NYACP
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# 2015

# New York Chapter ACP

# Annual Scientific Meeting

# Poster Competition

**Friday, February 6, 2015**

**Renaissance Westchester Hotel**
**80 West Red Oak Lane**
**West Harrison, NY 10604**

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# Medical Student

# Clinical Vignette

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# Medical Student Clinical Vignette

Author: **Cindy Agu**

Additional Authors: Nabeeh Hauter ,
Virginia Auguste,MD
Saqif Hasan,MD

Institution: Kingsbrook Jewish Medical Center

**Title: HIDDEN COLONY OF MUCORMYCOSIS**

Introduction:
Mucormycosis is a rare fungal infection that can be found in immunocompromised patients and usually acquired through the inhalation of spores. Mucormycosis species are angioinvasive and infarction of infected tissues in a hallmark of the disease. There are four types of mucormycosis infections; they include rhinocerebral, pulmonary, gastrointestinal and cutaneous. Pulmonary mucormycosis infections are more commonly seen in patients with blood cancers, diabetics, organ transplant recipients, IV drug users and AIDS patients. Patients with pulmonary mucormycosis most often times present with fever and severe hemoptysis. Here we report a case of an asymptomatic HIV patient with a suspicious lung mass, which, turned out to be pulmonary mucormycosis with a concomitant pneumocystic carinii infection.

Case Presentation:
A 45 year old incarcerated male with a history of substance abuse, shared needles, unprotected sexual intercourse, hepatitis B and recently diagnosed HIV (CD4 181) on Highly Active Anti-Retroviral Therapy (HAART), was admitted for the evaluation of a suspicious lung mass found on routine x ray. At time of admission, the patient was asymptomatic and denied fever, cough, weight loss, night sweats and anorexia. Computed Tomography (CT) of chest in the ED showed a right hilar lung mass measuring about 6.4 cm x 3.6 cm suggestive of T3N1M0 stage IIIa lung cancer. The patient underwent bronchoscopy and results showed a small area of necrosis in the posterior wall of the right middle lobe. Bronchial washing showed numerous aggregates of neutrophilic exudates in the background of necrosis and no tumor cells were observed. Periodic Acid Schiff (PAS) and Silver stains were positive for pneumocystic carinii and fungal organisms consistent with mucormycosis. To rule out Mycobacterium, an Acid Fast Bacilli (AFB) stain was done and results were negative. During his hospitalization he was treated with intravenous Amphotericin B, Caspofungin and oral Bactrim. He was discharged to acute rehab with advice to continue PCP therapy for 21 days and to continue treatment for mucormycosis until radiological improvement is noted.

Discussion:
The presentation of the suspected mucormycosis infection in this immunocompromised patient was not of the norm. As stated in the case presentation, on admittance the patient appeared to be in good health and was thought to have an invasive lung cancer. This case proves to be interesting because an immunocompromised patient with a mucromycosis infection at the stage in which the patient arrived in, should be severely ill and in possible distress. A case such as this is one that should be kept in the minds of physicians, as illnesses do not always present in the same way.

Author: **Matthew Basciotta**

Additional Authors: Kellis N; Mousa O, MD; Patel A, MD; Kahlon A, MD; and John S, MD.

Institution: SUNY Upstate Medical University

**Title: FIRST REPORTED CASE OF CRYSTAL METH-INDUCED ISCHEMIC HEPATITIS**

Amphetamine use has been previously associated with acute liver injury. Clinical courses described are similar to those observed in ischemic and hyperthermia induced liver injury. It has been proposed that the mechanism of injury involves ischemia as well as an unknown toxic metabolite. This presents a serious risk as hypoxic hepatitis has been shown to increase intensive care unit mortality rates by 4 fold. Case reports describing synthetic methamphetamine (Crystal Meth) induced hepatitis have not been previously reported. We present a 39 year old female who had a two week history of respiratory distress, nausea and severe abdominal pain. Past medical history included type I diabetes mellitus, end stage renal disease on hemodialysis, and ischemic cardiomyopathy. Review of medications was not suspicious for hepatotoxic drugs. There was no history to suggest a recent clinical illness or episode that could lead to hypotension or systemic hypoperfusion. At the time of presentation, she denied illicit drug use including cocaine. On exam she was agitated, had abdominal tenderness localizing to the epigastrium, and was without jaundice. Abdominal ultrasound and cholescintigraphy were negative. Doppler sonography of the abdomen showed patent hepatic, portal and mesenteric veins. Laboratory tests were significant for abnormal liver panel (AST 6122 IU/L, ALT 3809 IU/L, LDH 7979 IU/L, albumin 4.1 g/dL, total bilirubin 2.3 mg/dL and INR 2.41). Testing for other causes of hepatic injury was negative including viral hepatitis, autoimmune hepatitis and serum acetaminophen level. Empiric treatment was begun with N-acetylcysteine, intravenous fluids and pain control. After further questioning, the patient admitted to use of Crystal Meth for the first time in her life two weeks prior to her presentation. The patient left against medical advice on day 3 with some improvement of her symptoms and improving liver function panel (AST 3963 IU/L, ALT 1865 IU/L, LDH 6626 IU/L, total bilirubin 0.8 mg/dL and INR 1.56). This is a rare case of synthetic methamphetamine-induced hepatitis. Conservative management with close monitoring improved the status of our patient without the need for liver transplantation. It is imperative to raise awareness regarding this complication associated with use of Crystal Meth among the general population and healthcare professionals.

# Medical Student Clinical Vignette

Author: **Amruta Desai**
Additional Authors: Richard Maleski, MD
Institution: Peconic Bay Medical Center, Riverhead, NY

Institution: NYIT College of Osteopathic Medicine

## Title: A PUZZLING CASE OF SARCOIDOSIS

BACKGROUND:
Sarcoidosis is an inflammatory condition of an unknown etiology that is characterized by noncaseating granulomas and often has multisystem involvement. Epidemiologically, sarcoidosis has been found to be more prevalent in African Americans, females, and between the ages of 10-40 years. Affected individuals most commonly present with symptoms of lung involvement seen on imaging and extrapulmonary manifestations that include the skin and eyes. Other organ systems can be involved, but with much lower incidences including direct renal involvement, which occurs in <5% of patients. We report an atypical presentation of sarcoidosis with associated acute renal failure in an older Caucasian male.
CASE:
A 50 year old Caucasian male with a past medical history of glaucoma, uveitis, iritis and lyme disease presented with fatigue, constipation, polyuria, worsening of shortness of breath and a weight loss of twenty pounds. The physical exam was non-contributory except for mild distress. Patientâ€™s labs were significant for hypercalcemia (12.6mg/dl) and acute renal insufficiency (BUN: 38 mg/dl; creatinine: 2.7mg/dl), and thus intravenous fluids were started at 100cc/hr. Initial work up for malignancy and multiple myeloma was unremarkable. Renal sonogram was ordered to rule out obstruction and showed fullness of the right kidney and non-obstructing stones in the left kidney. Chest x-ray showed no acute findings but a non-contrast computed tomography (CT) chest showed multiple mediastinal lymph nodes with no hilar lymphadenopathy, scattered right-sided pleural and sub-pleural nodules. Since the patient refused biopsy of the lymph nodes and kidney, an angiotensin-converting enzyme (ACE) level was determined given sarcoidosis being high on the differential at this point and the patientâ€™s history of uveitis. The level was found to be high at 125 U/L. The parathyroid hormone and Vitamin D1 25(OH)2 levels were low and high at 4 and 131 pg/ml respectively. For the patientâ€™s shortness of breath, prednisone was started and pulmonary function tests were conducted, which were determined to be normal. After the administration of intravenous fluids and prednisone, the patient's renal function had improved and the calcium level normalized. Therefore, the patient was given the diagnosis of sarcoidosis and was recommended for outpatient follow up with a nephrologist and pulmonologist.
DISCUSSION:
This cases diagnosis of sarcoidosis was made difficult due to extrapulmonary involvement, lack of common diagnostic indicators, and uncharacteristic demographic findings. The discussed patientâ€™s pulmonary symptoms without the typical diagnostic findings of bilateral lymphadenopathy as well as the hypercalcemia associated acute renal failure are rare findings. These atypical findings create a diagnostic challenge but sarcoidosis should be considered as this case demonstrates.

Author: **Junaid Habibullah**

Additional Authors: Omar Mousa, MD, Amit Dhamoon, MD, PhD

Institution: SUNY Upstate Medical University

## Title: A Clue in the Canal

Title: A CLUE IN THE CANAL
Authors: Junaid Habibullah, MS, ACP Member, Omar Mousa, MD, Amit Dhamoon, MD, PhD
All authors from SUNY Upstate Medical University Syracuse, NY
Abstract
Ramsay Hunt Syndrome is a unique presentation of herpes zoster that is classically defined as facial nerve palsy with a vesicular rash located in the ear canal, auricle and/or mucous membrane of the oropharynx. There is a clinical variation of the rash, in terms of the number of vesicles and the timing of their appearance. This diversity in the presentation makes a simple diagnosis challenging.
A previously healthy 43-year-old male presented to the hospital with nausea, vomiting, photophobia, nuchal rigidity, vertigo, headaches and subjective fevers for one week and right-sided facial weakness, tinnitus, unilateral hearing loss for 2 days. The physical exam showed a hemorrhagic vesicle located in the ear canal on otoscopic exam, right facial paralysis and right sensorineural hearing loss. The patient underwent a lumbar puncture with cell count, gram stain, and culture and viral serologies for common causes of meningitis and encephalitis were sent. The CSF was pink and hazy with a WBC count of 568 mm3, 100% monocytes and 111 mg/dl of protein. The patient was started on empiric treatment with intravenous Acyclovir, Vancomycin, Ceftriaxone and dexamethasone for bacterial and viral meningitis as well Lyme disease. Other viral serologies were negative. LP was repeated prior to his discharge to rule out rare causes of encephalitis given that the previous viral serologies were negative. CSF cultures were positive for Varicella and serology also showed an abnormally high level of VZV antibodies.
The presence of a rash is not an absolute criterion for a diagnosis of Ramsay Hunt Syndrome. A previous study of 274 adult patients showed that 88.3% presented with herpetic vesicles. Although a serological workup can support the diagnosis, a high clinical suspicion and a meticulous physical exam can establish a diagnosis and a treatment plan for a patient with suspected Ramsay Hunt Syndrome. Even though the benefit of antiviral therapy is controversial, early initiation within 3 days can lead to complete recovery from facial paralysis in 75% of patients. Cognizance of this rare disease and its variable presentations saves medical resources, improves outcomes and decreases hospital length of stay.

# Medical Student Clinical Vignette

Author: **Nabeeh Hauter**

Additional Authors: Cindy Agu;
Saqif Hasan,MD, Malik Malik,MD

Institution: KINGSBROOK JEWISH MEDICAL CENTER

## Title: MY WHOLE BODY HURTS: A CHIKUGUNYA VIRUS INFECTION.

Introduction: Chikungunya is a virus that is passed on to humans by the aggressive daytime biters the Aedes albopictus and Aedes aegypti mosquitoes. The virus, which is a single stranded RNA virus, belongs to the Togaviridae family. Since its debut in Tanzania in 1952, Chikungunya has been identified in over 40 countries around the world. The majority of cases reported in the continental United States have been imported cases. Here we report, A Haitian female with recent travel history, who presented with urinary symptoms with debilitating generalized joint pain diagnosed to have sepsis from urinary infection with concomitant chikungunya virus infection.
Case Presentation: A 75 year old female presents to the Emergency Department (ED) complaining of generalized joint pain for two weeks. She had constant achy pain involving the large and small joints associated with fever, chills, nausea, generalized weakness and urinary symptoms. Her symptoms started during her recent stay with family in Haiti. She states that, two of her family members and a number of her neighbors had similar symptoms of fever associated with generalized weakness. Her past medical history was significant for diabetes mellitus and hyperlipidemia. Physical examination was unremarkable. No petechiae, organomegaly, joint swelling or tenderness were appreciated. Laboratory investigation showed, high leukocyte count (26.6) with left shift, predominant neutrophils and normal platelet. Urinalysis was positive for nitrate and white blood cell. Urine culture showed colony of E coli. She was hospitalized and treated with intravenous antibiotics. However, as the patient had persistant generalized arthralgia and bodyache with recent travel history other infectious etiology was suspected. Reverse Transcriptase PCR (RT-PCR) for Chikungunya returned positive. A week later after supportive care and antibiotics for the Urinary Tract Infection (UTI) the patient showed improvement and was discharged.
Discussion: Chickungunya is a fairly uncommon virus in the US. Without a suspicion of its presence symptoms can be mistaken for more common bacterial or viral infection. Though lymphopenia, thrombocytopenia are frequently seen, they are not invariably presents. In our patient the presence of an E-coli UTI masked the underlying chikungunya infection. It was patientâ€™s travel history and complaints of persistant joint pain that made us suspect chikungunya. The case illustrate a scenario where in two infection can exist side by side one masking the other. A patient with history of travel from endemic area who presents with a clear evidence of bacterial infection may also be infected with chikungunya. The later should be suspected, if joint pains are out of proportion to what one might expect from bacterial pneumonia or UTI.

