior Reversible Encephalopathy Syndrome (PRES) involving multiple foci of edema prominent in the occipital lobes and Call-Fleming syndrome characterized by diffuse cerebral arterial vasoconstriction with less edema than PRES. These MRI findings can be seen in the setting of eclampsia, non-obstetric hypertensive crisis, or vasospastic syndromes associated with vasoconstrictors or SSRIs. We will describe a third pattern of diffuse edema associated with postpartum eclampsia. **CASE:** A woman in her 30s with a history of depression and tension headaches presented with a headache 5 days after a Caesarean section. Presumed tension headache and depression did not respond to fluoxetine and pain medications or her mother's sumatriptan. On postpartum (PP) day #7 she presented to the hospital. Other signs and symptoms of preeclampsia were not present. She was afebrile with a BP of 151/75mmHg, normal neurological exam, negative Kernig's sign, and no edema. She seized in the emergency department, and was loaded with phenytoin but not with magnesium. Head CT and LP were normal. MR-Venogram excluded cerebral thrombosis and MRI showed multiple foci of increased T2/flair signal throughout the subcortical white matter. She was not treated for eclampsia. She was discharged after 4 days on phenytoin. MRI one month after discharge revealed complete resolution of previous findings. **CONCLUSION:** 25% of those with postpartum-eclampsia have normal blood pressures, and it can present out to PP day #14. The differential diagnosis of new peripartum seizures includes the usual causes, but one must consider eclampsia even in the absence of "typical" preeclampsia symptoms as postpartum cases tend to present "atypically" without the classic hypertension/proteinuria. Exclusion of other causes and the rapid resolution of the MRI findings in this patient support a diagnosis of eclampsia with a new MRI pattern of Diffuse Reversible Encephalopathy Syndrome (DRES) caused by the eclampsia-associated capillary leak. Call-Fleming can have edema, but it was excluded by the lack of vasoconstriction. If seizures and blood pressure in this reversible condition are not promptly controlled, permanent neurological damage or even death can occur. Eclampsia will continue to present us with diagnostic challenges, and efforts need to be directed at educating health care workers to recognize this potentially dangerous condition so that an MRI is used to exclude other causes rather than be the first tool to diagnose the syndrome.

PRIMARY FIBROELASTOMA PRESENTING AS MULTIFOCAL EMBOLIC STROKE

Nishant Patel, M.D.

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INTRODUCTION: Primary tumors of the heart are rare, with prevalence at autopsy of 0.002% to 0.33% and Papillary fibroelastoma account for less than 10% of these lesions. Historically, papillary fibroelastoma was an incidental autopsy finding, deemed to have no clinical significance. More recently, papillary fibroelastoma has been implicated in myocardial infarction and stroke suggesting it should be considered potentially dangerous. **CASE REPORT:** A 76 year-old woman with well controlled hypertension and type 2 diabetes mellitus was brought to the emergency room with slurred speech, left facial droop, nausea and vomiting. The facial droop improved on route to the ER but the dysarthria, nausea and vomiting persisted. There was no muscle weakness, tingling, numbness, gait or visual disturbances, headache or palpitation. She denied smoking or alcohol abuse. In the ER she was afebrile with a blood pressure 189/90 mmHg and heart rate 80/min. She was alert, awake and oriented. Speech was dysarthric with word finding difficulty but comprehension was preserved and cranial nerves, motor and sensory functions were intact. MRI revealed multiple areas of acute infarction involving the cerebellum and the junction of the right frontal and parietal lobe. Carotid Doppler was negative. Transesophageal echocardiogram revealed a 3mm fibroelastoma and a patent foramen ovale. Doppler ultrasound of the extremities revealed no deep venous thrombosis. The patient was initially started on aspirin and subsequently anticoagulated. Her nausea and vomiting improved but dysarthria persisted. Resection of the intracardiac mass was recommended but the patient declined and was discharged on coumadin.

DISCUSSION: Papillary Fibroelastoma are the third most common cardiac tumors after myxomas and lipomas but are extremely rare. These tumors have multiple papillary fronds arising from a central stalk (like a sea anemone). They may emanate from almost any endocardial surface but most often arise from the valvular endocardium. They may be identified by echocardiography, autopsy or after surgery. They may serve as a nidus for platelet and fibrin aggregation and lead to emboli resulting in MI or stroke. Surgical excision is generally recommended in individuals who have suffered an embolic event and in those with a highly mobile or large (>1cm) mass.

RENAL CELL CANCER AND THE “DOWNWINDERS”: A POSSIBLE CORRELATION

Anil Aleti, M.D.

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Introduction: Renal cell cancer (RCC) is responsible for 60-85% of all renal neoplasms. It occurs in the 6th to 8th decade of life, affecting about 3 in 10,000 people in the US annually. Risk factors include smoking, acquired renal cystic disease, genetic predisposition and occupational exposure to toxic compounds and possibly ionizing radiation. We present a patient with widely metastatic disease at presentation and speculate on the potential for surveillance in select populations. **Case Presentation:** A 58 year-old man presented with persistent, mild left sided facial droop for 1 week. He denied headache, numbness, tingling, slurred speech or blurred vision. CT revealed multiple hemorrhagic lesions consistent with metastases involving predominantly the frontal lobes. Subsequent CT scans demonstrated more than 50 pulmonary nodules and a large left renal mass, 11.6 x 10.4 cm, extending into the pancreas and left renal vein. Kidney biopsy confirmed the presence of clear cell renal cell carcinoma. IV Decadron was initiated. The patient was transferred to another center and treated with temsirolimus, sunitinib and whole brain radiation. He died four months later secondary to an unrelated surgical complication. **Discussion:** Flank pain, hematuria, and flank mass, though the classic triad of RCC, are seen in less than 10% of patients. Patients with RCC often present with advanced local disease or multiple metastases. Surgical resection of small, localized lesions is the only predictably curative therapy. While newer therapies with immunomodulating agents such as Interleukin-2 and tyrosine kinase inhibitors like sunitinib and sorafenib appear to offer improved efficacy in the treatment of RCC, the 5-year survival rate remains less than 5% for metastatic disease. Clearly identification of early disease would be highly desirable. Interestingly, our patient was born and raised in Arizona during the times of nuclear testing at Nevada and was considered to be among the “downwinders”, individuals exposed to radioactive contamination and nuclear fallout from atmospheric and underground nuclear weapon testing. Four of his siblings had died of various types of cancers. Further investigation is needed to determine optimum surveillance strategies in patients at high risk for malignancy.

RESPIRATORY FAILURE DUE TO ANCA-POSITIVE VASCULITIS ASSOCIATED WITH ENDOVASCULAR INFECTION OF A PACEMAKER WIRE WITH ROTHIA MUCILAGINOSA: CASE REPORT AND REVIEW OF THE LITERATURE

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INTRODUCTION: We present a unique case of respiratory failure due to ANCA-positive vasculitis associated with endovascular infection of a pacemaker wire with *Rothia mucilaginosa*.

CASE: A 66 year old man with pacemaker placement for tachy-brady syndrome and syncope presented with 10/10 sharp, substernal pain and syncope. Cardiac catheterization 3 years ago was normal. Because of a fever 10 days earlier levofloxacin had been started for possible pneumonia. On examination, he was afebrile with HR 110/min, BP104/65 mmHg supine, and 98/55 mmHg seated. Lungs were clear and there were no murmurs or stigmata of endocarditis. The WBC was 13,000/mm³, creatinine 1.9 mg/dL (no baseline available), troponin-I was 3.55 ng/ml, and ECG showed sinus tachycardia. Treatment for acute coronary syndrome and IV fluids were initiated. Fever to 105.6 F developed, and on Day # 5 broad spectrum antibiotics were started. On hospital Day # 7 blood culture grew gram positive cocci in pairs and on Day # 12 *Rothia mucilaginosa* was identified. Initial empiric treatment with linezolid, piperacillin-tazobactam and doxycycline was changed to penicillin. Transesophageal echocardiography identified vegetations on the pacemaker wire. His course was complicated by respiratory failure with bilateral infiltrates of unclear etiology prompting a lung biopsy that revealed fulminant necrotizing vasculitis and pneumonia against a background of chronic interstitial lung disease. This vasculitis was treated with 1 gram of methylprednisolone for 3 days and then a taper was started. Atypical ANCA (mixture of p-ANCA and c-ANCA pattern) was found to be positive. The patient was referred to another institution for removal of the pacemaker wire, but he died despite all efforts. **DISCUSSION:** According to our review of the literature this case is unique for a number of reasons. *R. mucilaginosa*, formerly *Stomatococcus mucilaginosa*, has only rarely been reported to cause infection in immunocompetent hosts (6 cases of endocarditis, two of which were associated with IV drug use ; 8 cases of lower respiratory tract infection, 1 cases of pneumonia; 11 cases of bacteremia, 1 case of vertebral osteomyelitis, 2 cases of peritonitis, 1 case of late prosthetic joint infection, and 1 case of meningitis in a 2 month old child) While there are rare reports of ANCA-associated vasculitis associated with IE, to our knowledge this is the first association of *Rothia* with vasculitis, and the first instance in which *Rothia* associated ANCA-positive vasculitis resulted in respiratory failure.

SECONDARY BACTERIAL PERITONITIS: A DIAGNOSIS LESS TRAVELED

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Background: Intraperitoneal chemotherapy is an effective treatment for ovarian cancer, but infectious complications are common.

Case: A woman presented to another hospital with a spontaneous empyema caused by methicillin sensitive staph aureus (MSSA), and an abdominal mass. The empyema was treated and she was transferred to our institution where surgery revealed ovarian cancer. An intraperitoneal (IP) catheter was placed for chemotherapy. After her third IP chemotherapy cycle she was admitted with increasing ascites, abdominal pain, and vomiting. A large volume paracentesis revealed no cancer cells and WBC count of 148. Antibiotics were not initiated. The Serum Albumin Gradient was <1.1, but impossible to interpret because her serum albumin was <1.0 mg/dL. She was afebrile, blood pressure at her baseline, HR normal, and there were no other signs/symptoms of infection. Two days after the paracentesis there were no clinical signs of sepsis but the abdominal symptoms persisted. MSSA grew from the fluid culture and the medicine service was asked to evaluate the patient. The consult team found and drained an abscess at the inferior wound margin. Nafcillin was initiated. The next day the wound was explored further in the OR, no communication was found between the wound and the peritoneum, and the IP catheter was removed. The culture from the wound abscess yielded polymicrobial growth but the surgical drainage was felt to be adequate therapy. Subsequently the patient's symptoms improved on the single antibiotic. **Conclusion:** "Spontaneous" bacterial peritonitis is a disease seen frequently on medical services in patients with portal hypertension, not ascites due to cancer. While a threshold of WBC count>200 is used to identify patients with spontaneous bacterial peritonitis, this patient had culture-proven secondary bacterial peritonitis suggesting that diagnostic criteria for spontaneous bacterial peritonitis may not be applicable in this setting. IP catheters facilitate treatment of gynecologic malignancies, but 2-9% become infected. As demonstrated here, internists must have a high index of suspicion for catheter complications as indicators of infection can be protean in immunocompromised and malnourished patients. The threshold to treat an infection must be lower when foreign bodies are present.

SEPTIC SHOCK DUE TO MONOMICROBIC EIKENELLA CORRODENS LIVER ABSCESS IN AN ADULT

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Introduction: Eikenella Corrodens is a facultative Gram-negative anaerobic bacillus found as a normal commensal in the human mouth and upper respiratory tract. It is an unusual cause of infection in adults and the vast majority of these uncommon infections occur in patients with diabetes or head and neck cancers; intravenous drug abusers who lick their needles; or as a result of human bites. Both monomicrobial liver abscess and severe infection are extremely rare with this organism. We report a patient with septic shock and liver abscess in which E. corrodens was the only pathogen.

Case Presentation: A 59 year old Hispanic man with history of alcoholism presented with intermittent fever (Tmax 101 F), chills, non-productive cough, SOB, and diaphoresis for 2 days. He also complained of diffuse abdominal pain. He denied intravenous drug abuse. In the ER, he was found to be afebrile and tachypneic with a blood pressure of 59/47 mmHg and pulse rate of 105 per minute. There was right upper quadrant abdominal tenderness with no guarding, rigidity or hepatomegaly. The WBC was 26,900 per mm³ with 48% bands; AST 91 IU/L, ALT 71 IU/L, ALP 203 IU/L. Arterial blood gas analysis showed metabolic acidosis with pH 7.1 and bicarbonate of 8.9 mmol/L. After 4 liters of normal saline the patient's blood pressure was still 94/31 mmHg. Norepinephrine, and broad spectrum antibiotics were administered and he was transferred to the ICU. Aspiration of a 6x6x5.6 cm, complex, heterogeneous mass seen on ultrasound showed inflammatory cells suggestive of hepatic abscess formation. Blood cultures grew E. Corrodens. Antibiotics were changed to ampicillin-sulbactam. No vegetations were seen on a 2-D echocardiogram. A pigtail catheter was placed under CT guidance and adequate drainage was achieved. The patient's condition improved and he was sent home in stable condition on amoxicillin-clavulanic acid.

Discussion: E. corrodens is an uncommon pathogen. It is seen in particular settings as noted above. When it occurs in intra-abdominal infections it is generally a co-pathogen. Only 2 cases of monomicrobial liver abscess and fewer than 10 cases of severe sepsis have been attributed to this organism over the past 50 years. This case illustrates the potential pathogenicity and importance of Eikenella as a cause of severe sepsis in adults, even as a single organism.

STUNNED PARATHYROID: HYPOMAGNESEMIA INDUCED HYPOCALCEMIA

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INTRODUCTION: Hypocalcaemia is a well known manifestation of hypomagnesaemia. End organ PTH resistance or impaired PTH secretion are postulated pathophysiological mechanisms. We report a case of severe hypomagnesaemia causing impaired secretion of PTH.

CASE REPORT: A 61-year-old female with a history of alcohol dependence presented with spasm of both hands for several hours. She had similar self-limited spasms 3 days earlier which lasted for 15 minutes. She also had tingling and numbness in her hands, forearms and perioral area as well as generalized fatigue and calf cramps for the last two weeks. She had lost 10 lbs over 1 month. She smoked 1 pack per day but refused to talk about her alcohol intake. Her medications include aspirin and pepcid as needed for gastritis. On exam she was cachectic with normal vital signs. Carpopedal spasm was noted in all extremities. Chvostek's sign was negative. Laboratory tests revealed serum magnesium of 0.6mg/dl (1.3-2.5), Calcium 6.8 mg/dl (8.5-10.5), albumin 3.3 g/dl (3.5-5.0), phosphorus 4.5mg/dl (2.5-4.6), and intact PTH was 5pg/ml (14-72). Magnesium sulfate, 1 gram, followed IV calcium gluconate were given intravenously. Her tetany resolved over the next 30-60 minutes. Six hours later the serum magnesium was 2.1mg/dl, corrected serum calcium 8.4mg/dl and intact PTH 126pg/ml. 25 -OH Vitamin D was 5ng/ml (30-80). The patient was discharged on Vitamin D, calcium and magnesium replacement. She was counseled against alcohol abuse. She remains eucalcemic and eumagnesemic at last follow up.

DISCUSSION: Hypomagnesemia has been known to cause hypocalcemia but the exact pathophysiological mechanism is still not clearly elucidated. Both impaired secretion and impaired action due to end organ resistance are believed to play a role in the etiology of hypocalcemia. As documented in our case, severe hypomagnesemia causes absolute deficiency of PTH due to impaired secretion and this secretory dysfunction is rapidly reversible once serum magnesium levels are normalized. However, hypocalcemia is likely to persist and take longer to recover. Because alcoholism may be accompanied by nutritional deficiencies (e.g. Vitamin D deficiency in our patient), factors other than magnesium deficiency may have been involved in the hypocalcemia and PTH refractoriness. It is important to treat the patient for other nutritional deficiencies as well to avoid recurrence.

SUBCLINICAL CUSHING'S DISEASE: IS TREATMENT WORSE THAN THE AILMENT?