Author: **Adam Johnson**

Institution: FHMC

## Title: Levamisole-Cocaine Induced Agranulocytosis

Introduction: Severe insidious agranulocytsis is a cause for alarm and has many etiologies. When a patient presents with signs and symptoms of immunodeficiency, drug use may be on the differential. Cocaine laced with the veterinary anti-helminthic agent, levamisole, has a rare side effect of severe agranulocytosis. It is important for health care workers to be aware of this cause of extreme agranulocytosis so we can treat effectively and educate on its dangers.
Case: A 30 y.o. female presents to the ER with sore throat, mucous stools approx. 20 times daily, 30lb unintentional weight loss and tactile fever at home. Other symptoms include headaches, fatigue, cough and myalgias. These symptoms had been reported as gradually getting worse over the last 2 weeks. Patient later mentions h/o regular cocaine use, most recent 5 days ago and seen 1 month ago for herpetic lesions to lips. On Review of systems, no chest pain, shortness of breath, abdominal pain, or rash. Physical examination showed an ill appearing patient with dry, erythematous oropharynx, scabbed herpetic lesions to left oral commissure, anterior cervical lymphadenopathy, tachycardia, and decreased breath sounds. Initial work-up showed HIV negative, CXR negative, EKG with no acute ST-T wave changes, NSR, throat culture negative for strep, WBC of 0.8 (x10E3/UL) and UA positive for cocaine and eventually levamisole. Normal WBC is between 4.0 and 10. Patient was given a course of 1 g IVPM Vancomycin, 1g and 2 g IVPM Cefepime, 300mcg injection Filgrastim and 100mg and 200mg Fluconazole tablets over the course of her 35 day hospital stay.
Discussion: Before levamisole was first withdrawn from markets in 1999, it was used as an anti-helminthic agent in humans and mostly animals. Possibly due to levamisoleâ€™s similar chemical properties, it was found by the DEA to be cut with more than 80% of seized cocaine in 2011. The dangers of levamisole are that it can cause agranulocytosis and also vasculitis, or, â€˜levamisole induced necrosis syndromeâ€™, where the patient presents with erythematous painful papules anywhere on the skin. It is interesting and still unexplained why many patients test positive for levamisole but very few, less than 5% of the studied population, experience serious illness. In the case of our patient, she suffered severe agranulocytosis and recovered almost immediately upon stopping her drug use and starting medications. Within 24 hours her WBC was at 3.8 and 9.5 by discharge. There are several articles detailing the few recorded cases of patients that used cocaine or crack cocaine that presented in an immunocompromised state. With the knowledge of adulterated cocaine we can be aware of other etiologies of agranulocytosis, we can rapidly alleviate patient symptoms and improve healthcare through the education of the dangers of drug use.

# Medical Student Clinical Vignette

Author: **Shian Liu**

Additional Authors: Olivia Fu, M.D., Charles Okamura, M.D.

Institution: New York University School of Medicine

**Title: A positive HIV ELISA during a profound immune response: a true blue?**

Enzyme-linked immunosorbent assays (ELISA) for HIV, is an excellent diagnostic test, with sensitivity and specificity greater than 99%. A false positive ELISA test is rare, occurring at a rate of 1 in 135,187 in a low risk patient population. Causes include rheumatologic disorders, infections, and disorders of immunoglobulins.

This is a case of a 30-year-old woman with no significant past medical history who presented with 2 days of malaise, chills, and tender anterior cervical lymphadenopathy. She had undergone an elective breast reduction surgery 6 weeks prior to admission. She has no HIV risk factors and a negative HIV ELISA on preoperative testing. Her surgery was uncomplicated; she received no transfusions. Post-operatively, she developed surgical dehiscence that did not improve after a course of trimethoprim/sulfamethoxazole. Examination was significant for a temperature of 104.5, and a 1.5cmx0.5cmx1 cm area of dehiscence with purulent exudate, induration, and blanching erythema upon palpation. CBC revealed a white count of 3,600/mcL with a normal differential, and an HIV ELISA was positive. Empiric therapy with IV vancomycin was begun for left breast cellulitis, which was later switched to IV ampicillin/sulbactam after an adverse reaction. HIV ELISA was repeated 6 hours later and again positive, so the team proceeded with an HIV work-up. On hospital day 3, her HIV quantitative PCR came back negative and white count normalized to 4.8. Western blot was also negative for HIV protein and CD4+ count was normal. Urine and blood cultures were negative, and gram stain from her wound culture was negative. Given she had no risk factors for HIV except for her breast reduction surgery, interpretation of her HIV panel was tentatively HIV-negative and she was advised to have a repeat ELISA and PCR in 3 weeks.

Surgery and health-care transmission of HIV, has only happened in one reported case of the literature and is not considered a risk factor. Her false positive test was attributed to her highly inflammatory state secondary to cellulitis.

ELISA works by linking the HIV antibody between the walls of wells coated with HIV antigen and a chromogenic molecule that changes color with a provocative solution. If a patientâ€™s serum has HIV antibody, it will bind the antigen, and the liquid in the wells changes to the color blue. Over 99% of the time, the HIV immunoglobulin binds the HIV antigen in the ELISA test. However, other conditions which produce high levels of immunoglobulins can bind the HIV antigen coated wells, causing a color change and false positive tests.Key takeaways: ELISA is a qualitative test (binary: blue or clear). The HIV ELISA test is highly accurate, but should be interpreted in the context of the patients HIV risk and conditions that could effect immunoglobulin production.

Author: **Amy Spallone**
Additional Authors: Ellis Tobin, MD (1,2), and Sudha Chaturvedi, Ph. D. (3)
1) Department of Internal Medicine, Albany Medical College, Albany, NY
2) Department of Internal Medicine, Division of Infectious Disease, Albany Medical Center, Albany, NY
3) The Mycology Laboratory, Wadsworth Center, New York State Department of Health, Albany, NY
Institution: Albany Medical College

**Title: FUNGUS AMONG US: A CASE OF CENTRAL NERVOUS SYSTEM BLASTOMYCOSIS IN A NEW YORK STATE RESIDENT WITHOUT TRAVEL TO KNOWN ENDEMIC AREA**

Blastomyces dermatitidis is a dimorphic fungal pathogen thought to be endemic to regions along the Ohio and Mississippi River basins, Great Lakes, and St. Lawrence River, including regions of western New York State (NYS). The lungs are the primary route of infection due to inhalation of conidia. The spectrum of disease ranges from subclinical infections to potentially fatal systemic dissemination. Single organ involvement with B. dermatitidis has been demonstrated in 69-80% of cases, most commonly isolated to the lungs. Extrapulmonary dissemination often includes the skin, bone, and genitourinary systems. Involvement of the central nervous system (CNS) occurs in 5-10% of disseminated cases. There are rare reports of isolated CNS blastomycosis. We report a case of isolated CNS blastomycosis in a NYS resident without travel history to a known endemic region.

A 59-year-old Caucasian female with a history of hypertension, osteoarthritis, and no history of perceivable immunocompromise presented with two months of vertigo. She was experiencing difficulty with balance, but denied headaches, nausea, vision changes, or difficulty with speech and movement. Magnetic resonance imaging revealed a 3-centimeter lobulated mass within the right temporoparietal lobe with significant surrounding vasogenic edema. Differential diagnosis included primary and metastatic neoplasm and abscess, for which the patient was seen by neurosurgery. Operative dissection revealed a firm, pedunculated mass that contained purulent material. A specimen submitted for Gramâ€™s stain revealed few polymorphonuclear leukocytes and no organisms. Remaining tissue was submitted for histopathology, which showed necrotizing granulomatous inflammation containing broad-based budding yeasts suggestive of CNS blastomycosis. Liposomal amphotericin B (LAB) was immediately initiated. Culture of brain tissue ultimately grew small, white fungal colonies on day seven of incubation. The culture was confirmed as B. dermatitidis by conventional and real-time PCR assays and by its conversion to the yeast form on cottonseed agar at 37°C. Additionally, the formalin-fixed paraffin embedded tissue was positive for B. dermatitidis DNA by real-time PCR assay. B. dermatitidis was also identified by urine antigen test. After eight days, LAB was discontinued due to nephrotoxicity and oral voriconazole was initiated. No other anatomic focus of infection was identified. Detailed travel history revealed no travel to known endemic regions within the past 12 months. At three months of follow up, the patient is doing well and has returned to her normal activities of daily living. CNS blastomycosis is an uncommon manifestation of disseminated disease and isolated CNS blastomycosis is exceedingly rare. Blastomyces dermatitidis should be recognized as an emerging pathogen outside the confines of its traditional geographic distribution. This case, as well as other sporadic case reports from NYS, should prompt reconsideration of the endemicity of this organism.

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# Medical Student

# Research

**Friday, February 6, 2015**

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Author: **Lu Chen**

Additional Authors: BHAIRAVI SHESHADRI, MD
ED CAPIRO, LIJO ILLIPPARAMBIL MS
PARAG MEHTA, MD

Institution: NEW YORK METHODIST HOSPITAL

**Title: RACING AGAINST THE CLOCK - INTERNAL MEDICINE RESIDENTS' USE OF ELECTRONIC MEDICAL RECORDS**

Purpose: Electronic Medical Records (EMR) plays an increasingly significant role in patient care today. A common complaint from both physicians and patients is that physicians spend more time on the computer instead of the patient. The purpose of this abstract is to identify and analyze the time spent using EMR by Internal Medicine (IM) residents in our hospital.

Methods: Active EMR usage data was collected from the EMR user database for IM residents from 5/1/2014-5/31/2014. With the exception of a few tablet devices, active EMR use time was mostly spent at a computer station away from the bedside. Active EMR use was defined as more than 3 mouse clicks per minute or more than 1,700 mousemiles per minute or more than 15 keystrokes per minute. EMR usage activities were divided into Chart Review, Orders, Documentations, and Others. A total of 101 residents (36 PGY1s, 35 PGY2s, and 30 PGY3s) were identified and included in the study. Residents participated in ambulatory, emergency and in-patient care. EMR usage data were sorted by average time residents spent per patient chart. Residents were then divided into two groups based on usage above or below the median. 50 residents who averaged less time per patient chart were termed more efficient users (MEU). Remaining residents were termed less efficient users (LEU).

Results: In a month span, 101 residents accumulated 7,779 hours of active EMR usage over 12,911 individual patient charts. Each resident spent on average 75 hours of active use, averaging 47 minutes per patient chart. PGY1s spent the least amount of time and PGY3s spent the most amount of time (29 vs. 61 minutes) per patient chart. Relative to the total time spent per patient chart, residents spent most of the time on chart reviews (18 minutes, 36-39%), and least on placing orders (8 minutes, 12-19%). Compared to PGY3s, PGY1s used more time placing orders (19% vs. 12%). Compared to LEUs, MEUs were more likely to be PGY1s (66% vs. 6%), have viewed more patient charts (176 vs. 81), and used less time per patient chart in all categories ($p < 0.01$). Per patient chart, MEUs on average saved 15 minutes (61%) on chart review, 5 minutes (52%) on orders, 7 minutes (49%) on documentations and 9 minutes (61%) on other activities (all $p < 0.01$).

Conclusions: Residents spend a significant amount of time actively using EMR. Most of their time was spent on chart review. MEUs saved the most time in chart review, suggesting that chart review time could be reduced. Larger prospective studies are in progress to confirm our results. Emphasis should be placed on reducing time needed for EMR usage to increase time at the bed side.

Author: **Perry Fisher**

Additional Authors: Hamed Nasrabadi1, Marios S. Pattichis2 Dept. of Electrical and Computer Engineering The University of New Mexico Albuquerque, NM 87106, USA 1nasrabadi@ece.unm.edu, 2pattichis@ece.unm.edu A.N. Nicolaides3, Maura Griffin4, G Makris5 Vascular Screening and Diagnostic Centre London, SW7 1AZ, U.K. 3anicolaides1@gmail.com, 4maurabgriffin@gmail.com, 5g.makris09@imperial.ac.uk C.S. Pattichis Dept. of Computer Science University of Cyprus Nicosia, Cyprus pattichi@ucy.ac.cy

Institution: Albany Medical College

**Title: Measurement of Motion of Carotid Bifurcation Plaques**

Atherosclerotic carotid bifurcation plaque rupture is a major cause of ischemic stroke and transient ischemic attacks. It is recognized that this is due to the occlusion of blood vessels by detached fragments of plaque or fragments of a thrombus that has formed on the ulcerated plaque. However, while much is known about the pathology of atherosclerotic plaques, the cause of plaque rupture is not entirely understood. Evidence shows that plaque extrication occurs subsequent to lymphocytic erosion of a plaqueâ€™s fibrous cap. In addition to this, it has been proposed that mechanical forces contribute to the ultimate phenomenon of plaque rupture or ulceration. These mechanical forces are produced by the blood pressure oscillations, blood flow and blood vessel movement throughout the cardiac cycle. It has been suggested that asymptomatic plaques, which do not rupture, have all of their components moving in the same direction as they are influenced by mechanical forces. Conversely, plaques that tend to rupture display uncoordinated movement throughout the cardiac cycle. Therefore, nonsynchronous motion, with different portions of a plaque moving in opposing directions, may be a determinant factor in its symptomaticity â€“ a matter that this ongoing research aims to elucidate and a factor that may be able to predict risk. Using standard ultrasound imaging, ultimately, this research will allow for a non-invasive, easily-accessible, cost-effective method of assessing the utility and risk of intervention such as carotid endarterectomy. Video loops of B-mode ultrasound images of 35 carotid bifurcation plaques were obtained (4 symptomatic and 31 asymptomatic) from patients with carotid bifurcation atherosclerosis. Video loops were classified visually as showing concordant (n=22) or discordant motion (n=13). Concordant plaques were characterized by uniform orientation of motion throughout the cardiac cycle. Discordant plaques exhibited significant spread in motion orientation at different parts of the cardiac cycle, especially at systole. We developed real-time motion analysis software that applies Farnebackâ€™s method to estimate velocities between consecutive video frames, and can be easily utilized in conjunction with standard ultrasound imaging. Over each frame of the B-mode ultrasound video loops, we measured the spread of the motion orientation around the dominant orientation. For each video, we looked at the spreads of the motion orientations for different motion magnitudes. Using these motion-spread measurements, we quantified discordant movement. Motion spread measurements were analyzed in terms of Sum of Maximum Fan Widths (SMFW), a measure derived from pixel motion vectors. A median value of 100 degrees and inter-quartile range (IQR) of (80, 110) degrees was established for the concordant plaques and 270, (230, 430) for the discordant plaques ($P < 0.001$). Therefore, we have a new tool to differentiate between concordant and discordant plaques, and are one step closer to an effective, efficient diagnostic tool.