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Introduction: Classical Cushing's syndrome is exceedingly rare (2 per million). More common is subclinical Cushing's syndrome, characterized by autonomous cortisol production with few clinical manifestations. We present a case of subclinical hypercortisolism due to Cushing's disease and discuss the pitfalls of treatment. **Case Report:** A 40 year old woman was evaluated for fatigue and a random serum cortisol was paradoxically noted to be elevated at 26mcg/dl (6-23). She had gained twenty pounds over several years. She was normotensive and did not appear cushingoid but was overweight (BMI 28 kg/m²) and had supraclavicular fat pads. Fasting blood sugar was 89 mg/dL and serum ACTH was 49pg/ml (6-45). Twenty four hour urine collections documented elevated cortisol excretion, 60 mcg/day and 73 mcg/day (repeated) (0-50). Overnight (1 mg) and low dose (2 mg) dexamethasone suppression tests failed to suppress serum cortisol. A high dose dexamethasone suppression test (8mg) suppressed the ACTH level from 44pg/ml to 6pg/ml (6-45). A diagnosis of Cushing's disease was made. No pituitary adenoma was seen on MRI. Bone mineral density revealed osteopenia (T-score -1.8). Inferior Petrosal Sinus Sampling lateralized the source of cortisol to the left. Transsphenoidal surgical exploration of the sella was performed twice with successful resolution of hypercortisolism. Fatigue improved but over 3 months it recurred, accompanied by myalgias, depression, polyuria and amenorrhea. Laboratory evaluation was consistent with panhypopituitarism and Diabetes Insipidus. Hydrocortisone, levothyroxine, conjugated estrogen + progesterone, growth hormone and desmopressin were administered. The patient remains stable having lost 15 lbs since surgery.

Discussion: Subclinical Cushing's syndrome is frequently due to autonomous cortisol secretion from the adrenal gland. Uncommonly, a pituitary adenoma is responsible. While patients with Cushing's syndrome are at high risk of premature cardiovascular disease, it is unclear whether a similar degree of risk is conferred by its subclinical version. Treatment has been recommended to correct autonomous cortisol secretion and to reestablish the diurnal rhythm, but such recommendations have generally not factored in the risks of pituitary surgery. Absent data from clinical trials, clinicians must weigh the risks of pituitary surgery against those of expectant management with close monitoring for patients in whom subclinical Cushing's syndrome is due to Cushing's disease.

TESTICULAR CANCER PRESENTING AS EXTENSIVE DEEP VEIN THROMBOSIS (DVT): A CASE REPORT

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Introduction: The incidence of thromboembolic events is increased in men who are undergoing cisplatin-based chemotherapy for testicular carcinoma. We report an unusual case of extensive DVT involving the inferior vena cava as a presenting feature in a 20 year old male diagnosed with embryonal carcinoma of the testes.

Case Report: A 20 year old Mexican male presented to the emergency room with pain and swelling of the right lower extremity of 2 days duration. He denied any significant past medical or surgical history. He denied recent travel or fever. He had mild shortness of breath on exertion and low back pain radiating to the epigastric region. On physical examination his vital signs were normal. There was moderate swelling of the right leg along with erythema and tenderness. He also had a firm irregular mass on palpation of the upper half of the right testicle. The rest of the exam was normal. Duplex Doppler Ultrasound done in the emergency room revealed DVT extending from the right common femoral vein to popliteal vein. Scrotal ultrasound demonstrated an irregular heterogeneous mass in the right testicle. CT of the chest, abdomen and pelvis revealed multiple pulmonary nodules and retroperitoneal lymphadenopathy along with acute thrombosis of the infra-renal inferior vena cava with extension to the right iliac system into the right leg and the proximal portion of left iliac system. Biopsy of a retroperitoneal lymph node was diagnostic for non-seminomatous germ cell tumor. A right radical orchiectomy was performed and pathology confirmed the diagnosis of non-seminiferous germ cell tumor (Embryonal Carcinoma). Intravenous heparin was transitioned to Coumadin. Chemotherapy with bleomycin, etoposide and cisplatin was initiated.

Conclusion: Although rare, venous thromboembolism may be the first manifestation of a testicular cancer.

TOTAL HORMONAL ABLATION VERSUS SYSTEMIC CHEMOTHERAPY IN SMALL CELL CARCINOMA OF THE PROSTATE: A CASE REPORT

Ditina Ghetia, M.D.

Co-Author: Peter Mencil, MD

Introduction: Small cell carcinoma (SCC) arising in the prostate is rare and generally much more aggressive than typical adenocarcinoma of the prostate. At diagnosis, patients often have locally advanced lesions, usually in association with nodal and/or distant metastases. The optimal treatment for this entity remains controversial. This case presentation emphasizes important considerations in the treatment of SCC of the prostate.

Case Report: A 51 year old man was found to have an abnormal prostate during a routine physical exam in February 2008. The exam had been normal and PSA 0.77 μ g/L in 2006. Biopsy revealed poorly differentiated carcinoma with extensive neuroendocrine/small cell carcinoma features. CT of chest, abdomen and pelvis were normal as was colonoscopy. FDG-PET imaging showed several 2mm nodules in the lung fields. CEA was 49.1 μ g/L and PSA of 2.37. At re-evaluation at this point, approximately three months from the original finding, the patient complained of constipation and dry ejaculate. A digital rectal exam revealed a markedly enlarged, rock-hard prostate mass with a prominent nodule on the right and volume of greater than 75cc. Bone scan was negative. Repeat CT of the abdomen and pelvis demonstrated retroperitoneal adenopathy confirmed by PET scan as being four lymph node sites. Aggressive chemotherapy was recommended but the patient sought a second opinion at an out of state cancer specialty hospital. Total hormonal ablation therapy was advised and the patient elected to follow this recommendation. The patient's condition continued to deteriorate with progressive constipation and renal failure secondary to prostatic enlargement as well as the development of a pathological fracture. Nephrostomy tubes and a colostomy were placed. Local radiation was administered and systemic chemotherapy was initiated but the course was complicated by urosepsis and spinal cord compression.

Conclusion: Although small cell carcinoma is a usually fatal disease, immediate palliative chemotherapy should be considered. There is no evidence that androgen deprivation therapy is useful in patients with pure SCC of the prostate.

USE OF CARBIDOPA/LEVODOPA AS AN ADJUNCTIVE TREATMENT IN WILSON'S DISEASE WITH PARKINSONIAN-LIKE FEATURES

Bhavikaben Babaria, M.D.

Co-Authors: Adriana Fitzsimmons, MD, Paul Kostoulakos, MD, Deborah Alpert, MD, PhD

Introduction: Wilson's disease (WD), also called hepatolenticular degeneration, is a rare autosomal recessive defect of copper metabolism. WD usually presents between the first and third decades of life with hepatic and/or neuropsychiatric manifestations. The prevalence is 30 individuals per million population. WD is caused by mutations in the gene encoding a copper transporting ATPase known as ATP7B, which is important for copper excretion into bile. It is characterized by pathological accumulation of copper in organs such as the liver, brain, kidney and peripheral cornea. Because effective treatment is available, early diagnosis is important.

Case Report: A 19 year old Asian male with history of depression and prior suicide attempts was brought to the emergency room following an automobile accident in the setting of aripiprazole and bupropion overdose. He suffered a cervical vertebral fracture, which was surgically stabilized. Laboratory evaluations demonstrated thrombocytopenia and elevated transaminases. During his hospitalization, he developed progressive dystonia and catatonic rigidity, which were initially attributed to neuroleptic malignant syndrome related to antipsychotic overdose. He did not improve over time, despite treatment with benzodiazepines and dantrolene. CT scan of the brain demonstrated symmetric low density basal ganglia lesions, and MRI of the brain demonstrated abnormal high signal in the bilateral caudate and putamen, concerning for metabolic disorder such as Wilson's disease. Bedside ophthalmologic evaluation did not reveal Kayser-Fleischer rings. Ultrasound of the liver demonstrated nodular cirrhosis, confirmed by liver biopsy. Further workup demonstrated low ceruloplasmin, low serum copper, and high 24 hour urine copper levels. Workup for other causes of cirrhosis was unrevealing. Copper chelation therapy with trientine was initiated, and he received multiple sessions of electroconvulsive therapy. Given his basal ganglia involvement with Parkinsonian symptoms, he was additionally treated with carbidopa/levodopa. Over several weeks, his rigidity and depression steadily improved, although he required continued assistance with daily activities.

Discussion: Neurological manifestations of WD include increased or abnormal movements, such as atypical wing-beating tremors or dystonia, or a relative loss of movement resembling Parkinsonian rigidity. As copper accumulation in the brain leads to disturbance in basal ganglia function, patients may present with hypo- or hyperkinetic extrapyramidal symptoms. As a result, Parkinsonism is very common. WD may be associated with a dopaminergic deficit. Excess copper accumulation in the brain can lead to the formation of copper-dopamine complexes, leading to the oxidation and inactivation of dopamine. Additionally, several copper-containing enzymes in the brain may have altered function in the setting of copper overload. WD is treated with lifelong use of chelating agents such as penicillamine, trientine, or tetrathiomolybdate. In our patient, given his basal ganglia involvement and Parkinsonian symptoms, we additionally added carbidopa/levodopa to his regimen. Carbidopa/levodopa may be beneficial as an adjunctive treatment for Parkinsonian-like complications of neurologic WD.

MONMOUTH MEDICAL CENTER

AORTOENTERIC FISTULA COUPLED WITH LOWER EXTREMITY ISCHEMIA

Yuliya Nudelman, M.D.

Aortoenteric fistula is an abnormal communication between aorta (either native or graft) and gastrointestinal tract. It is uncommon, life-threatening cause of GI bleedings, and if not recognized leads to 100% fatal outcome. 76 year-old male presented with melena, back pain, mild abdominal pain, generalized weakness, difficulty walking, pain in the left lower extremity since the day of admission. Patient reported episode of GI bleeding two weeks before admission, when upper/lower endoscopy failed to reveal any source of bleeding. He had elective repair of abdominal aortic aneurysm 5 years ago. On admission patient was tachycardic (PS-128/min), hypotensive (BP-89/25 mm/hg), with hemoglobin - 7.2 gm/dl. Physical examination: abdomen soft, non-tender, positive bowel sounds, healed longitudinal scar, no pulsatile masses or abdominal bruit. Rectal exam - grossly bloody. Naso-gastric tube revealed fresh red blood and clots. Left lower extremity was mottled, cold, with decreased movements and sensation from the knee down, femoral and distal pulses were decreased. Urgent upper endoscopy showed residual blood in stomach, clot in the duodenum suspicious for adherent clot of an aortoenteric fistula (AEF); it was not flushed due to possible risk of re-bleeding. CT scan of the abdomen/pelvis revealed air within the aortic stent graft lumen, and high density in the left common iliac portion of the graft, extending into the left common femoral artery, suspicious for diffuse thrombosis. On admission patient and family refused leg amputation, against medical advice. Second vascular surgery opinion stated irreversible leg ischemia and possibility of overwhelming toxemia during surgery plus possibility of massive hemorrhage from AEF; AEF repair would be too extensive and would necessitate lifetime hemodialysis because of bypass reconstruction. Patient was transferred to hospice due to overall poor prognosis and in agreement with his Living Will. Reviewed literature demonstrated mortality from AEF as 100% if left untreated or overlooked, and with operative attempt mortality - 50-90%. Condition usually manifests with herald bleed, then period of "silence" with clot formation and then massive bleeding when patient exsanguinates. Secondary AEF is an uncommon (2%) but catastrophic complication of reconstructive aortic surgery. In this patient there was an unfortunate association of two potentially deadly conditions (AEF and leg ischemia), when time is crucial for any of them.

MORRISTOWN MEMORIAL HOSPITAL

SPONTANEOUS RENAL ARTERY DISSECTION (SRAD) IN AN OTHERWISE HEALTHY MALE

Alpiniano Pintor

Spontaneous Renal Artery Dissection (SRAD) is a rare occurrence with fewer than 200 cases reported in the literature and is usually diagnosed after imaging studies are performed for abdominal or flank pain. Due to its rarity, SRAD may be difficult to diagnose and treat and thus may be a challenge to physicians. A healthy 47 year old white male presented to our emergency room with complaints of sudden onset of periumbilical pain with radiation to both flanks (right greater than the left) while he was driving at the NJ turnpike from a business trip in Philadelphia. This was associated with nausea but no vomiting. Past medical/surgical histories were unremarkable. He only takes multivitamins on a regular basis. Family history was significant for cardiac arrhythmia, coronary artery disease and peripheral vascular disease. He smoked a pack per day of cigarettes for eight years but quit 20 years ago. He usually drinks 1-2 shots of scotch daily. Review of systems was unremarkable. Due to severe pain, patient was already given dilaudid by the time we have examined him. Thus, the physical exam was essentially normal. Initial laboratory results revealed slight leukocytosis, anemia, increase in urea but normal creatinine, hyperbilirubinemia, transaminitis and an elevated lactate dehydrogenase. Urinalysis had some blood and protein. Computed tomography (CT) of the abdomen and pelvis revealed bilateral renal hypodensities, right more than the left, suspicious for renal infarction. Hypercoagulability and vasculitis work-ups done were both negative. A transthoracic and a transesophageal echocardiogram looking for sources of thromboemboli were both negative. Bilateral lower extremities venous dopplers were negative for any deep venous thrombosis. CT Angiogram of the abdomen revealed a right renal artery posterior division branch dissection with resulting right posterior renal infarction. With renal infarcts, patient was initially started on intravenous anticoagulation with heparin. But on discharge with patient being asymptomatic, he was maintained on aspirin and plavix upon the recommendation of the vascular surgeon. Follow-up CT scans will be done on an outpatient basis to monitor if there will be any progression of the dissection. Isolated SRAD is rare and often presents as a diagnostic and therapeutic challenge. It may be misdiagnosed because its clinical presentation may mimic other common diseases like nephrolithiasis or pyelonephritis. Because of this, a delay in the diagnosis and treatment may occur and this may result in uncontrollable hypertension and renal insufficiency; bilateral dissections may compromise both renal arteries. Advanced imaging modalities are helpful in making the diagnosis, but angiography remains the definitive study. The treatment and long-term management of patients with this condition are poorly understood and controversial, and a commitment to long-term follow-up is often required from both the patient and the physicians involved.

RESIDENT HEALTH CLINIC COMPLIANCE WITH JNC 7 GUIDELINES

Susmitha Dhanyamraju, MD

Co-Author: Rebecca Griffith, MD

Resident Health Clinic Compliance with the Seventh Report of the Joint National Committee on Hypertension (JNC-7)

Introduction: JNC-7 guidelines define hypertension in patients with diabetes, chronic kidney disease, and coronary artery disease as blood pressure >130/80mm Hg. The guidelines advocate the practice of step dose therapy and the use of appropriate pharmacologic agents based on comorbidities. The goal of this study was to determine whether hypertension was being appropriately managed in a resident health clinic according to JNC-7 guidelines. Methods: Retrospective chart review of a sample of one hundred patients with hypertension and at least one of the following comorbidities: diabetes mellitus, hyperlipidemia, chronic kidney disease, and coronary artery disease was conducted. Patient data was collected from June 2006 to June 2008 at the Family Health Center of Morristown, a resident staffed clinic. The patients' blood pressure readings, antihypertensive agents and doses were collected during follow up visits which averaged every three to six months. Results: Fifty five percent (55/100) of patients had uncontrolled blood pressure. Among these, eighty-six percent (47/55) were on appropriate pharmacologic agents based on their comorbidities. Sixty-nine percent (38/55) of uncontrolled hypertensive patients were not on optimal doses of their antihypertensive agents. Conclusion: Hypertension is not adequately controlled in the resident health clinic. Although appropriate anti-hypertensive agents were used in the majority of patients, step dose therapy was practice infrequently.

OVERLOOK HOSPITAL

PULMONARY ALVEOLAR HEMORRHAGE AND MIXED CRYOGLOBULINEMIC MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS IN A PATIENT WITH RHEUMATOID ARTHRITIS, NON-ACTIVE HEPATITIS C

Neelima Upputuri, M.D.