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## Resident/Fellow

## Clinical Vignette

**Renaissance Westchester Hotel**
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**Author: Diego Adrianzen Herrera MD**

Institution: Mount Sinai St. Luke's - Mount Sinai Roosevelt

**Title: FATAL RECURRENT SICKLE CELL INTRAHEPATIC CHOLESTASIS**

Introduction
Sickle cell intrahepatic cholestasis (SCIC) is a rare and potentially fatal complication caused by sickling within the hepatic endothelium leading to vascular stasis and ischemia, characterized by severe direct hyperbilirubinemia and usually associated with renal failure and coagulopathy. Early exchange transfusion is the best treatment option.

Case description
A 50-year-old African-American male with sickle cell disease (SCD) was admitted for right upper quadrant pain, jaundice and anemia. His history included stage II chronic kidney disease (CKD), secondary hemochromatosis and SCIC 6 months prior to admission. Laboratory initially showed severe anemia, leukocytosis and mild hyperbilirubinemia and transaminitis. He was treated with transfusions, fluids, hydromorphone and hydroxyurea. After 48 hours he suddenly developed worsening pain and abdominal distention, fever, altered mental status, venipuncture site bleeding and acute hypoxemic respiratory failure from pulmonary edema. Laboratory showed new extreme direct hyperbilirubinemia (39.8 mg/dL), rising INR and thrombocytopenia, worsening renal function, stable transaminases and schistocytes and target cells in the peripheral smear. Hepatitis serologies, blood and urine cultures were negative. Ultrasound showed marked hepatomegaly, ascites, right pleural effusion and no extrahepatic cholestasis. He was treated in the ICU with empiric vancomycin and cefepime, diuresis, non-invasive mechanical ventilation, FFPs, vitamin K and cryoprecipitate, strict volume restriction, ursodeoxycholic acid and full automated-exchange transfusion with successful decline in hemoglobin S (HbS) levels from 40% to 12%, however, his coagulopathy, non-oliguric renal injury and volume overload worsened with pericardial effusion requiring pericardial window and hemodyalisis. The hyperbilirubinemia persisted despite a second exchange transfusion. He succumbed to multiorgan failure.

Discussion
SCIC is an unusual complication of SCD easily confused with the self-limited sickle cell hepatic crisis and mistakenly treated with simple transfusions leading to hyperviscosity complications. Extreme hyperbilirubinemia with conjugated fraction higher than 50% is the distinctive hallmark from hepatic sequestration which has a hepatotoxic rather than cholestatic pattern. Coagulopathy, leukocytosis, fever, encephalopathy and renal failure are common. Data shows survival benefit from early exchange transfusion and HbS reduction. We present a male whose first episode was successfully treated but perished despite supportive measures and successful exchange transfusion after recurrence. To our knowledge no other recurrent case has been reported. Record review of the initial episode showed coagulopathy, kidney injury and extreme hyperbilirubinemia that resolved on discharge with recovery of full functionality and baseline creatinine, bilirubin and coagulation profile levels. It is unknown if recurrence is common as no long term outcomes are described in reported cases. In our patient, recurrence occurred at 6 months and showed lower peak bilirubin levels but more severe multiorgan involvement, absent response to exchange transfusion and ultimately a fatal outcome. Older age and comorbidities as hemochromatosis and CKD might have contributed to the poor outcome.

**Author: Mohammed Al-Sofiani MBBS**
Additional Authors: Peterkin Lee Kwen, MD

Institution: University at Buffalo - Catholic Health System

**Title: Isolated medial rectus nuclear palsy as a rare presentation of midbrain infarction.**

Introduction:
Diplopia is a common subjective complaint which can be the first manifestation of a serious pathology. Here, we report a rare case of midbrain infarction involving the lateral subnucleus of the occulomotor nuclear complex presented as diplopia with no other stroke manifestations. We outline the clinical approach taken to identifying the etiology of diplopia including images and video demonstrating the key physical findings in this patient.
Case Description:
83 year old right handed Caucasian male with past medical history of diabetes mellitus, hypertension, dyslipidemia, and coronary artery disease presented to the emergency department (ED) with diplopia and unsteadiness for 2 days. Two days prior to admission, he woke up with mild frontal headache which was dull in nature and had gotten worse since then. He developed constant horizontal diplopia and had unsteadiness which limited his daily activities and led to a fall at home. The patient denied any weakness or clumsiness, nausea, vomiting, photophobia, fever or chills. Ocular exam showed a disconjugate gaze at rest, weakness of the left medial rectus muscle, impaired convergence test and bilateral 3-mm reactive pupils (figure 1)(video 1). The diplopia resolved by closing the left eye. The remaining extraocular muscles and other cranial nerves were normal. There was no nystagmus, ptosis or visual field deficit. Sensation and muscle tone and strength were normal in all extremities. Magnetic resonance image of the brain revealed a tiny focus of restricted diffusion in the left posterior lateral midbrain and an incidental finding of advanced amyloid angiopathy.
Discussion:
An appropriate history and physical examination is essential to diagnose and manage diplopia. The binocular diplopia in our patient limited our differential diagnosis to impaired neural control or function of the extraocular muscles. Horizontal diplopia further shortened the differential diagnosis list to impairment of the medial rectus, lateral rectus or both. The diagnosis of midbrain infarction involving the lateral subnucleus of the occulomotor nuclear complex was confirmed by MRI. Isolated extraocular palsy is usually thought to be caused by orbital lesions or muscular diseases. However, this case exemplifies the importance of systematic clinical approach to elucidate the etiology of diplopia and avoid missing a serious underlying diagnosis such as cerebrovascular accidents.

**Author: Erik Anderson MD**
Additional Authors:Asha Patnaik, MD; Heidi Roppelt, MD

Institution:Stony Brook University Hospital

**Title: THE RS3PE SYNDROME**

Introduction

Remitting seronegative symmetrical synovitis with pitting edema (RS3PE) is a rare inflammatory arthritis that was first described in 1985. Since that time, there have been several case reports describing its association with malignancy. Recognition of this syndrome is imperative since it has an associated malignancy rate reported as high as 54%.

Case Presentation

A 65-year-old African-American man with a remote history of colonic adenocarcinoma (stage II, status-post chemotherapy and resection in 1993) presented with sudden onset bilateral hand swelling and pain, along with bilateral shoulder and hip pain, that persisted for 6 weeks. He described an 11-kilogram involuntary weight loss over that period. He had diffuse pitting edema and tenderness to palpation of both hands. Laboratory results were significant for a negative rheumatoid factor (RF) and anti-nuclear antibody, and mildly elevated acute phase reactants. X-rays did not show erosive changes. A diagnosis of RS3PE was considered and the patient was started on 30 mg of daily prednisone. Computed tomography of the chest/abdomen/pelvis was negative for mass or lymphadenopathy. He was treated with prednisone for 3 weeks with an incomplete response. A colonoscopy performed 5 months later revealed a large polyp (3.3 cm) in the hepatic flexure and a small sigmoid polyp, both negative for carcinoma. The patient subsequently underwent a right hemicolectomy given the high risk of cancer recurrence in the large polyp.

Discussion

RS3PE is a syndrome characterized by bilateral pitting edema of the hands, sudden onset of polyarthritis, age greater than 50, and seronegativity for RF. Seronegative rheumatoid arthritis and polymyalgia rheumatica are important diagnostic alternatives that should be considered. The majority of RS3PE cases have occurred prior to or concurrent with a cancer diagnosis. There are two case reports of RS3PE occurring subsequent to the initial cancer diagnosis; however, these patients had active cancer at the time of diagnosis.

Our case is unique in that the diagnosis of RS3PE was made 20 years after the cancer diagnosis, in the absence of active or recurrent cancer. Also, the majority of RS3PE cases described thus far have associated concurrent malignancy with suboptimal steroid response; however, in this case concurrent malignancy was not present, despite an incomplete clinical response to steroids.

A proposed mechanism for the pathogenesis of RS3PE involves increased production of growth factor (VEGF) and/or cytokine (IL-6), influenced by tumor cells. Glucocorticoids are the mainstay of therapy; tocilizumab, a novel IL-6 inhibitor, has been shown in small studies to be an effective treatment for RS3PE in cases refractory to glucocorticoid therapy.

In conclusion, RS3PE is associated with multiple cancers, but may also present years after malignancy or in the absence of active cancer. However, all patients who present with RS3PE should have an appropriate evaluation for malignancy.

**Author: Erik Anderson MD**

Institution: Stony Brook University Hospital

**Title: OBSTRUCTIVE UROPATHY DUE TO A URETEROINGUINAL HERNIA**

Introduction

Obstructive uropathy caused by incarceration of the ureter secondary to a hernia is rare. When present, the majority of cases are associated with inguinal hernias; however, incarceration is uncommon in these hernias due to their invariably large size. Ureteroinguinal hernia as a cause of hydronephrosis can be included in the differential when a hernia is found on imaging or upon detecting a hernia on physical exam.

Case Presentation

An 87-year-old male with a history of polycystic kidney disease status post bilateral nephrectomies and a living related donor transplant in 2001 presented with fevers and shortness of breath. He had left lower lobe crackles and a reducible right inguinal hernia. He was diagnosed with a lobar pneumonia and treated with intravenous antibiotics. On hospital day 3, he developed an acute kidney injury (AKI), thought to be secondary to antibiotics versus dehydration, and was started on normal saline. He subsequently developed respiratory distress secondary to pulmonary edema. An ultrasound to evaluate progressively worsening AKI revealed moderate to severe hydronephrosis of the right transplant kidney that was new compared to a prior computed tomography (CT) scan of the abdomen/pelvis. It also showed moderate hydroureter with the distal aspect of the ureter not visualized, suspicious for obstruction. A CT cystogram was performed to evaluate for obstruction and revealed marked hydronephrosis and hydroureter, demonstrating reflux into the collecting system. Furthermore, it revealed incarceration of a dilated ureter along with the anterior portion of the bladder within a fat-containing right inguinal hernia. Urology was consulted and the patient underwent a right nephrostomy tube placement. After relief of the obstruction, his creatinine returned to baseline levels. He was scheduled for outpatient ureteral reconstruction and right inguinal hernia repair.

Discussion

Herniation of the ureter into an inguinal hernia is an extremely uncommon scenario; however, if it is missed it can have serious complications. It typically presents as a mass in the groin without urinary symptoms. A recent case report describes a patient with the left ureter present in an inguinal hernia, with associated mild left ureterohydronephrosis, although no mention of incarceration. It appears that incarceration is especially uncommon; however, the present case illustrates the benefit of early recognition.

Nephrostomy tube placement is an important adjuvant treatment that allows relief of the obstruction while at the same time allowing surgical exploration under optimal conditions. Surgical repair involves careful dissection of the ureter free of the hernia, followed by simple reduction of the hernia into the abdomen. The surgeon must be aware of the possibility of ureteroinguinal hernia in order to avoid ureteral injury during hernia repair, which makes recognition of the condition important.

**Author: Arun Kumar Arumugam Raajasekar MBBS**

Additional Authors:Anuradha Belur MBBS, Jay Lipshitz MD

Institution:Maimonides Medical Center

**Title:A RARE CASE OF POEMS SYNDROME WITH BICLONAL IMMUNOGLOBULIN SPIKE**

INTRODUCTION:
POEMS syndrome (polyneuropathy, organomegaly, endocrinopathy, monoclonal protein and skin changes) is believed to be secondary to chronic overproduction of pro-inflammatory cytokines [1]. The median age of presentation is 51 and the majority are men [2]. Polyneuropathy and a monoclonal spike which is almost always lambda chain [3] are essential for diagnosis. We present a patient with POEMS syndrome with a previously unreported bi-clonal immunoglobulin spike.
CASE PRESENTATION
A 71 year old female presented to the clinic with paresthesia of her fingers, dyspepsia and a ten pond weight loss over 3 months. Physical exam was significant for pedal edema. Computed tomography of the abdomen and pelvis showed several sclerotic lesions of the lumbar spine and pelvis and gastric wall thickening. An upper gastrointestinal endoscopy showed the presence of antral gastropathy. Serum vascular endothelial growth factor (VEGF) was four times higher than normal. Serum protein electrophoresis showed a bi-clonal IgG kappa and IgG lambda spike. Bone biopsy of the sclerotic lesion showed the presence of clonal plasma cells with the same biclonal spike. The patient was diagnosed with POEMS syndrome and started on lenalidomide, bortezomib and dexamethasone.
DISCUSSION
POEMS syndrome is diagnosed using the Mayo clinic criteria [4]. The two mandatory criteria are polyneuropathy and a monoclonal plasma cell proliferation. In addition one major (osteosclerotic lesions, Castlemanâ€™s disease and elevated VEGF levels) and one minor criteria (organomegaly, extravascular volume overload including peripheral edema, endocrinopathy, skin changes, papilledema and thrombocytosis or polycythemia) must be present for making a diagnosis. The underlying monoclonal spike on immunofixation almost exclusively involves the lambda light chain [3]. The patient described above is the first documented with a biclonal spike. Treatment is similar to multiple myeloma and may involve radiation, melphlan, dexamethasone, lenolidamide or bortezomib. The estimated five-year survival is 60 percent [5].
KEY POINTS
-POEMS syndrome has a variety of clinical manifestations that may be missed in isolation.
-It is important for the internist to know the protean manifestations of this rare disease which is treated similar to multiple myeloma.
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**Author: Arun Kumar Arumugam Raajasekar MBBS**
Additional Authors:SRIKANT NANNAPANENI MBBS MS,
WILLIAM SOLOMON MD.

Institution: Maimonides Medical Center

**Title:IMMUNE HEMOLYSIS AND ASEPTIC MENIGITIS AFTER INTRAVENOUS IMMUNOGLOBULIN**

INTRODUCTION
Human intravenous immunoglobulin (IVIG) products are generally considered safe and are used for the treatment of a variety of autoimmune and pro-inflammatory states. However, controlled trials involving IVIG have been of small size with limited power and described only the most common adverse effects [1]. IVIG related hemolysis is a rare adverse effect and is usually self-limited, rarely requiring blood transfusions. A few case reports have also described aseptic meningitis after IVIG use. We describe a patient who developed severe hemolytic anemia and aseptic meningitis after IVIG use.
CASE PRESENTATION
A 54 year old woman was admitted with severe head ache, dark colored urine and shortness of breath twenty four hours after receiving IVIG. She was diagnosed with chronic inflammatory demyelinating polyneuropathy (CIDP) a month ago and was started on IVIG. Physical exam was unremarkable. Laboratory tests showed severe anemia with a hemoglobin of 6.1 g/dl (baseline 13 g/dl), indirect hyper-bilirubinemia, low haptoglobin, elevated LDH and hemoglobinuria confirming hemolysis. Hematological testing revealed a positive direct antiglobulin test (DAT), DAT IgG, DAT CD3 and eluate testing were also positive. A lumbar puncture showed neutrophil pleocytosis but no bacteria. A diagnosis of IVIG induced hemolysis and aseptic meningitis was made. O negative packed red blood cells were transfused for severe symptomatic anemia and non-steroidal anti-inflammatory drugs were given for headache. Steroids were deferred since the source of antibodies causing hemolysis was extrinsic. She improved clinically and blood counts returned to baseline in three weeks.
DISCUSSION
Pooled IVIG is extracted from at least 1000 individuals and contains highly purified polyvalent IgG [2]. Hemolysis after IVIG is thought to be secondary to donor antibodies against host red blood cell antigens and resolves once IVIG is stopped. Risk factors for hemolysis include non-O blood types and the use of high dose IVIG. The patient described was of AB Rh positive blood type but did not receive high dose IVIG. Aseptic meningitis is another rare adverse effect that has been postulated to be related to antibodies in the IVIG that mimic antineutophil cytoplasmic antibodies (ANCA) which activate neutrophils causing neutrophilic pleocytosis [3]. The internist must be aware of these two rare complications of a commonly used drug.
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**Author: Sameer Chadha**
Additional Authors:Syed Iman Husain, Shikha Mehta, Elliot Borgen, Gerald Hollander, Robert Frankel, Jacob Shani

Institution:Maimonides Medical Center

**Title:Anomalous Origin of Left Main Coronary Artery from Right Sinus of Valsalva**

Introduction - Coronary artery anomalies are rare, with an estimated prevalence of around 5% [1]. These anomalies vary with respect to number, location, orientation of the ostia, and origin of the coronary arteries. Some anomalies are merely anatomic variants without any clinical relevance; others can present with chest pain, syncope, or sudden cardiac death. 
Case Presentation - A 46 year old male presented to our emergency department (ED) with complaints of sudden onset chest pain while running for a cab. He denied any associated shortness of breath, palpitations or dizziness. His vital signs were stable, and the results of respiratory and cardiovascular examination were normal. An electrocardiogram performed in the ED showed ST segment elevation in leads I, avL and V1 through V5 with reciprocal depression in the inferior leads. The patient was rushed for an urgent cardiac catheterization which revealed narrowing at the ostium of Left Main Coronary Artery (LMCA) that did not resolve after intra-coronary nitroglycerin was administered. However, there was no evident atherosclerotic disease.
To better characterize the lesion, a Coronary CT Angiogram was performed, which showed that the LMCA was anomalously originating at an acute angle from the â€˜rightâ€™ coronary sinus. The CT also highlighted the subsequent malignant course of the LMCA between proximal ascending aorta and the pulmonary trunk. 
The patient was offered corrective surgical repair for this very high-risk anomaly; however, he refused intervention despite aggressive counseling. The rest of his hospitalization was uneventful, and he was discharged in stable condition.
Discussion - Anomalous origin of Left Main coronary artery from the Right Sinus of Valsalva (approximate prevalence, 0.15%) is a subgroup of coronary artery anomalies that has the highest risk for clinical repercussions. The outward expansion of the aortic root and pulmonary trunk during exertion can lead to external compression of the LMCA which can result in acute Myocardial Infarction or Sudden Cardiac Death [2]. The only definitive treatment is surgical repair.
Conclusion - The origin of Left Main coronary artery from the right Sinus of Valsalva is an extremely rare coronary anomaly which can result in acute Myocardial Infarction or Sudden Cardiac Death in young patients. 
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**Author: Andrew Creighton D.O.**
Additional Authors:Usman Khan M.B.B.S., Saad Shariff M.B.B.S., Olaide Akande M.B.B.S.,Stephen Chrzanowski M.D., Henri Woodman M.D., Yubao Wang M.D.

Institution: University at Buffalo - Catholic Health Systems Internal Medicine Training Program

**Title:Shiga-toxin Positive Hemolytic Uremic Syndrome in an HIV-negative Adult Patient with Kaposi Sarcoma and Follicular Lymphoma**

Introduction: Hemolytic uremic syndrome (HUS) is most often seen in a pediatric population and is characterized by neurologic symptoms, fever, anemia, thrombocytopenia, and acute renal failure. Pathophysiologically, a toxin that directly damages endothelial cells can cause HUS. Perhaps the best-characterized infectious agent that damages endothelial cells is Shiga-toxin producing Escherichia coli, most commonly E. coli 0157:H7. However, in 2010 a case series of five adults with HUS in Oklahoma was published. These five adults were part of the largest outbreak of Shiga-toxin producing E. coli 0111 reported in the United States. This case report involves a rare case of HUS caused by Shiga-toxin producing E. coli 0111 in an immunocompromised adult.
Case description: A 69 year-old middle-eastern male who had been diagnosed with Follicular Lymphoma in May 2014 and Kaposi Sarcoma in July 2014 presented in September 2014 with confusion, weakness, and diarrhea for three days. Over the course of those three days, the patient admitted to subjective fever and chills, lightheadedness, decreased appetite, dysuria, and urinary frequency. The patient had completed his second cycle of doxorubicin one week prior for Kaposi Sarcoma, and he noted a visit to Yemen four months prior. Physical exam revealed an afebrile, confused male with a cachectic, dehydrated appearance. Examination of the lower extremities revealed pitting edema, plethoric discoloration, and venous changes. The patientâ€™s feet were desquamated and showed evidence of dry skin along with a 3 x 2 centimeter open biopsy wound of the left foot. Laboratory evaluation revealed an HIV negative patient with a hemoglobin of 10g/dL, platelet count of 53µL, and creatinine of 1.88mg/dL. Haptoglobin level was <30mg/dL. The peripheral smear revealed the presence of schistocytes. The patientâ€™s admission stool studies came back positive for Shiga-toxin 1 and 2, and E. coli serogroup 0111 was isolated. Given these results, the medical team urgently scheduled plasma exchange therapy. Following two weeks of plasma exchange therapy, the patient responded with improvement in platelet count and renal function.
Discussion: Shiga-toxin positive HUS in an adult with E. coli 0111 isolated as the causative agent is rare. This patientâ€™s immunocompromised state after finishing chemotherapy may have increased his susceptibility to E. coli 0111 infection. The Department of Health was notified as this could be a potential index case for infectious outbreak. The rapidity of this patientâ€™s decline underlies the urgency required for treatment when HUS is suspected.

**Author: Shilpa Deshmukh MD**
Additional Authors:Neville Jadeja, Vinuta Mohan MD, Tasneem Zahra MD

Institution:Lincoln Medical and Mental Center

**Title: A RARE CASE OF SERTOLI LEYDIG CELL TUMOR OCCURRING IN A PATIENT TREATED FOR PAPILLARY THYROID CANCER.**

INTRODUCTION: Sertoli Leydig cell tumors (SLCT) are extremely rare tumors which account for about 1% of sex cord stromal tumors and less than 0.2% of all primary ovarian neoplasms. A few cases in literature describe the occurrence of SLCT along with thyroid adenoma. There is one case reported about SLCT occurring with papillary thyroid cancer. We here describe the second case where SLCT was found in a woman with history of papillary thyroid cancer.
CASE: 41 year old female was seen in endocrine clinic for hirsutism. Patient complained of having excess facial hair requiring her to shave every day, excess body hair, balding, facial acne and hoarseness of voice. She had noticed these changes over the last 1-2 years. She had normal menstrual cycles. Past medical history was significant for papillary thyroid cancer treated with total thyroidectomy and I131 more than 10 years ago. Patient was taking 200 mcg of levothyroxine daily and denied using any other prescription or over the counter drugs. On physical exam vitals were stable, BMI-34. Excess facial hair and male pattern baldness were noted. Cardiorespiratory and abdominal exam were normal. Pelvic exam showed clitoromegaly. Laboratory tests showed TSH- 2.56, total testosterone level of 543 ng/dl and free testosterone level of 129 pg/ml. DHEA- sulfate level was 189 microgram/dl. These values were repeated and confirmed. A pelvic ultrasound showed the right ovary measuring 4.3x2.9x4.5 cm. A solid vascular echogenic structure 3x3 cm with vascularity was noted in the right ovary. Abdominal CT scan was normal and adrenal tumor was ruled out. Our probable diagnosis was an androgen producing ovarian tumor most likely a Leydig cell tumor. Patient was referred to onco-gynecology and Laparoscopic right salpingo-oophorectomy and left salpingectomy were performed. Pathology report showed a well differentiated sertoli leydig cell tumor. Three months later the patient was seen in clinic. Her acne was clearing and repeat testosterone level was 21 ng/dl.
DISCUSSION: SLCT are usually seen in women in the age group of 30-50 years. They are unilateral in 98% of patients and associated with hyper androgenic symptoms. SLCT have a good prognosis and 5 year survival is 70-90%. Poorly differentiated tumors are associated with a worse prognosis. Surgical excision is the gold standard of treatment. It is generally believed that unilateral salpingo-oophorectomy is the treatment of choice in young women who wish to have children. Total abdominal hysterectomy with bilateral salpingo-oophorectomy is considered for women who do not wish to preserve their fertility.
CONCLUSION: To our knowledge this is the second report of SLCT occurring in a woman with history of papillary thyroid cancer. This may suggest common etiology. Further studies are needed to find if any association exists between these 2 endocrine tumors.

**Author: Christine Garcia MD, MPH**
Additional Authors:Kester Haye, MD, Joseph Mailman, MD

Institution: Stony Brook University Hospital - Department of Internal Medicine

**Title: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS (HLH) TOOK MY BREATH AWAY**

Purpose for study
Hemophagocytic lymphohistiocytosis (HLH) is an aggressive and life-threatening syndrome of excessive immune activation with initial signs and symptoms that can mimic common infections, fever of unknown origin, hepatitis, and encephalitis.
Case Description
A 44 year-old African-American male who is an active smoker without previous medical history or surgeries, presented to the Emergency Room with a 4-day history of back pain, headaches, nausea, vomiting, photophobia, diarrhea and nose bleeds. He denied trauma, insect bites, tick bites, travel or sick contacts. He was diagnosed with PNA/UTI, and sent home on azithromycin/cephalexin. He returned to the hospital 2 days later as symptoms worsened. He developed respiratory distress requiring intubation and ventilator support and synchronized shock for polymorphic VT. Amiodarone and lidocaine infusions were initiated and he was transferred to the intensive care unit. Labs revealed WBC 7.66, microcytic anemia with hemoglobin 9 g/dl, MCV 65.9 and lactic acid 2.7 mmol/L. Infectious workup was negative for Legionella and pneumococcal urinary antigens, Lyme, Babesia, HSV, Parvovirus, and CMV. Ferritin was 12876 ng/mL and triglycerides were 1,021 mg/dL.
The presence of persistent fever, elevated triglycerides and elevated ferritin support the diagnosis of Hemophagocytic Lymphohistocytosis (HLH). This patient had marked thrombocytopenia but the Hgb level was >9 g/dL and splenomegaly was not seen on the CT-scan. He was started empirically on IV decadron. Bone marrow biopsy demonstrated phagocytosis of platelets by macrophages consistent with HLH (see image). Patient improved on IV steroids, was extubated and started on Etoposide.
Importance of this Case
Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening emergency and delay in diagnosis and/or treatment is associated with high mortality. Findings suggestive of HLH include fever, cytopenia (thrombocytopenia), markedly elevated ferritin, and CNS symptoms. Respiratory abnormalities may require urgent ventilatory support and death from acute respiratory distress syndrome can occur. It most frequently affects infants from birth to 18 months of age, however the disease is rarely observed in children and adults. Immune activation is initiated by infection, most commonly viral infections, especially Epstein-Barr virus (EBV). Nearly all patients with HLH have very high ferritin levels and abnormal liver function tests. Increased triglycerides and abnormal coagulation parameters (elevated D-dimer) caused by hepatic dysfunction and disseminated intravascular coagulopathy are also seen. HLH has been reported in association with malignancies, most commonly lymphoid cancers, including T, NK, and anaplastic large cell lymphomas and leukemias.

**Author: Frank Genese DO**

Institution:Mount Sinai St.Lukes Roosevelt Hospital Center

**Title:An Interesting Etiology of Resistant Hypertension**

Introduction
Hypertension can be classified as primary or essential in greater than 90% of cases but recognizing secondary causes of hypertension can be critical to patient management and are often overlooked by clinicians.