Case presentation: A 48-year old woman with a past medical history of rheumatoid arthritis (on methotrexate and Abatacept), non-active Hepatitis C presented with worsening shortness of breath and chest heaviness. CT scan of the chest revealed pathologic mediastinal lymphadenopathy, scattered patchy areas of parenchymal disease and small pleural effusions bilaterally. Pneumonia was suspected and antibiotics were started. Subsequent CT scan revealed markedly worse diffuse alveolar infiltrates. Later she had new onset hemoptysis and developed acute renal failure. Bronchoalveolar lavage revealed pulmonary alveolar hemorrhage. Work up for the etiology of Pulmonary-Renal syndrome was initiated. Complement levels obtained initially and later have been low. Hep C antibody was reactive, but Hep C RNA quantitative was <615 (i.e. low viral load). c-ANCA, p-ANCA, ANA, RNP antibody, ASO, antiphospholipid antibodies and anti-GBM were negative. Rheumatoid factor was 162. A VATS procedure with lung wedge biopsy showed acute and organizing pulmonary hemorrhage compatible with diffuse alveolar hemorrhage of immune-mediated etiology. Renal biopsy showed diffuse endocapillary proliferative glomerulonephritis with membranoproliferative features and focal crescent formation, highly suggestive of acute type II mixed cryoglobulinemic glomerulonephritis (CGN). Treatment with cyclophosphamide, rituximab and pulse steroids was initiated. Serum Cryoglobulins, IgG, IgA were low, and IgM was normal. Serum viscosity was low. Serum free light chains showed increased kappa but normal lambda suggesting monoclonality. Immunofixation showed two monoclonal IgM Kappa proteins, a monoclonal IgG kappa, none of which could be quantified. However bone marrow biopsy showed normocellular marrow with no evidence of malignant lymphoma or plasma cell dyscrasia. Patient's clinical condition improved minimally initially but later progressively deteriorated. She also developed purpura, severe arthralgia and malaise. Her renal function worsened requiring dialysis, continued to have hemoptysis and respiratory distress requiring multiple intubations. Plasmapheresis and Mycophenolate were also started. Despite aggressive treatment she succumbed to death. Discussion: We describe a female with a medical history of Rheumatoid arthritis, non-active Hepatitis C presenting with pulmonary alveolar hemorrhage and CGN. There is no literature suggesting association of Abatacept with pulmonary hemorrhage. Initial considerations included pulmonary-renal syndromes. Negative serology made etiologies such as Goodpasture's syndrome, pauci-immune glomerulonephritis and Churg-Strauss syndrome less likely. Alveolar hemorrhage in association with hepatitis C virus related mixed cryoglobulinemia was previously described in very few cases. This is the first case in which a patient with Alveolar Hemorrhage and CGN has presented with acute deterioration leading to death. Furthermore, two concomitant major systems attributing to clinical outcome has rarely been reported. Conclusion: This is a case of a patient with non-active Hepatitis C found to have mixed cryoglobulinemic membranoproliferative GN and pulmonary alveolar hemorrhage in the setting of active Rheumatoid Arthritis. Alveolar Hemorrhage is a rare manifestation of CGN and the combination of both signifies a poor prognosis with high mortality.

RARITAN BAY MEDICAL CENTER

IMMUNE RECONSTITUTION SYNDROME, THE DOWNSIDE OF ANTIRETROVIRAL THERAPY

Vishnu Sundaresh MD

Co-Authors: Mohamed Radwan MD, Jose Cofino MD, and Nina Regevik MD FACP

In HIV-infected individuals, paradoxical worsening of preexisting infectious processes following the initiation of highly active antiretroviral therapy (HAART) is referred to as Immune Reconstitution Inflammatory Syndrome (IRIS). If immune function improves rapidly following the commencement of HAART, systemic or local inflammatory reactions may occur at the site or sites of the preexisting infection. This inflammatory reaction is usually self-limited, especially if the preexisting infection is effectively treated. However, long-term sequelae and fatal outcomes may rarely occur, particularly when neurologic structures are involved. We present three cases of this syndrome which were diagnosed and treated in our hospital. A 20-Year-Old female who was diagnosed with AIDS in her 25th week of pregnancy. Her viral load was 750,000 copies and her CD4 count was 92 at that time. One month prior to that she had a PAP smear that showed a low grade intraepithelial lesion. She was started on HAART. She underwent colposcopy and biopsy 3 months later which showed severe squamous dysplasia despite an undetected viral load and a CD4 count of 623. Her repeat biopsy in 1 and 2 months showed moderate and mild squamous dysplasia respectively. Her CD4 count was 536 with undetectable viral load. The second case is a 37-year-old male who presented to the emergency department with fever, headache, neck stiffness, and lethargy. His CSF analysis was positive for Cryptococcal neoformans. His HIV test was positive. His CD4 count was 63 and his viral load was 700,000. He was started on HAART and was treated appropriately for cryptococcal meningitis. Within 2 months of therapy his mental status deteriorated and repeat CSF analysis remained positive. His motor function was severely compromised despite a noticeable increase in his CD4 count to 299 and undetectable viral load. His functional status gradually improved afterwards but he remains hospitalized. Finally, a 48-year-old female with HIV diagnosed upon diagnosis of Pulmonary TB. She was started on HAART and anti-tuberculous medications but she developed worsening diffuse lymphadenopathy. A lymph node biopsy showed caseating granuloma. Her cervical lymphadenopathy compromised her breathing. Her HAART was stopped and her anti-tuberculosis medications were continued. Her lymphadenopathy subsided and her HAART was resumed. The diagnosis of IRIS poses a significant challenge as it could simulate other opportunistic infections and lead to a diagnostic dilemma. Meanwhile, the treatment of such cases is not yet clear. The management of symptoms, particularly through the use of anti-inflammatory drugs, has been recommended but not usually beneficial. Steroids represent an important, although double-edged, measure. Immune modulation is warranted, but specific drugs and protocols are lacking.

THYROTOXIC PERIODIC PARALYSIS IN A PATIENT FROM ARGENTINA

Vishnu Sundaresh MD

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Thyrotoxic Periodic Paralysis (ThPP) is a very rare neuromuscular disorder of uncertain etiology, related to a defect in muscle ion channels, and manifest by episodes of painless muscle weakness. It is most widely reported and studied in Asian populations, in whom there is a 10-fold higher incidence of this complication than in white populations. The increasing trans-continental migration has made it more common in the United States. We present a case of ThPP in a 27 year male from Argentina. A 27 year old male presented with sudden onset of severe proximal muscle weakness in all four extremities. He had a couple of similar episodes in the past with spontaneous resolution within few hours. He had been suffering from hand tremors for the last 6 years. He did not have any family history of thyroid disease. On physical examination, he had tachycardia, a diffusely palpable thyroid gland, and tremors of the outstretched hands. The motor function was 0/5 in his lower extremities and 3/5 in the upper extremities. His deep tendon reflexes were 0 bilaterally in the lower extremities and 2+ bilaterally in the upper extremities. His serum potassium was 2 mEq/ml, magnesium was 1.9 mEq/ml, and his phosphorus was 2.8 mEq/ml. His creatinine phosphokinase was 136. The thyroid function tests showed a Free T4 of 3.83 ng/dl, Total T3 ng/dl of 480, and TSH of <0.01 microU/ml. The anti-thyroglobulin antibody was 77 IU/ml and the anti-thyroid peroxidase antibody was 653 IU/ml. The EKG revealed a prolonged QT interval. His potassium was replaced which resulted in a dramatic improvement in his symptoms. The ultrasonography of the thyroid gland revealed a heterogeneous and enlarged thyroid gland without evidence of discrete nodules. There was an abnormal elevation of thyroid uptake on Iodine-123 scan. The patient was diagnosed to have Thyrotoxic Hypokalemic Periodic Paralysis and was started on therapy with Propranolol and Methimazole. The patient was discharged home on daily potassium supplement and methimazole, and was advised outpatient follow up to monitor thyroid function test and serum potassium level. This case illustrates a rare presentation of Thyrotoxic Hypokalemic Periodic Paralysis resolved almost immediately with potassium replacement. One more interesting fact is that this patient is from Argentina, whereas thyrotoxic periodic hypokalemic paralysis is more common in the eastern part of the world. In patients presenting with acute onset of weakness and hypokalemia, regardless of their continent of origin, it is always prudent to rule out thyroid dysfunction and exclude this rare but rather treatable condition.

UMDNJ – ROBERT WOOD JOHNSON MEDICAL SCHOOL – COOPER HOSPITAL – CAMDEN

DIFFUSE LARGE B-CELL LYMPHOMA PRESENTING IN THE PERIANAL FISTULOUS TRACT IN A PATIENT WITH CROHN'S DISEASE AFTER IMMUNOSUPPRESSIVE THERAPY

Florence Momplaisir, M.D.