Case Presentation
47M with PMH of IDDM, CKD stage 3, CAD, CHF, and chronic hypertension presented for evaluation of left-sided chest and flank pain. Upon presentation the patient was noted to be hypertensive (199/98) despite compliance with four anti-hypertensive agents. Serum chemistries were remarkable for hypokalemia. Of note, previous routine blood work demonstrated that the patient had a history of hypokalemia refractory to oral supplementation. While routine cardiac workup was unremarkable, urine studies exhibited significant proteinuria and a transtubular potassium gradient of 4.03 consistent with renal potassium wasting. Noncontrast CT abdomen revealed a 1.8 cm left adrenal adenoma. Plasma renin activity and aldosterone level were 0.15 and 9 respectively, resulting in a PAC/PRA ratio of 60. Given the suspicion for Connâ€™s syndrome, the patient was started on spironolactone with modest improvement in BP control and given appropriate follow up for adrenal venous sampling for possible subsequent unilateral adrenalectomy.
Discussion
Becoming increasing prevalent in the hypertensive population, clinical awareness of primary hyperaldosteronism is paramount given its greater associated cardiovascular risk, including stroke, nonfatal MI, and increased left ventricular mass, compared to primary hypertension.1 Primary aldosteronism is generally subdivided into aldosterone producing adenomas and bilateral adrenal hyperplasia (IHA), although other less common pathologic entities have been identified. Diagnosis involves a high degree of clinical suspicion, especially in younger patients with refractory hypertension. While typically associated with hypokalemic metabolic alkalosis, patients with primary aldosteronism can be normokalemic on presentation due to various potassium sparing mechanisms. Initial approaches to evaluation include obtaining plasma renin activity (PRA) and aldosterone levels (PAC), with an elevated PAC/PRA ratio being indicative of mineralocorticoid excess. Confirmatory testing is often required with salt-loading tests (oral sodium chloride tablets or saline-infusion) to demonstrate inappropriately elevated plasma / urine aldosterone levels. Although IHA generally involves a more indolent course, adrenal imaging is frequently pursued to delineate the subtype of primary aldosteronism given the varied treatment approaches. If an adrenal adenoma is identified, patients may be referred for unilateral adrenalectomy if desired, although the Endocrine Society currently recommends that unilateral disease should be confirmed with adrenal vein sampling with or without concomitant cosyntropin use.2 Conversely, medical management with aldosterone antagonists are the treatment of choice for bilateral adrenal hyperplasia.
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**Author: Hayas Haseer Koya MD**
Additional Authors:Dona Varghese MD(ACP Associate Member ),
Roshni Radhakrishna MD(ACP Associate Member),
Christopher Curtiss MD,
Tanya George MD, Dinesh John MD.
SUNY Upstate Medical University, Syracuse, NY

Institution: SUNY Upstate Medical University

**Title:A CURIOUS CASE OF MUCOCUTANEOUS ULCERATIONS DUE TO DRUG TOXICITY**

Introduction: Liver toxicity, bone marrow suppression and gastrointestinal side effects are well known adverse effects of methotrexate. However, cutaneous ulceration as a sign of methotrexate toxicity in patients without psoriasis has been rarely reported. We report a patient with rheumatoid arthritis who presented with cutaneous ulcers as a sign of methotrexate toxicity.
Case Presentation: A 64 year old lady with known history of rheumatoid arthritis, Type2 Diabetes mellitus, cirrhosis and portal hypertension presented to our hospital with complaints of skin ulcers and fatigue. On further evaluation she was found to have pancytopenia (WBC-1.8, Hemoglobin-6.3, Platelet count-10000) and mucocutaneous ulcerative lesions,1 to 2 cm in diameter involving the oral mucosa, abdomen, arms and legs(Image1). She was on methotrexate for the past 2 years without adequate medical follow up and non-compliant with folate supplementation. An extensive work up including vasculitis panel and viral hepatitis serologies were negative. Bone marrow biopsy revealed hypoplastic marrow with megaloblastic/dysplastic changes. Skin biopsy showed apoptotic keratinocytes and mild superficial perivascular dermatitis(Image1-bottom right). Both these biopsy findings were consistent with methotrexate toxicity. Methotrexate was discontinued and patient was given supportive treatment including skin care, folate supplementation and transfusion of blood products. Follow up examination over the next 2 weeks revealed improvement of blood counts and resolution of her symptoms including mucocutaneous lesions.
Discussion: Cutaneous adverse effects associated with methotrexate are usually dose related, and include toxic epidermal necrolysis and photosensitivity. Cutaneous ulceration is a rare occurrence and is often reported in psoriatic patients. The most common risk factors for toxicity are renal insufficiency, concomitant use of non-steroidal anti-inflammatory drugs, older age, infection, alteration in the dosage or methotrexate being restarted after a hiatus. Methotrexate acts by depleting intracellular stores of activated folate, thus disrupting DNA, RNA and protein synthesis. Folate or folinic acid supplementation can reduce methotrexate toxicity, specifically liver and gastrointestinal effects and oral ulcers.
Conclusion: We reiterate the need for considering methotrexate as a cause of mucocutaneous ulcerations in the appropriate clinical setting. Albeit a rare finding , it may be the initial clinical manifestation of methotrexate toxicity and underlying pancytopenia.

**Author: Jamael Hoosain MD**
Additional Authors:Hoosain, Jamael (Drexel U College of Medicine); Thachil, Rosy (DUCOM); Setyono, Devy (DUCOM); Grant, Joshua (LSU); Banka, Sahil (DUCOM)

Institution:Hahnemann University Hospital/Drexel University College of Medicine

**Title:Abstract: Mixed Atrial Septal Defect: an uncommon but dangerous congenital defect**

Introduction: An atrial septal defect (ASD) is a congenital communication between the left and right cardiac chambers. The three major recognized types are ostium secundum, ostium primum, and sinus venosus, with ostium secundum being the most common. While often initially asymptomatic, these defects do have the potential over time to cause hemodynamic complications, arrhythmias, and/or paradoxical embolisms. While an isolated ASD is not an uncommon finding with an incidence of 10-20%, the presence of a mixed defect (defects in two or more atrial septal zones) as in our patient is relatively rare.

Case: We present the case of a twenty-year-old Chinese woman with medical history of bipolar disorder who was referred to the outpatient cardiology office for recurrent syncope. Initial evaluation with electrocardiogram revealed sinus arrhythmia with an incomplete right bundle branch block and nonspecific T wave changes in the inferior leads. Transthoracic echocardiogram (TTE) demonstrated a normal ejection fraction but was notable for significant left to right color Doppler flow across the interatrial septum and images were consistent with a secundum ASD. Her right ventricular function was grossly intact, though both the right atrium and ventricle were significantly dilated. Further evaluation with transesophageal echo (TEE) not only confirmed the suspected ostium secundum defect but also revealed a sinus venosus ASD at the junction of the SVC and right atrium. Subsequent right heart catheterization showed pulmonary blood flow to systemic blood flow ratio (Qp/Qs) of 3.9, consistent with a large shunt. Pulmonary pressures were found to be normal and transpulmonary gradient was low. Given the symptoms, the presence of the sinus venosus ASD and right heart dilation, percutaneous closure was not feasible and she was referred for surgical intervention.

Discussion: This patient demonstrates defects in two separate zones of the atrial septum, thereby demonstrating a mixed defect. Her relatively young age of presentation was attributed to the magnitude of the shunt; many adults with congenital ASD do not present until the fifth decade of life. Such mixed defects occur in only 7% of ASDs, and prognosis is directly correlated with size of ASD, degree of right heart dilatation, and timely surgical intervention.

**Author: Betty Hsiao MD**
Additional Authors:Mohammed Elfekey MD, Deborah Asnis MD, Karen Beekman MD.

Institution: Flushing Hospital Medical Center

**Title:Kew Gardens Fever Returns**

During the summer of 1946, an outbreak of 124 people presented with similar rashes resembling chickenpox to their respective physicians in Kew Gardens, Queens. The patients resided within 3 apartment buildings, all within 3 square city blocks. The presentation of disease was described as a triad: an initial eschar, fever, and a papulo-vesicular rash. Within 5 months, New York City Department of Health (NYCDOH) and US Public Health Service were able to isolate, characterize and identify the causative agent and illness: Rickettsialpox, a mite-borne zoonosis caused by Rickettsia akari, is one of a few spotted fever group rickettsioses with a cosmopolitan distribution. Only about 800 non-fatal cases have ever been reported, with the majority surfacing in the 1940-1950s. Additional cases were reported in other Northeast metropolitan areas. The early 2000s witnessed another peak of incidences during the time of anthrax attacks, as the initial lesion of Rickettialpox resembles anthrax without the edema. Since then, the NYCDOH typically reports about 12-15 new cases each year.

Case Presentation

A 36 year old otherwise healthy man presented to ER with fever for 6 days, headache, sore throat, myalgias, and skin lesions on both legs. Patient denied pets, trauma, insect bites, sick contacts or recent travel. On exam, temperature was 102.6F with skin showing a maculopapular rash over the trunk and back with 2 eschar-like lesions on both legs. Bloodwork revealed elevated LFTS. Patient was started on doxycycline and ceftriaxone, while initial serology tested negative for rickettialpox. Repeat serology for rickettialpox and skin biopsy of eschar were sent for analysis. Subsequently, the rash spread, becoming papulovesicular without mucous membrane involvement or lymphadenopathy; patientâ€™s symptoms improved slowly. Results showed positive Rickettial antibodies in serology as well as a positive skin biopsy by immunohistochemical stains for ricksettial organisms, confirmed by PCR, all performed at the CDC. Patient was discharged with course of doxcycyline.

Discussion

Ricksettialpox is caused by Rickettia akari, transmitted by the mouse mite, residing in its hostâ€"the house mouse. During the initial outbreak investigation, the apartment incinerators were not operated daily, causing a buildup of waste, which attracted the mice. When rodent infestation is severe or when measures are undertaken for rodent control, the mites feed on humans. Most patients have no recollection of the bite, as mites do not attach or feed for long periods of time. Eschars are present in 90% but may go undetected due to paucity of symptoms. Rickettsialpox is under-diagnosed and as rodent control is required for prevention, prompt recognition and treatment is necessary for the sake of public health. In this case, even when initial serology was negative, clinical suspicion prompted for biopsy, which confirmed diagnosis, allowing for tailored treatment and satisfactory results.

**Author: Betty Hsiao MD**
Additional Authors:Mamta Shah, MD, Priyank Trivedi, MD, Alexander Mirzoev, Anil Kapoor, MD.

Institution:Flushing Hospital Medical Center

**Title:Anti-NMDA-receptor Encephalitis-Not that Unusual!**

Introduction:
Anti-NMDA-receptor encephalitis is a paraneoplastic autoimmune disorder, first described in young women with ovarian teratomas who presented with neuropsychiatric illness. Pathophysiology includes the development of autoantibodies against the NR1 and NR2 subunits of the NMDA receptors; it is postulated that the autoantibodies are produced by the teratoma and if a neoplasm is not found, an autoimmune phenomenon is suspected. Patients may experience a viral prodrome preceding symptoms. Affected patients develop significant psychiatric symptoms, seizures, memory deficits, and decreased levels of consciousness. Patients are underdiagnosed due to a lack of awareness among clinicians and the error of attributing the presenting symptoms to a psychiatric condition.
Case:
A 20 year-old Chinese-American non-gravid female without prior history of seizures presented in status epilepticus to the ER, where she was intubated and sedated with Dilantin & Propofol, after Lorazepam failed to break seizure activity. Past medical history was significant for anxiety as well as recent URI. Initial labs showed mild leukocytosis, rhabdomyolysis, with negative urine toxicology and serum alcohol. Initial CT head was negative for acute changes, lumbar puncture showed lymphocytic pleocytosis. Patient was empirically started on intravenous Rocephin, Vancomycin, Acyclovir, Ampicillin, discontinued after CSF studies ruled out infection. MRI demonstrated restricted diffusion in left hippocampus and medial temporal lobe. EEG did not reveal abnormalities. Patient was extubated after 3 days; she was awake, alert, without focal deficits or any subsequent seizure events but had periods of confusion and agitation. The patientâ€™s clinical picture prompted suspicion for NMDA encephalitisâ€”positive NMDA titers resulted, confirming diagnosis. Pelvic ultrasound showed a 1.4 cm right adnexal irregularly shaped cystic structure. She was transferred to a Neuro ICU, given IVIG and pulse steroids. Patient underwent successful right salpingo-oopherectomy with removal of ovarian teratoma. The patient continued to experience behavioral swings as well as autonomic instability with fluctuations in pulse and blood pressure, treated with rituximab and cyclophosphamide with notable improvement in her mental state.
Conclusion:
This case illustrates the importance of considering NMDA receptor encephalitis in the differential diagnosis of young females presenting with new onset seizures or psychiatric symptoms. It is crucial to promote awareness among colleagues so that prompt recognition of this disorder may allow for early therapy and more favorable outcomes. Prognosis is better if a neoplasm is found, as the neoplasm may be surgically resected, eliminating the source of autoantibodies. Main treatment modalities include tumor removal, along with immunotherapy such as steroids, IVIG, and plasmapharesis. Secondline immunotherapy such as rituximab and cyclophosmamide may also be used. NMDA receptor encephalitis is potentially lethalâ€”however, with early intervention, there is a high probability of recovery.

**Author: Uroosa Ibrahim**
Additional Authors:Amina Saqib, MBBS
Marcel Odaimi, MD
Frank Forte, MD
Jean Paul Atallah, MD

Institution: Staten Island University Hospital

**Title:A Rare Case of Severe Spurious Hypophosphatemia in a Patient with Multiple Myeloma**

Introduction: Symptomatic profound hypophosphatemia of <1 mg/dL is a rare finding and may be seen in the setting of trauma, alcoholism and sepsis. Signs and symptoms may include paresthesias, irritability, confusion, seizures, coma, proximal myopathy, ileus, diaphragmatic muscle dysfunction with respiratory failure, hemolysis and thrombocytopenia. Classical etiologies of true hypophosphatemia include acute respiratory alkalosis, acute volume expansion, hyperinsulinemia during refeeding, antacid ingestion, hyperparathyroidism, vitamin D deficiency, and Fanconi syndrome. Spurious hypophosphatemia can be seen with benign and malignant lymphoproliferative disorders.
Case: We present a case of a 60 year old male with a history of diabetes mellitus type 2, compensated hepatitis C with cirrhosis, hypertension, and a recent diagnosis of multiple myeloma not having begun treatment, presenting with a mechanical fall with trauma to the head and chest, and confusion. Social history was positive for 40 pack years of smoking, cocaine, marijuana, intravenous heroin, oxycodone use, and alcohol abuse. On presentation, he was in moderate respiratory distress, hypoxic and agitated, and was admitted to the ICU. Work up was pertinent for an inorganic phosphorus of <1mg/dL, intact PTH of 51.6 pg/mL, corrected calcium of 9.5 mg/dL, total protein of 9.5 g/dL, ammonia level of 81 µmol/L, beta2 microglobulin of 3353 ng/mL, quantitative IgG of 7274 mg/dL with evidence of IgG lambda protein on serum immunofixation. Fractional excretion of phosphorus in the urine was 84%. The patient was started on chemotherapy with bortezomib and dexamethasone. Inorganic phosphorus testing was recurrently reported as <1mg/dL. Intravenous and oral phosphorus and oral vitamin D were administered without improvement of symptoms or correction of the phosphorus levels. Therefore, spurious hypophosphatemia was suspected and the sample was sent to an outside laboratory that reported a value of 5.2 mg/dL. Upon further investigation, it was identified that a different method of detection (Vitros chemistry system, Ortho-Clinical Diagnostics, Rochester, NY) was used to test the sample. The patientâ€™s symptoms gradually improved over 10 days and by day 24, the serum phosphorus normalized and the total protein level was 5.8 g/dL.
Discussion: Spurious hypophosphatemia has been described with polyclonal paraproteins such as in diabetes and in polyclonal gammopathy. It is hypothesized that paraproteins interfere with either phosphorus alone or with the phosphomolybdate complex altering colorimetric absorbance causing spurious results of hypo- or hyperphosphatemia. Reported cases were analyzed by machines using Beckman technology and were overcome by manual deproteinization or use of Vitros and Kodak Ektachem systems that use a reducing agent p-methylaminophenol sulfate that stabilizes the phosphomolybdate coloric complex before measurement of absorbance. Further studies are required to clarify the exact physicochemical mechanism causing interference of paraproteins with measurement of phosphorus levels in order to guide appropriate treatment decisions.

**Author: Thaofiq Ijaiya**
Additional Authors:Ashutossh Naaraayan MD; Linda Williams MD; Prasanta Basak MD, Stephen Jesmajian MD.

Institution:Montefiore New rochelle hospital and Albert Einstein College of Medicine

**Title:Rare case of small cell breast carcinoma with second primary of ductal carcinoma**

The literature describes less than 40 cases of small cell breast cancer. Most patients are in the sixth or seventh decades of life. The overall prognosis of small cell breast carcinoma is unclear due to only a small number of cases being reported. The presence of a second primary with a different histology pattern has rarely been reported. We present a case of small cell breast cancer with a second primary ductal carcinoma.
A 49 year old female G3P2 presented with a rapidly enlarging right breast lump of one month duration. She was not on any medication. She had irregular menstrual period and had a normal mammogram a year prior to presentation. There was no relevant family history and no significant smoking or alcohol history. Examination of the right breast revealed a palpable 6 x 6 cm firm mass in the superolateral quadrant with mobile,non tender axillary nodes. Left breast examination was unremarkable. Mammogram revealed a mass in the 9 oâ€™clock position measuring 10 x 9 cm with abnormal right axillary lymph nodes. Further radiological investigation did not reveal evidence of malignancy at any other site. Ultrasound guided biopsy of the mass revealed small cell neuroendocrine carcinoma (SCNC) of the right breast. She initially underwent chemotherapy with cisplatin and etoposide. Following mastectomy,multiple biopsy from the excised mass did not show residual tumor at 9 o'clock but was positive for second primary tumor at 6 oâ€™clock position in form of infiltrating ductal carcinoma. Lymph node biopsy was significant for adenocarcinoma in two of three lymph nodes with histology similar to tumor at 6 oâ€™clock. She is still undergoing chemotherapy with adriamycin and cyclophosphamide with no sign of distant metastasis 9 months after initial diagnosis.
It has been suggested that SCNC is a variant of metastatic carcinoma arising from usual lobular or ductal carcinoma. However, some believe that SCNC is a distinct type of breast cancer different from the the usual types of carcinoma with variable degrees of neuroendocrine differentiation and carrying a worse prognosis. The term neuroendocrine tumor is now applied when more than 50% of the tumor shows such differentiation as in our patient .The discrepancy in prognosis in several reports may hinge on the non separation of pure SCNC from carcinoma of the usual types with areas of neuroendocrine differentiation. While some of the apparent pure SCNC cases show an appreciably worse prognosis, our patient appears to have responded to aggressive chemotherapy with no neuroendocrine cell component found on the excision specimen. Whilst prognosis may not be as grim as some reports would suggest, it would be important for further case reports to delineate pure SCNC from the usual type with areas of neuroendocrine differentiation.

**Author: Chaitanya Iragavarapu MBBS**
Additional Authors:Arunabh Sekhri, MD; John Savooji, MD; Sachin Sule, MD; and Delong Liu, MD

Institution: New York Medical College at Westchester Medical Center

**Title:Adult Onset Acute Lymphoblastic Leukemia presenting with Hypercalcemia from Osteolysis**

Introduction: Acute Lymphoblastic Leukemia is a malignant disorder originating from Progenitor B- or T-cells. It is the most common malignancy diagnosis in patients younger than 15 years of age but has a bimodal distribution with a second peak in the 6th decade of life. Patients generally present with manifestations of cytopenias such as infection, bleeding and exertional dyspnea. In children, especially young children, bone pain associated with limp is common. Here, we describe an adult patient who presented with bone pain from diffuse osteolysis and diagnosed with Acute Lymphoblastic Leukemia.
Case: 29 year old male with past history significant for diabetes mellitus, hypertension and traumatic paraplegia at C6 8 years prior presented to an outside hospital with complaints of nausea, vomiting and generalized weakness. He was found to be hypercalcemic on admission, but left against medical advice prior to work up. A bone scan at a second hospital revealed multiple lytic lesions with pathological fractures of ribs and right hip. A bone marrow biopsy revealed B-cell Acute Lymphoblastic Leukemia and the patient was transferred to our center for management. At the time of admission, the patient complained of continued nausea with bilious vomiting and bone pain. He reported chills with headaches, but denied fevers, night sweats and lethargy. His exam was significant for a blood pressure 162/89 and significant tenderness over his Right Chest wall and Right Hip. However, there was no pallor, bruising, lymphadenopathy or organomegaly. His complete blood count revealed mild anemia at 10.2 g/dL, mild thrombocytopenia at 120 and unremarkable differential count, barring a marginal elevation of immature granulocytes (1.2%). His chemistries revealed a creatinine level of 1.8 mg/dL, calcium of 13 mg/dL and uric acid of 9.3 mg/dL. Imaging revealed diffuse osteolysis of the skull, vertebra, ribcage and long bones. The patient was admitted to the Oncology Service and was started on HyperCVAD with good effect. Bisphosphonate therapy was attempted for acute treatment of his hypercalcemia but after initiation of chemotherapy, he became persistently hypocalcemic needing extensive calcium repletion. He was discharged after completion of Induction Therapy and stable Calcium levels.
Discussion: Acute Lymphoblastic Leukemia is described as a blood disorder with production of immature white cells. It is the most common leukemia in children and accounts for only 20% of Acute Leukemia in adults. The usual presentation is fatigue, fever and lethargy, with anemia induced dyspnea and angina occurring more often in adults. Bone pain is uncommon in adults and is almost never associated with osteolysis. Hypercalcemia in this setting is likely related to Parathyroid hormone related peptide (PTHrP) released by Blast Cells. While Bisphosphonate therapy has been used to good effect in children, it caused significant hypocalcemia in our patient after initiation of Chemotherapy.

**Author: Chaitanya Iragavarapu MBBS**
Additional Authors:Arunabh Sekhri, MD; Pallak Agarwal, MD; Sachin Sule, MD; and Robert Lerner, MD

Institution:New York Medical College at Westchester Medical Center

**Title:Multiple Myeloma presenting as Cardiac Amyloidosis without Monoclonal Spike**

Introduction: Multiple Myeloma is a plasma cell dyscrasia usually suspected in patients with anemia, renal failure, bone pain and recurrent infections. Though there is no standard screening test, usual practice is testing serum protein electrophoresis with immunofixation and Free Light Chains. If abnormal, further work up can be done. Here, we present an eighty year old male with no cancer history who was diagnosed with Multiple Myeloma after presenting with Cardiac Amyloidosis.
Case: Eighty year old male with history significant for benign prostatic hyperplasia and neuropathy initially presented to our Cardiology Service with worsening exertional dyspnea. He was found to have a positive Nuclear Stress Test showing Anterior Wall Ischemia and underwent triple vessel coronary artery bypass grafting. His ECHO, of note, showed preserved left ventricular ejection fraction (60%) with grade III diastolic dysfunction. Post procedure, he developed pneumonia and pleural effusion. He was treated with pig tail catheter placement and antibiotics and discharged in stable condition. He presented again within 1 week with similar complaints of dyspnea and fatigue. His admission vitals and examination were unremarkable. His labs were only significant for hemoglobin of 11.3 mg/dL, creatinine of 0.81, alkaline phosphatase of 143 and calcium of 9.4 mmol/L. His. His chest X-ray showed large bilateral pleural effusions. Repeat ECHO was done which again showed preserved left ventricular ejection fraction (54%) and significant diastolic dysfunction. Given suspicion for Cardiac Amyloidosis, work up was started with Serum Protein Electrophoresis with Immunfixation and Free Light Chains which only revealed elevated Kappa light chains (K:L ratio of 104). He subsequently had Urine Protein Electrophoresis which showed 3 monoclonal spikes and Urine Immunofixation which showed a monoclonal spike. Cardiac Biopsy showed a kappa light chain type Cardiac Amyloidosis; and Bone Marrow Biospy showed multiple myeloma with 60 to 70% plasma cells. Skeletal Survey showed subcentimeter lucent lesions within the skull as well as the distal femoral shafts bilaterally. Patient was started on therapy with Bortezomib and Dexamethasone, with plan to add Lenalidomide at a later date.
Discussion:Multiple myeloma is a mutisystemic disorder with poor prognosis, and usually presents with anemia, abnormal protein levels or renal function, hypercalcemia and/or bone pain. Amyloidosis is usually suspected in patients with diastolic heart failure with preserved Ejection fraction (HFpEF). Cardiac amyloidosis as sole manifestation of multiple myeloma has not been reported previously in the literature. Early recognition of this disease is important as timely therapy can help in quality of life and likely prolong the survival of these patients. Treatment for cardiac amyloidosis is essentially the same as Multiple myeloma, with three drugs. The two most common regimens are Revlimid, Bortezomib and Dexamethasone (RVD); or Cyclophosphamide, Bortezomib and Dexamethasone (CyBorD).

**Author: Sakshi Jasra MD**
Additional Authors:Suman Majumdar, MD
Bharath Rajagopalan, MD
Stanley Fernandez, MD

Institution: University At Buffalo, Department of Internal Medicine

**Title:Lazarus Syndrome: A Rare Case of Auto-resuscitation**

A 46-year old Caucasian male with past medical history of Type 1 Diabetes had a witnessed cardiac arrest at a supermarket. Cardiopulmonary resuscitation was initiated on site. The initial cardiac rhythm was noted to be ventricular fibrillation. The patient received three cycles of biphasic defibrillation, one dose of Intravenous Amiodarone 300mg, and five doses of Intravenous Epinephrine 1mg, as per ACLS protocol. The patient was intubated and resuscitation efforts were continued for 30 minutes, followed by return of spontaneous circulation (RoSC). The patient was then bought to an outside hospital ER. Upon arrival, EKG showed a junctional rhythm. However, before any further steps could be taken, the patient was noted to be asystolic. ACLS protocol was re-started for another 30 minutes. Bedside ultrasound did not reveal any cardiac activity and the patient was pronounced dead. One hour later, patient moved spontaneously and a pulse was checked. Return of spontaneous circulation was noted. Noninvasive blood pressure monitoring revealed a blood pressure of 115/80 and a heart rate of 92 BPM. Patient was alert and awake, and intermittently followed simple commands. Repeat EKG showed an infero-posterior ST-elevation myocardial infarction. The patient was started on a heparin drip and transferred to our facility to undergo cardiac catheterization.

During cardiac catheterization, he was noted to have complete occlusion of the left circumflex artery. A drug eluting stent was placed and he was started on an Integrillin drip. Chest x-ray showed right-sided pneumothorax occupying 50% of the lung volume with rib fractures. A chest tube was emergently placed. However, despite being maximally ventilated, the patientâ€™s oxygenation continued to decline. Patient was in severe cardiogenic shock. Despite aggressive resuscitation with intravenous fluids, ionotropic support and blood products, the patientâ€™s cardiopulmonary status continued to decline until he became asystolic. Resuscitation was continued for another 45 minutes without RoSC. Patient was pronounced dead.

Auto-resuscitation, colloquially known as â€œLazarus Syndromeâ€• after the biblical story of Lazarus, is an extremely rare phenomenon. It is defined as the unassisted return of spontaneous circulation after cardiopulmonary arrest (1). Possible explanations include the late effect of inotropic drugs, hyperventilation causing decreased cardiac output and myocardial infarction leading to stunning (2). It has been suggested that patients should be watched for at least 10 minutes prior to pronouncement, in order to allow drugs given during ACLS to wear off. Auto-resuscitation has grave implications regarding the definition of â€œdeathâ€• and when efforts to revive a patient should cease.

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**Author: Hari hara phani varma Kakarlapudi**
Additional Authors:Speirs S, Lal AP, Alaie D, Petrillo R, Mohammed A, Kolanuvada B, Bhargava M

Institution:Montefiore Mount Vernon Hospital

**Title:Cervical spine osteomyelitis presenting as Urosepsis.**

INTRODUCTION: Vertebral osteomyelitis is a rare condition seen more commonly in the fifth decade of life. Isolated involvement of cervical vertebrae is uncommon. Some predisposing factors for involvement of cervical vertebrae include Immunocompromised status, chemo-radiotherapy for malignancies of the neck, surgeries of the spine, pharynx and upper respiratory tract, trauma to the neck, dental extraction and IV drug abuse.

Hematogenous spread of urosepsis to the vertebrae causing secondary osteomyelitis has been reported but involvement of cervical vertebrae in urosepsis is rare. The case presented here is interesting as the patient came with symptoms of urinary tract infection and mild neck discomfort and was later diagnosed to have cervical osteomyelitis.

CASE PRESENTATION: A 73 year old male presented with a 1 week history of anorexia, worsening neck pain and dysuria. Past history includes fracture of right leg and appendicitis. He used to work as a handyman. Habits include 25 pack year smoking history and social alcohol use. Physical exam revealed tachycardia, fever, dry mucosa, mildly tender prostate and pain on active and passive movements of the neck.