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Introduction: The inflammatory bowel diseases (IBD) Crohn's disease and ulcerative colitis have traditionally been treated with steroids, 5-ASA agents, and other biological agents. Recently, immunomodulators such as 6-mercaptopurine (6-MP) and infliximab have emerged as effective therapeutic agents for IBD and other inflammatory diseases. With the increase use of these immunomodulators, studies linking lymphoma and the long term use of these agents have emerged. Here, we report a case of diffuse large B-cell lymphoma (DLBCL) presenting in the fistulous tract of a patient with Crohn's disease. **Case:** A 42 year old perimenopausal female with a long standing history of hypertension and Crohns disease complicated by a non-healing perianal fistula received treatment with 6-MP and infliximab for six years. Her other medications included asacol, atenolol, hydrochlorothiazide, spirinolactone, folic acid and norethindrone. She underwent multiple debridements of her perianal abscesses. She eventually became asymptomatic and her immunosuppressive therapy was discontinued. A year later, she developed a painful and exudative mass which on physical exam extended from the perianal area into the labia majora and posteriorly half way into the gluteal muscle. This mass originally was thought to be a recurrent abscess and was treated as such for four months. Eventually, a biopsy showed it to be an aggressive DLBCL.

Discussion: The immunomodulators infliximab and 6-MP are widely used in IBD patients. Current indications include maintenance therapy, steroid-dependant disease, fistula closure, and prevention of Crohn's disease recurrence. As internists, we need to be aware of potential harmful side effects associated with these treatments. A recent meta-analysis suggests an approximate fourfold increase risk of lymphoma in IBD patients treated with immunomodulators but no prior case of DLBCL have been reported. DLBCL is the most common histologic subtype of Non Hodgkin's Lymphoma (NHL) accounting for approximately 31 percent of patients with NHL. DLBCL is a heterogeneous group of malignancies composed of transformed B-cells. Infliximab induces apoptosis of monocytes and activated T lymphocytes, but its effect on B lymphocytes is unknown. Recent in vitro data shows infliximab has a direct effect on apoptosis of B cells but instead affect impaired T cells.

Conclusion: The immunomodulators infliximab and 6-MP have become the standard of care for complicated IBD. As internist, we need to monitor our patients closely for new symptoms suggestive of lymphoma. This case is unusual because to our knowledge, it is the first case of DLBCL presenting as a perianal mass in the fistulous tract in a patient with Crohn's disease after prolonged treatment of infliximab and 6-MP.

UMDNJ – ROBERT WOOD JOHNSON UNIVERSITY HOSPITAL – NEW BRUNSWICK

THE PREVALENCE AND ROLE OF FIBROMYALGIA IN INFLAMMATORY BOWEL DISEASE

Daniel Blachman, M.D.

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Purpose: It is known that patients with Inflammatory Bowel Disease (IBD) have peripheral arthritis, spondylitis, fatigue and elevated rates of depression. Fibromyalgia (FM) or a FM-like illness involving depression is common in other autoimmune diseases and may be common in IBD. The objective of a larger study is to determine the prevalence of FM in IBD and assess the relationship between FM, depression and other variables. Herein we report the preliminary results. **Methods:** Thirty-five consecutive patients with IBD between the ages of 18 and 80 were recruited from the Crohn's and Colitis Center of New Jersey at the University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School. Participants were consented and then asked to complete questionnaires including the Quick Inventory of Depressive Symptomatology-Self Report (QIDS). Next, physical examinations to evaluate cumulative tender point counts were performed using a dolorimeter. Patients were divided into two groups: FM-like illness (tender points >7) and no FM (tender points = 0 to 7). **Results:** Six patients (17.1%) met full criteria for FM, and another 5 patients (14.3%) had 8 or more tender points. Thus, a total of 11 patients (31.4%) were assigned to the FM or FM-like illness group (n=11). The mean score on the QIDS for those with FM or FM-like illness was 9.36 (SD 5.9) vs. 5.58 (SD 3.7) for IBD patients without FM or FM-like illness. QIDS scores > 9 are associated with mild depression. Using an independent samples t-test, we found that compared to IBD patients without FM or FM-like illness, those with FM or FM-like illness are more likely to be depressed (P= 0.026).

Conclusion: Our findings suggest an association between FM or an FM-like illness and IBD. It should be noted that the prevalence of FM in the general population is approximately 2%. Our results are consistent with those of others reporting elevated rates of FM in other autoimmune disorders like systemic lupus erythematosus and rheumatoid arthritis. We propose that symptoms frequently seen in FM such as depression and diffuse pain or tender points should be important components in the evaluation and management of IBD patients. Most IBD patients in this small sample had only mild depression yet inquiring about and treating such symptoms could greatly enhance quality of life. In the future, the role of musculoskeletal pain, depression and sleep disturbance in IBD should be explored.

SAINT BARNABAS MEDICAL CENTER

DO WE NEED "BRIDGING THERAPY" ON WARFARIN INTERRUPTION FOR INVASIVE PROCEDURES?

Nirav Jasani, MD

Co-Author: Sunil Sapru, MD.

Introduction: There is uncertainty about optimal management of patients who must discontinue warfarin sodium therapy before an invasive procedure. We therefore report our experience with a patient who developed thromboembolism in such a situation and review the pertinent literature.

Patient Presentation: A 50 year-old Caucasian gentleman developed sudden onset of left flank pain of almost 10 hours' duration. The pain was constant, non radiating, moderately severe in intensity and felt like a "pulling" sensation in the loin region. He was recently diagnosed with type 2 diabetes mellitus. He had coronary artery disease, with percutaneous coronary intervention with stenting (report not available) approximately 12 years ago and ischemic cardiomyopathy with an implantable cardioverter-defibrillator (ICD) 3 years ago. He also had atrial fibrillation and he was on coumadin for anticoagulation. A recent visit to cardiologist revealed ICD malfunction; a two-dimensional transthoracic echocardiography showed left ventricular ejection fraction of 10 % with a possible apical left ventricular clot. A trans-esophageal echocardiography and ICD upgrade as well as a coronary angiography was planned. Coumadin was held 3 days prior to his hospitalization. The examination revealed atrial fibrillation with a fast ventricular response. A computed tomography with intravenous contrast showed a moderately large area of decreased attenuation involving the middle third of lateral aspect of left kidney, consistent with a renal infarct. He was hospitalized and treatment with low molecular weight heparin was initiated.

Discussion: Our patient illustrated the dilemma of defining optimal management of patients who must discontinue warfarin therapy before an invasive procedure. We are unaware of published information about the risk of thromboembolism associated with short term warfarin therapy interruption. There is uncertainty about the efficacy of "bridging therapy" on interruption of warfarin with either unfractionated heparin or low molecular weight heparin and also a risk of perioperative bleeding with it. Most authorities recommend its use for patients with the highest risk of embolism; this varies from 1 to 20 % depending on the risk factors (i.e., previous embolism, hypertension, age > 75 years, left ventricular dysfunction, diabetes and mitral stenosis). Based on the predicted decline in the International Normalized Ratio (starts to fall approximately 29 hours after the last dose of warfarin and then decreases with a half life of approximately 22 hours) it is reasonable to start "bridging therapy" approximately 60 hours after the last dose of warfarin (e.g. the third morning after the last evening dose). The risks and benefits of such therapy should be discussed with patients.

SAINT JOSEPH'S REGIONAL MEDICAL CENTER

ENDOVASCULAR APPROACH TO THE MANAGEMENT OF AORTIC COARCTATION

Enis Alberaqqdar, M.D.

Co-Authors: Joseph Daoko MD, and Aiman Hamdan MD FACC

Introduction: Aortic coarctation is a congenital defect characterized by narrowing or constriction of the lumen of the aorta that may occur anywhere along its length but most commonly is distal to the origin of the left subclavian artery at the insertion of the ligamentum arteriosum. For a long time surgical repair was the only option for these patients, but it had a long list of complications, and significant morbidity and mortality rates. Therefore an endovascular approach was developed by interventional cardiologists to perform angioplasty and stenting at the site of coarctation.

Case Report: Our patient is a 37 year old male with past medical history of HTN, CVA, ex-smoker, and ex-cocaine abuser (until a year ago). He came to the ER complaining of episodes of substernal chest pain for 3 days each lasting 30 Min, of 7/10 in intensity with numbness of the left arm, no aggravating factors, not associated with shortness of breath or palpitation, relieved spontaneously. Physical examination showed pulsus parvus et tardus in the lower extremities compared to the upper extremities. The patient had 2/6 ejection murmur at the apex. BP in the upper extremities was 128/64; in the lower extremities it was 78/55. Cardiac enzymes were negative. EKG was normal. CXR showed notching of the inferior aspects of the ribs with prominent aortic shadow. CT scan of the chest showed 7.7 mm coarctation of the proximal descending aorta and dilation distal to the lesion of 23 mm. 2D echo showed normal aortic valve with normal myocardium (EF 60%). Patient opted for angioplasty over surgery and was taken to the cath lab; the procedure was done with stenting successfully.

Discussion: Endovascular approach in the management of aortic coarctation may be a better choice for a carefully selected population of patients without associated cardiac anomalies.

CARDIOMYOPATHY IN A PATIENT WITH DUCHENNE MUSCULAR DYSTROPHY

Enis Alberaqqdar, M.D.

Co-Authors: Joseph Daoko MD, Aiman Hamdan MD FACC, and Mahesh Bikkina MD, FACC

Introduction: Duchenne dystrophy is an X-linked disease with proximal muscle weakness and cardiomyopathy. Dystrophin gene mutation is responsible for the disease. Death usually results from respiratory failure or cardiorespiratory failure. Presentation age usually is between age 20 and 50 years, usually with arrhythmias.

Case Report: Our patient is a 26-year-old Hispanic male who came to ER complaining of shortness of breath for the past week with cough that is productive of white sputum and some wheezing. The patient describes similar episodes in the past with persistent shortness of breath but this time it was severe enough to come to ER. The patient has past medical history of Duchenne muscular dystrophy that was diagnosed at the age of 7 years old, since then the patient was following with his PMD but not on regular bases. Last office visit was two and half years ago as per patient. Physical examination showed temp of 98.6 HR of 92 and BP of 85/42 with Prominent JVD in the neck. On heart examination, the heart sounds were distant; PMI was displaced laterally without murmurs. The lung examination revealed scattered rales BLT and wheezing. EXTs were atrophic without edema. EKG showed characteristic changes of myocardial involvement in Duchenne syndrome marked by positive R wave in lead V1 Patient was admitted to the hospital with the diagnosis of CHF exacerbation due to worsening of cardiomyopathy caused by Duchenne muscular dystrophy. The patient was started on diuretics, ASA, ACEIs. 2D echo was done and showed EF of 10-15% without valvular abnormality or hypokinesis. The patient symptoms improved significantly and the patient respiration improved. Subsequently an AICD was placed successfully prior to discharge due the high rate of sudden death and arrhythmias in patients with Duchenne.

Conclusion: Duchenne dystrophy is a devastating disease that destroys the skeletal muscles and do not spare the cardiac muscle from destruction with characteristic EKG changes causing death via cardiac and/or respiratory failure.

SAINT MICHAEL'S MEDICAL CENTER

BRUGADA SYNDROME DIAGNOSED 15 YEARS AFTER SUDDEN CARDIAC DEATH

Chady Sarraf, M.D.

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Brugada Syndrome is the syndrome of pseudo- right bundle branch block, ST segment elevation in V1 to V3 and sudden death. It is a clinical-electrocardiographic diagnosis based on syncopal or sudden death episodes in patients with a structurally normal heart. the disease is responsible for 4 to 12 % of unexpected sudden deaths. We present a case of Brugada syndrome diagnosed after 15 years of the first episode of sudden cardiac death. We present a case of a 47 years old Turkish male with past medical history significant for cardiac arrest of unknown etiology s/p AICD placement 15 years ago, history of retractile mesenteritis diagnosed in 1997 after he presented with an acute abdomen necessitating an extensive small bowel resection and causing subsequently a short bowel syndrome and chronic electrolytes disturbance. The patient was admitted to our institution for 5 days history of diarrhea and fever. At admission his serum magnesium was 1.5, so he was given an infusion of two grams of magnesium sulfate over 2 hours. Few hours later he developed recurrent episodes of polymorphic ventricular tachycardia terminated each time by a shock from his defibrillator. The arrhythmia was suppressed by the administration of Amiodarone and Lidocaine infusions. One of the serial ECG's showed a characteristic pattern of BRUGADA syndrome, which is pseudo RBBB with ST elevation in precordial leads. Adding his history of sudden cardiac arrest and recurrent ventricular arrhythmia to the ECG findings, the diagnosis of BRUGADA syndrome type 1 was confirmed. A variety of factors contribute to the clinical manifestations of Brugada Syndrome including right ventricular abnormalities, mutations in sodium channel gene SCN5A, autonomic tone, and the use of cocaine and psychotropic drugs. Our patient ventricular arrhythmia is most probably due to the electrolytes abnormality and fever. This disease is genetically determined. The incidence of sudden death in this syndrome is very high and, at present, can only be prevented by implanting a cardioverter-defibrillator. The etiology of retractile mesenteritis remains obscure; it includes previous abdominal surgery or trauma, autoimmunity, paraneoplastic syndrome, ischemic injury, and infection. Brugada syndrome is a new entity. We raise the question about the presence of a common factor or Gene mutation that contributed to the manifestation of these two rare syndromes in our patient.

UNUSUAL PRESENTATION OF MIXED CRYOGLOBULINEMIA IN A HEPATITIS-C NEGATIVE PATIENT

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Cryoglobulinemia is an immune-complex mediated disease characterized by immunoglobulins that undergo reversible precipitation at low temperature. Three types of cryoglobulinemia are described. Type I is the result of a monoclonal immunoglobulin. Type II and III (mixed cryoglobulinemia) are associated with rheumatoid factor activity; the latter may be monoclonal (Type II) or polyclonal (Type III). Mixed cryoglobulinemia is associated with hepatitis C virus (HCV) in majority of cases (>80%) and usually presents with purpura, myalgia and arthralgia although symptomatology may be heterogeneous. We present a case of an HCV negative mixed cryoglobulinemia presenting with acute respiratory failure. A 29-year-old Hispanic female, with past medical history of mild mental retardation and seizure disorder secondary to childhood meningitis, presented to our institution with two weeks history of worsening shortness of breath, productive cough and abdominal pain; this was associated with a few episodes of vomiting and reddish urination for the past few days. Physical examination revealed hypertension, tachycardia and decreased breath sound at bilateral lung bases. The chest x-ray showed bilateral pleural effusion. Abnormal laboratory results included creatinine 2.9mg/dl, hypoalbuminemia (2.6gm/dl), hypocomplementemia with selective depression of C4 level, a high ESR (105 mmhr) and elevated rheumatoid factor (395 IU/ml). Urinalysis revealed 3+ blood with RBC casts and severe proteinuria (8.27gm/24 hr). Trans-thoracic echocardiography was unremarkable. Kidney ultrasound and renal artery duplex were normal. On the 2nd day of admission, the patient became increasingly dyspneic and had to be intubated; she subsequently remained on a mechanical ventilator for 3 days and was initially treated with antibiotics. On the 7th day of admission, she developed maculo-papular, purpuric skin rashes on bilateral lower extremities; the skin biopsy specimen showed a leukocytoclastic vasculitis. The viral screening was negative for hepatitis B and C by serology as well as by PCR. The serum was negative for anti-nuclear antibody (ANA) and positive for trace cryoglobulin S. A kidney biopsy was performed which revealed immune-complex mediated mesangial and endocapillary proliferation with ultra-structural features suggestive of cryoglobulinemic glomerulonephritis. A diagnosis of hepatitis-C negative mixed cryoglobulinemia with glomerulonephritis was made and treatment started with prednisone and azathioprine. Her kidney function improved dramatically and she was discharged on the same medications. At two months follow-up, she was normo-tensive with normal renal functions. Mixed cryoglobulinemia is a systemic vasculitis characterized by rheumatoid factors which form complexes with Fc portion of polyclonal IgG. It is usually associated with viral infections (particularly HCV) and chronic inflammatory conditions such as systemic lupus erythematosus (SLE) and Sjogren's syndrome. Common clinical manifestations include purpura, arthralgia, weakness, Raynaud's phenomenon and nephrotic syndrome. Our patient presented with respiratory symptoms in association with nephrotic syndrome and purpuric skin lesions in an HCV negative background.