Investigation revealed leukocytosis, neutrophilia, urinalysis positive for blood, WBC & bacteria and no valvular vegetations on Echocardiogram. Prostate specific antigen, ESR and CRP were grossly elevated at 28.30ng/ml, 87mm/hr and 258mg/l respectively.

Patient was empirically treated for urosepsis. Urine and blood cultures both isolated Methicillin susceptible Staphylococcus aureus (MSSA). Cervical spine MRI showed septic discitis of C5-C6 disc, as well as marrow edema in C5, C6, and C2 suggestive of osteomyelitis.

Patientâ€™s symptoms improved on treatment with intravenous antibiotics and neck brace to prevent compression fracture of the vertebral bodies.

DISCUSSION: Hematogenous spread of infection is the most common cause of vertebral osteomyelitis. Common sources of hematogenous spread include the genitourinary tract, skin and soft tissue, respiratory tract, infected intravenous catheter sites, postoperative wound infection, endocarditis, and dental infection. Contiguous spread of infection may occur from tissues such as the aorta, esophagus, or bowel that are adjacent to the spine. In many including our patient, however, the primary site of infection cannot be determined.

In our case it is unclear whether, Staphlococcus aureus bacteriuria (SABU) was secondary to Staphylococcus bacteremia (SAB) from cervical spine infection or whether the SAB was caused by primary infection within the urinary tract. Studies by Asgeirsson et. al. and Choi SH et al concluded that SABU appears to be secondary to SAB in some cases while it is the primary infection within the urinary tract causing SAB in others. This article hopes to present a case in which the presenting complaint was dysuria but cervical vertebra osteomyelitis was diagnosed later in the absence of any identifiable risk factors mentioned above.

**Author: Prerna Kumar MD**
Additional Authors:Waqas Nawaz, MD, Colette M. Knight, MD

Institution: Montefiore Medical Center (Wakefield)

**Title:Subclinical Hyperthyroidism and Amiodarone: A Cautionary Tale**

INTRODUCTION

Amiodarone is commonly used for the treatment of cardiac arrhythmias. Multiple adverse effects have been documented but careful monitoring of non-cardiac parameters remains suboptimal to date. We present a case of a patient with a history of subclinical hyperthyroidism that was started on amiodarone therapy which led to thyrotoxicosis.

CASE

71-year-old man with a history of atrial fibrillation-s/p pacemaker placement for sick sinus syndrome, gout, emphysema and prostate carcinoma-s/p radiation therapy presented with shortness of breath and dizziness for 3 days. The patient had been maintained on amiodarone 200mg daily, metoprolol 12.5mg PO q12h and digoxin 0.125mg PO daily for atrial fibrillation for three years and had adequate control of arrythmia. Physical exam revealed thyroid nodules and an irregularly irregular pulse of 150 bpm. Carotid doppler and ECHO were unrevealing. Bloodwork showed abnormal TSH of 0.03 uU/mL (0.5â€“6.0 µU/ml) and free T4 of 1.73 ng/dL (0.7â€“1.7 ng/dl) suggestive of hyperthyroidism. Amiodarone was stopped for suspected Type-1 amiodarone-associated hyperthyroidism causing decompensation of rate-controlled atrial fibrillation. Methimazole 10mg orally bid was initiated and later doubled to 20mg after the patient failed to respond. Thyroid ultrasound showed thyromegaly and two large nodules with hypervascularity. He was discharged on pradaxa, digoxin and metoprolol after rate control was achieved. One month later, the patient was readmitted for postural hypotension, increased sweating, diarrhea and a 10 lb. unintentional weight loss. Repeat thyroid panel showed free-T4 of 1.42 ng/dL, slightly improved from the previous 1.73 ng/dL and a TSH of 0.02 uU/mL. A retrospective review of thyroid panels over four years showed the evidence of subclinical hyperthyroidism with TSH 0.135 uU/mL and free T4 of 1.04 ng/dL. This was not taken into consideration before starting amiodarone.

DISCUSSION

Amiodarone is a lipophilic drug rich in iodine that has been associated with thyroid disorders. 2-12% of patients on chronic amiodarone therapy eventually develop amiodarone-induced thyrotoxicosis (AIT). While there is general agreement on following thyroid panels for patients with overt disease, no clear guidelines exist on management of subclinical disease in patients being considered for amiodarone therapy. Subclinical hyperthyroidism has independently been associated with worse cardiovascular outcomes. It also increases the risk of amiodarone-induced thyrotoxicosis (AIT), which could further worsen the cardiac problems being treated. As a result, in patients for whom there is indication to use amiodarone, a full assessment of baseline thyroid function tests is required. Patients with serological evidence of subclinical hyperthyroidism should be fully evaluated to determine the etiology of thyroid condition and closely monitored. AIT should be considered when treated patients develop clinical signs of thyrotoxicosis or decompensation of previously rate controlled atrial fibrillation.

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**Author: Anand Kumthekar MBBS**
Additional Authors:Jessica Kiarashi, MD.
Alexis Vien, MD.

Institution:Jacobi Medical Center

**Title:Pay Heed to the Contaminant : Invasive Achromobacter Pneumonia in an Immunocompromised Host**

INTRODUCTION:
Pneumonia in an immunocompromised host is unique with respect to the organisms involved. The emergence of new pathogens has significant impact on therapy. Achromobacter xylosoxidans (A.xylosoxidans) is an uncommon pathogen known to cause serious infections in immunocompromised hosts. Despite its ubiquitous existence, community acquired infections are rare. We report a case of A.xylosoxidans causing invasive cavitary pneumonia.
CASE:
A 53 year-old male with a history of HIV (unknown CD4 count, viral load) presented with ethanol intoxication. He denied any fevers, chills, cough, recent travel or sick contacts. Exam was significant for temperature of 101.9 F , sinus tachycardia, cachexia, and bronchial breath sound over the left upper lobe of the lung. Laboratory results showed a white blood cell count of 6.6k/ul, positive drug screen for cocaine and cannabis and elevated blood alcohol level. Computed tomography (CT) of the head was unremarkable and CT scan of the chest demonstrated an apical cavitary lesion with patchy opacity in the left upper lobe (figure 1). His CD4 count was 2 cells/ul and a viral load of 9058 copies/ml. Infectious work up including blood cultures, AFB smears, pneumocystis smear, aspergillus antibody and lumbar puncture were negative. Deep tracheal aspirate culture grew A.xylosoxidans. He was treated with cefepime for 14 days with clinical and radiological improvement (figure 2).
DISCUSSION:
A.xylosoxidans, isolated by Yabucci and Ohyama is an aerobic, gram-negative, catalase and oxidase positive bacilli. It is an opportunistic bacteria with low virulence but immunocompromised hosts with HIV or malignancy can experience significant morbidity and mortality. A.xylosoxidans is rarely isolated from clinical material and can be confused with other gram negative rods, like Pseudomonas and Burkholderia underestimating its role as a significant pathogen. A.xylosoxidans pneumonia is associated with a high case-fatality rate of 67% and complications include empyema, adult respiratory distress syndrome, chronic scarring and recurrent pneumonia . It is inherently resistant to many antibiotics but most isolates are susceptible to carbapenems and antipseudomonal penicillin. Due to the multidrug resistance and high case fatality, a drug sensitivity pattern is crucial to optimise therapy. Being so uncommonly isolated, A.xylosoxidans is either overlooked or discarded as a contaminant. In light of the propensity for misidentification, sequencing offers a more reliable method of definitive diagnosis, though its utility in clinical practice is unknown.
CONCLUSION:
Although A.xylosoxidans is routinely regarded as a contaminant, it can be a clinically significant pathogen causing invasive pneumonia in immunocompromised hosts with very high morbidity and mortality.

**Author: Joseph Mailman MD**
Additional Authors:Hassan Kidwai MD Member ACP, Michael Gavalas MS IV, Marlene Zawin MD, Satish Nagula MD

Institution: SUNY at Stony Brook

**Title:COLON CANCER RIDES IN ON A "CHARLIE HORSE" • : A CASE OF COLON CANCER PRESENTING AS HIP PAIN.**

Purpose: Consideration of a psoas abscess must be given in a patient presenting with nonspecific symptoms and lateral leg pain, and may be a critical clue to underlying malignancy.
Case: A 49-year-old male presented to the emergency department three times in as many weeks complaining of worsening right hip pain, described as a â€œcharlie horse,â€ • aggravated by weight bearing and forward flexion. Each time he was diagnosed with a musculoskeletal syndrome and discharged with supportive care. Three weeks later, he developed a rapidly growing mass on the anterolateral aspect of the right hip/groin. He denied abdominal pain or change in bowel habits. An MRI demonstrated a 17x5x10 cm mass in the right groin.

He had no routine medical care, took no medications, had a 20-pack year smoking history, and consumed a poor diet. Vital signs were normal, he was cachectic (BMI 18) and appeared chronically ill. He maintained his right hip in a flexed position as extension was painful. There was a 6x6x3 cm fluctuant, tender mass on the right groin, without inguinal adenopathy. There was no rash or penile discharge.
Labs demonstrated leukocytosis, microcytic anemia, and an ESR of 73. Contrast enhanced CT of the abdomen suggested a retroperitoneally perforated cecal mass with extension of an abscess adjacent to the iliopsoas bursa and right groin. 500 mL of purulent material was drained and grew Streptococcus bovis. Blood cultures were negative.
The abscess was formed by a micro-perforation from the cecal mass. While colonoscopy is risky in the setting of a perforation, gastroenterology deemed the benefits outweighed the risks given the extended duration from presentation and recent IR drainage. Biopsy of the cecal mass confirmed a moderately differentiated invasive adenocarcinoma. The patient underwent a hemicolectomy and began chemotherapy with FOLFOX.
Discussion: Iliopsoas abscess is a rare condition associated with a high degree of morbidity and mortality if misdiagnosed. Colorectal carcinoma is an uncommon, potentially life-threatening cause of secondary psoas abscesses, with abscess formation occurring in 0.3-0.4% of cases. Perforated colorectal tumors typically occur intraperitoneally and present with peritoneal irritation. Retroperitoneal perforation is unusual and insidious, and can delay diagnosis. Streptococcus bovis was cultured from the abscess in the presence of colorectal cancer without concurrent bacteremia, which is exceedingly rare. In a patient with nonspecific symptoms and lateral leg pain a high degree of clinical suspicion is required to suspect a psoas abscess, which may be critical in diagnosing underlying malignancy.

**Author: Sutapa Maiti MD**
Additional Authors:Sutapa Maiti MD, Minhaj Musa MD, and Kurt Kodroff MD, MMM,

Institution:KINGSBROOK JEWISH MEDICAL CENTER

**Title: QUALITY IMPROVEMENT PROJECT TO REDUCE BLOOD TRANSFUSION WASTE**

Quality Improvement Project to Reduce Blood Transfusion Waste
Sutapa Maiti MD, Minhaj Musa MD, and Kurt Kodroff MD, MMM,
Kingsbrook Jewish Medical Center, Brooklyn, New York
Goal of Project:
It is now generally accepted that a more restrictive policy for transfusions at a hemoglobin (Hgb) concentration of 7 to 8 g/dL is safe and effective care for most patients. Many patients only meet criteria for a transfusion when their hemoglobin is less than 7g/dl. The goal of this study is to eliminate unnecessary blood transfusions thereby reducing waste and avoiding transfusion complications by establishing a rapid feedback loop on performance.
Methods:
All patients receiving blood transfusions under the medical service at Kingsbrook Jewish Medical Center for the month of September 2013 were included in the analysis. Using our hospitalâ€™s electronic health record system,(Clinicianâ€™s View,) we gathered transfusion data retrospectively for the month of September 2013. All transfusions under the medicineâ€™s service were identified along with the following
-hemoglobin levels pre and post transfusion
-reasons given for transfusion
-names of attending ordering transfusion
Report cards were created for attending physicians with greater than two transfusions. These reports included the evidence based guidelines for transfusions, and each attendingâ€™s confidential data for each transfusion during the study period. Feedback was provided in a non-punitive manner. Additional the summative data was presented at the hospital wide quality assurance committee and in the Department of Medicine quarterly meeting.
Transfusion rates for the subsequent three months were analyzed and compared to the pre-intervention data.
Results: Primary outcome was a net reduction in transfusion of packed RBC per month corrective for discharges. Prior to the intervention, the ratio of transfusions to discharges was 0.40 transfusion per discharge. For the 4th quarter of 2013, the period post intervention, the ratio had decreased to 0.30. The intervention decreased the rate by 25%. We also compared the pre-intervention 4th quarter 2012 and found a ratio of 0.41 compared to the ratio of 0.30 during the post-intervention period of 4th quarter 2013. Thereafter a follow up study in June 2014 has been done to further monitor progress. Our ongoing study is to continually make efforts for improving adherence to recommended transfusion protocol.
Conclusion: This study demonstrated that a simple feedback system on performance delivered confidentially and non-punitively could reduce waste and improve adherence to evidence based medicine in a community teaching hospital. Keys to success include involving housestaff in analyzing data, presenting findings at institutional conferences, having a report delivered by IT that avoided any chart audits, and creating an efficient rapid system to deliver feedback focusing on the most clinically active physicians. The project is easily replicable, low cost and is associated with substantial financial savings and improved patient safety.