TRINITAS REGIONAL MEDICAL CENTER

MEMBRANOUS NEPHROPATHY WITH HEAVY PROTEINURIA AND SPHERULES ON ELECTRON MICROSCOPY

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Membranous nephropathy(MGN) remains the most common cause of idiopathic nephrotic syndrome in white adults. One-third of cases go into spontaneous remission while 40% reach end-stage renal failure in 10 years. Kidney biopsy will reveal thickened glomerular basement membrane, spikes on silver/PAS stain and subepithelial deposits on electron microscopy(EM). Immunofluorescence microscopy will show granular Immunoglobulin along the capillary walls. Few case reports of MGN described unique subepithelial accumulations of an unusual type of immune deposit composed of spherular structures. The identity of such structures as nuclear pores has been suggested, but not established. A 54-year old Hispanic male was admitted because of pleuritic chest pain for 2 days with worsening bipedal/hand edema. Physical exam showed BP was 195/115 with grade 3 bipedal pitting edema, bilateral hand swelling, right calf muscle atrophy from childhood poliomyelitis, periorbital edema and decreased breath sounds on both lung bases. A CT scan of the chest showed minimal bilateral pleural effusion and was negative for pulmonary embolism. Chemistry showed BUN of 14 mg/dL, Creatinine 1.70 mg/dL, Albumin 1.2 mg/dL and hyperlipidemia. Urinalysis showed proteinuria, glucosuria and microscopic hematuria. His 24-hour urine protein was 31,320 mg. RA screen, Anti-GBM, Complement levels, Hepatitis serology, ANA were unremarkable. He was treated with Enalapril, Furosemide, Gemfibrozil, Hydrochlorothiazide, Metoprolol and Simvastatin. Renal biopsy showed stage 2-3 Membranous GN with microspherules and focal segmental sclerosing features. Patient was seen at the outpatient nephrology clinic and was offered the Ponticelli regimen consisting of alternating monthly Prednisone/methylprednisolone and cyclophosphamide for 6 months. Currently, he is closely being followed at the clinic and will start the regimen as soon as he agrees to. MGN with spherules is an uncommon variant with obscure pathogenesis. Few case reports described this entity and as of now no specific clinical association was identified and the clinical features and course appears similar to classic MGN. In a cohort of patients identified by Kowaleska et.al., Fourteen patients with MGN and spherules were reviewed. The molecular identity of these spherular structures as nuclear pores was tested using immunofluorescence microscopy and immunohistochemistry with mouse monoclonal antinuclear pore antibodies and anti-Nuclear Pore-O-Linked Glycoprotein antibodies. This supported the case reported by Dales and Wallace which suggested, based on the morphology and size of the spherules, that they might be nuclear pores. These cases form a rare, but distinctive, morphological subclass of membranous glomerulopathy. The antigenic specificity of immune deposits in these cases remains elusive and further characterization of this molecular entity needs to be done and correlated clinically if applicable.

ILIACUS MUSCLE ABSCESS IN AN INTRAVENOUS DRUG USER, A VERY RARE CONDITION: CASE REPORT

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Background: Currently considered a rarity in medical literature with reported worldwide incidences varying from 12 to approximately 16 cases per year, iliopsoas muscle abscesses represent a challenge for the clinician. Virtually all cases reported affect the psoas muscle with or without affecting the iliacus muscle, which shares a common distal tendinous insertion. Primary hematogenous origin is at present one the more common etiologies, with *S. aureus*, as the main pathogen isolated. Contiguous spread and secondary phenomena from intra-abdominal infections also have significant incidence. Here we report a case of isolated primary iliacus muscle abscess associated to intravenous drug abuse (IVDA) of opiates in a 38 year-old African-American female who presented with a five-day history of severe left lower limb pain, fever, left lower abdominal quadrant pain and prostration. Patient reported injection only in upper extremity veins, and there was no evidence of injection in the lower limbs. The patient had simultaneously active untreated skin psoriasis and Hepatitis C infection. Physical exam revealed a temperature of 102.4° F, normal heart and lungs, and tenderness of the left lower abdominal quadrant without rebound or mass. There was limited range of motion in the left hip joint, severe pain to flexion of the thigh without motor or sensory deficits. An initial clinical impression of left iliopsoas abscess was confirmed on abdominal CT (12 x 17mm collection). Dorso-lumbar spine MRI was ruled out paravertebral or epidural abscess. Blood cultures revealed Methicillin-Sensitive *S. aureus* on specimens of 3 consecutive days. Initial blood counts showed leukocytosis with neutrophilia, elevated erythrocytation rate, and C reactive protein. HIV serology was negative. Transesophageal echocardiogram was negative for endocarditis. Initial treatment with vancomycin and piperacillin, which was changed later to cefazolin (1 g 8-hourly) plus gentamycin, produced rapid clinical improvement. No surgical intervention was required in our case. Prolonged course of antibiotics and physical therapy provided an almost full recovery after 3 weeks.

Conclusions: A high index of suspicion is required to diagnose this rare but increasing entity. HIV and IVDA epidemics account for some of the increase in incidence. Iliopsoas Abscesses are more frequently located superiorly in the abdomen, affecting the psoas major muscle. Ratios male to female of 2:1, and right vs. left of 3:1 have been described in other series. Conservative antibiotic treatment is effective for the management of small deeply localized abscesses where percutaneous drainage is perhaps more risky. To our knowledge after literature review, this is the first reported case of isolated iliacus muscle abscess associated to IVDA. **KEYWORDS:** iliacus abscess, ilio-psoas abscess, iliopsoas abscess, IVDA, Intravenous drug use, Heroin abuse, *Staphylococcus aureus*, pyomyositis.

PULMONARY TUBERCULOSIS IN AN IMMUNOCOMPETENT HOST WITH ATYPICAL RADIOGRAPHIC FINDINGS

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Introduction Pulmonary tuberculosis (TB) is generally thought of as a disease seen in recent immigrants from endemic regions of the world or in those with chronic debilitating conditions causing significant immunosuppression. However, there is a sub-segment of the population, who are otherwise healthy, lack risk factors for TB and present with mild, protean symptoms, where a high index of suspicion for TB, requiring a thorough work up is warranted. Any hospital that subserves a large immigrant population who originate from regions where TB is common will find itself faced with a range of presentations of this illness. Indeed a high index of suspicion for TB is always warranted when faced with cough and a peculiar infiltrate on x-ray. However, what made this case of interest was the disconnect between our patient's mild symptomatology, his presence in the U.S. for the last 18 years, lack of apparent risk factors (including HIV seronegativity) and sick contacts, his striking radiographs and initial negative TB work up. **Case Report** The following case describes an atypical clinical presentation of pulmonary TB in an otherwise healthy, foreign born young man. He is a 23 year-old male from El Salvador who presented with 2 months history of mild non-productive cough accompanied initially by nasal congestion and sore throat. He denied any fever, weight loss and night sweats. He immigrated from El Salvador in 1990 where he had lived and worked on a farm, farming mostly corn and grains. He denied any significant illnesses in El Salvador, exposure to tuberculosis or recent sick contacts of any kind and denied any allergies. Chest radiography demonstrated impressive bilateral granulomatous appearing infiltrates and consolidations, which evoked a wide differential diagnosis. Initial work up, including PPD testing and sputum AFB were negative. Bronchoscopy was performed and after 30 days of incubation the bronchial washing culture specimen turned positive for AFB. DNA probe testing confirmed the specimen to be *M. tuberculosis* and appropriate treatment was instituted. **Conclusions** Atypical presentations of pulmonary TB in young, healthy, immunocompetent foreign-born individuals exist. These cases can be challenging as a high index of suspicion for TB is required in the setting of mild symptomatology and atypical radiographic findings. Often invasive diagnostic testing, including bronchoscopy may be needed to confirm a diagnosis so that appropriate medical treatment can be instituted and measures taken to prevent further spread of a potential fatal disease."