**Author: Mariam Murtaza Ali**
Additional Authors:Aisha Siraj MD

Institution: Bronx Lebanon Hospital

**Title: SUBOXONE: A RARE BUT LIFE THREATENING CAUSE OF RESPIRATORY FAILURE**

INTRODUCTION:
Suboxone belongs to the class of drugs used to prevent symptoms of opiate withdrawal, and is especially useful for treatment of patients addicted to heroin. Respiratory depression and central nervous system symptoms are rare but significant adverse effects. We report a rare case of suboxone related respiratory failure due to various drug interactions.
CASE REPORT:
A 45 year old male was admitted to a chemical dependence rehabilitation facility for opiate addiction. He was started on sublingual suboxone and lorazepam. Forty eight hours after admission, patient developed upper respiratory tract symptoms and was started on oral azithromycin. On the same day, he experienced difficulty breathing progressing to hypoxia and cyanosis when he was transferred to our Emergency Department. Imaging and lab results were significant for leukocytosis with a white blood cell count of 24,000, mildly elevated transaminases and bilateral pulmonary infiltrates on plain film. Patient was empirically treated with antibiotics for pneumonia and required noninvasive positive pressure ventilation. Within one day of admission, he had dramatic improvement in respiratory status along with resolution of infiltrates on repeat chest x-ray.
Upon review of the case, it was determined that the patient was admitted to the rehabilitation center for cocaine and tramadol addiction and was treated with suboxone and benzodiazepine which could have been the trigger for central nervous system depression. Subsequently, the administered macrolide antibiotic could augment the effects of suboxone. Early identification of potential negative drug-interactions with suboxone can be life saving.
DISCUSSION:
Suboxone (buprenorphine naloxone) is an opioid agonist-antagonist with a â€˜ceiling effectâ€™ for respiratory depression. Due to its unique pharmacology it offers practical advantages and enhanced safety when prescribed as recommended and supervised by a physician. Deaths have been reported from suboxone with concomitant sedative drug ingestion, such as benzodiazepines. The role of nor-buprenorphine, the main N-dealkylated buprenorphine metabolite with potent respiratory depressor activity, remains unclear. Experimental studies investigating the respiratory effects of combinations of high doses of buprenorphine and benzodiazepines suggested that this drug-drug interaction may result from pharmacodynamic interactions as well as inhibitors of CYP3A4 (such as azole anti-fungals, macrolide, antibacterials and HIV protease inhibitors) may increase plasma concentrations of buprenorphine, and patients concomitantly receiving these medications should be closely monitored and may require suboxone dosage reduction.
CONCLUSION:
Suboxone should be prescribed with caution in the presence of opioid analgesics, general anesthetics, benzodiazepines, phenothiazines, other tranquilizers, sedative/hypnotics, or other CNS depressants (including alcohol).

**Author: Praneetha Musty MBBS**
Additional Authors:Joshua Scheers-Masters,MD

Institution:Maimonides Medical Center

**Title:STATIN INDUCED IMMUNE-MEDIATED NECROTIZING MYOPATHY**

INTRODUCTION:
Statin induced necrotizing myopathy is a rare immune-mediated myopathy characterized by persistence of proximal muscle weakness, marked elevation of creatinine kinase (CK) levels despite discontinuation of statin, histological evidence of myonecrosis without significant inflammation and favorable response to immunosuppression[1].
CASE PRESENTATION:
A 67 year old male with a past medical history of hypertension, diabetes mellitus and coronary artery disease, on atorvastatin 80mg daily, presented with worsening proximal muscle weakness and elevated CK levels of two months duration despite the atorvastatin discontinuation. He had symmetric proximal arm and leg weakness on examination. Laboratory results revealed CK-4100 U/L (normal range 30-200 U/L) with normal TSH and renal function. Myositis antibody panel and workup for malignancy was negative.
Electromyography/Nerve conduction studies indicated generalized myopathy. Left thigh muscle biopsy revealed myonecrosis with sparse inflammation. He responded well to prednisone 60mg daily which was tapered off after 2 weeks. Patient was asymptomatic for one year with CK rising up slowly. He had a relapse 18 months after the steroid taper associated with CK of 11,523 U/L. Repeat muscle biopsy of right thigh showed active myonecrosis, regeneration with limited inflammation and widespread MHC class-1 immunoexpression. Anti-3-Hydroxy-3-methylglutaryl-coenzyme A reductase autoantibody was not checked due to financial constraints. Patient was symptomatic despite being on prednisone 60mg and azathioprine 100mg daily and therefore he received 2 cycles of IVIG infusion. He had significant clinical improvement with IVIG and CK trended down to 1000 U/L within 2 months after the infusion.
DISCUSSION:
Statin induced immune-mediated necrotizing myopathy has been strongly associated with antiâ€“HMGCR autoantibodies which target the 3-Hydroxy-3-methylglutaryl-coenzyme A reductase protein expressed by the regenerating muscle fibers and is up-regulated by the statins[2]. Muscle biopsy usually reveals extensive myonecrosis with regenerating fibers with minimal inflammation and sarcolemmal MHC-1 expression on non-necrotic fibers[3,4]. Steroids are initially given followed by long term immunosuppression with steroid sparing agents like methotrexate, mycophenolate and azathioprine or IVIG as frequent relapses can occur when the treatment is tapered[3].
CONCLUSION:
Statin induced necrotizing myopathy is considered as a diagnosis of exclusion and suspected when the workup is negative for other etiologies[3]. Early recognition of this rare entity is important because of the autoimmune process triggered by statin which requires immunosuppressive therapy.
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**Author: Ashutossh Naaraayan, MD**
Additional Authors:Thaofiq Olatunde Ijaiya MD; Prakriti Bista MD; Prasanta Basak MD; Stephen Jesmajian MD.

Institution: Montefiore New Rochelle Hospital and Albert Einstein College of Medicine

**Title:HYPOGLYCEMIC HEMIPLEGIA**

Introduction
Serum glucose less than 40mg/dL is a well-recognized cause of hypoglycemic encephalopathy. Hypoglycemia manifests either as adrenergic symptoms like anxiety, restlessness, sweating, tachycardia, hunger and irritability; or as neuroglycopenic symptoms like headache, dizziness, confusion, perioral tingling, seizures, stupor and coma. Hypoglycemic hemiplegia (HH) is a rare manifestation of hypoglycemia and is found in about 4% of severe hypoglycemic cases. We present a case of an elderly diabetic female with HH.
Case report
A 65 year old female with history of DM on insulin, was brought to the emergency department (ED) from a nursing home for lethargy and dysarthria for 3 hours. There was no prior history of cerebrovascular accident, migraine, or seizure disorder. In the ED she was noted to have right-sided hemiparesis and dysarthria. CT scan of the head was negative for any acute intracranial event. Vitals recorded on admission: BP 187/82 mm hg, HR 110/min, RR 14/min and temperature: 99.2 F. She was lethargic, mildly diaphoretic, and unable to speak clearly. A right sided facial droop was noticed. She could move her left upper and lower extremities but not the limbs on the right side. Motor strength was 4/5 on the left extremities and 0/5 on the right side. Pupils were equal and sluggishly reactive to light. Cardiac and respiratory examination was normal. Blood glucose was 25 mg/dL. 50 ml of 50 % dextrose was administered immediately and patientâ€™s level of awareness improved instantly. She was able to speak fluently, follow simple commands and motor strength was 5/5 on all extremities. The facial paralysis also resolved. She was able to walk without ataxia within 15 minutes of administration of dextrose. Doppler ultrasound of carotids was normal. Her insulin regimen was adjusted and symptoms did not recur during the hospital course.
Discussion
HH is a well-defined but rare manifestation of hypoglycemia with about 200 cases reported. The average serum glucose at which HH develops is 32 mg/dL. For unexplained reasons right sided hemiplegia is seen in 66% of cases. The mechanism of HH is not fully understood. Several hypotheses have been proposed which include local cerebral vasospasm, failed auto-regulation of cerebral blood flow and selective vulnerability of certain regions of brain to the hypoglycemic insult. Reduced glucose delivery to neurons leads to failure of high energy membrane ion pumps, eventually leading to cell death. Increased oxidative damage in severe hypoglycemia has also been described.
HH is readily reversible and should be considered as a cause of hemiplegia. We recommend finger stick glucose testing in the preliminary evaluation of all individuals presenting with suspected cerebrovascular accident. Such an approach can avoid unnecessary and expensive investigations. Suspicion of HH should be especially high in diabetics on hypoglycemic agents.

**Author: Amir Naraghi MD**
Additional Authors:Pramod Theetha Kariyanna, MD

Institution:Brookdale University hospital and medical center

**Title:Synthesized seizures in an elderly male: are the old growing young in use of synthetic cannabinoid?**

Objective:
1) To demonstrate how an extensive history is key in the diagnosis of Synthetic cannabinoids (SC) induced seizure
2) To identify SC abuse as a growing cause of concern in elderly
Clinical vignette:
69 year old African American male presented to the ED with generalized tonic-clonic seizures (TCS) after smoking K2. Patient had four episodes of seizures, one of which occurred in his home, one in the ambulance and two in the ED. Appropriate aspiration precautions were followed in ED. Each episode lasted approximately three minutes and was not associated with bowel or bladder incontinence. He has a past medical history of gout, hypertension, benign prostatic hypertrophy and Gastroesophageal reflux disease. Upon physical examination, the patient was agitated with a blood pressure was 180/100, pulse rate of 120, and ecchymoses were detected. Patient received Haldol and lorazepam for agitation and seizures, respectively. Upon laboratory investigation, the significant findings were as follows: serum creatinine of 1.8 and a CPK level of 363 which ultimately trended up to 60414; urine toxicology screening was negative. No intracranial pathology was detected in CT/MRI head and TTE revealed normal systolic function. Patient was admitted for monitoring during which he had two episodes of TCS , that was managed with levetiracetam.
Discussion:
SC are synthetic agonists at CB1 receptors and are chemically dissimilar to cannabinoids. SCâ€™s are sprayed onto an herbal mix (not tobacco or cannabis) and are often marketed in legal retail outlets as K2, spice, etc, labeled, â€œnot for human useâ€• , to evade FDA scrutiny. These drugs rank second among the illicit drugs used by this age group. CB1R in the brain mediates the psychotropic effect of tetrahydrocannabinol (THC). SCâ€™s have higher affinity to CB1R and are more potent than natural cannabinoids (NC).
NC possess antiepileptic activity by unknown mechanisms, however, this activity is hypothesized to be attributed to the decreased transmission in glutamate systems due to the fact that cannabinoids decrease synaptic transmission in glutamate and ?-aminobutyric acid (GABA) systems in the brain. The inhibition of ?-aminobutyric acid (GABA) however may increase the risk of seizures. Tetrahydrocannabinol (THC) is a partial agonist at CB1R, whose anticonvulsive mechanism is yet to be understood. Cannabidiol (CBD) has low affinity for CB1R. In vitro and in vivo seizure animal studies suggested that the anticonvulsant action of CBD is independent of CB1R.
Patients who use SC present with symptoms not usually seen with natural marijuana such as seizures, hallucinations, agitation, psychosis, tachycardia, or elevated BP. SC evade detection as they are chemically very distinct from THC. Our case shows that a thorough history is vital in identifying the cause of SC induced seizures and raises concern of increased SC use in the elderly.

**Author: Waqas Nawaz Resident PGY2**
Additional Authors:Barry Fomberstein MD

Institution: Montefiore Medical Center (Wakefield Division) Albert Einstein College of Medicine

**Title:Hypothermia Caused By Quetiapine: A Case Report**

INTRODUCTION:
Hypothermia is defined as a core body temperature below 95ºF. Atypical antipsychotics, e.g. risperidone and olanzapin , have traditionally been linked with hypothermia. The following scenario illustrates a case where the cause of hypothermia remained undetermined, leading to multiple hospital admissions in one year. Ultimately, quetiapine was found to be the causative factor, and stopping the medication corrected the hypothermia.
CASE:
The patient is a 59 y woman with a history of hypothyroidism, mental retardation, seizure and bipolar disorder, who had been taking quetiapine presented with altered mental status and hypothermia of 91F. Routine laboratory testing, including WBC count, thyroid function tests and cortisol levels, was normal. She was initially treated for sepsis of unknown origin and was started on broad spectrum antibiotics. The patient had 4 episodes of hypothermia during the hospital stay with a specific pattern that was related to her dosing of quetiapine. These episodes occurred around 10 PM, after she had taken the 3 daily doses of quetiapine. The previous record showed that she was initially started on quetiapine 200 mg daily without any episodes of hypothermia noted. Subsequently the dose was increased to 200 mg TID and this was followed by 3 subsequent admissions for hypothermia in one year. After discontinuation of quetiapine, no further episodes of hypothermia were noted. Divalproex was continued as treatment for seizure disorder. Given the strong correlation between the discontinuation of quetiapine and the disappearance of the hypothermia, quetiapine was presumed to be the cause for this phenomenon.
DISCUSSION:
Hypothermia in patients on atypical antipsychotics is a serious, unpredictable, idiosyncratic adverse reaction that frequently leads to hospital admission and infrequent fatalities. Among atypical antipsychotics, hypothermia is attributed more often to olanzapine and resperidone, due to their antagonistic actions on D2 and 5HT2 receptors. However, there are a few case reports that described quetiapine-induced hypothermia. Quetiapine has antagonistic action on D2, 5H2A and alpha 2 receptors with the highest affinity for alpha-2 receptors. The alpha 2 receptor blocking inhibits the peripheral vasoconstriction and shivering , and thus may play a role in inducing the hypothermia. The high 5HT-2/D2 affinity ratio also contributes to this adverse effect by disrupting the thermoregulation. Interestingly, the hypothermia caused by quetiapine is dose dependent and is more marked in patients with CNS disorders
Conclusions:
1. In cases of recurrent hypothermia, other causes, such as medications, should also be explored besides the more commonly expected causes such as sepsis, malnutrition and endocrine disorders.
2. Quetiapine can cause hypothermia in a dose dependent manner, especially in patients with CNS disorders
3. The antagonism of alpha-2 receptors is main mechanism of quetiapine-induced hypothermia due to antagonism of the D2 and 5HT2 receptors.
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**Author: Waqas Nawaz Resident PGY2**
Additional Authors:Prerna Kumar MD, Colette M. Knight MD

Institution:Montefiore Medical Center , Wakefiled Division, Albert Einstein College of Medicine

**Title:Diabetic Third Nerve Palsy with Pupillary Involvement: "Open up" eyes for Diabetes: a case report**

INTRODUCTION:
The 3rd nerve palsy caused by diabetes is usually considered to cause complete external dysfunction with eye down and out sparing the pupil completely (no internal dysfunction). But there are a few cases where diabetic 3rd nerve palsy involves the pupil partially causing anisocoria. The following case describes a case report where diabetes presented with atypical 3rd nerve palsy.
CASE:
51 year old woman with a history of long-standing uncontrolled diabetes with HbA1c 12% (normal <6.4%), hypertension and hyperlipidemia presenting with 6 day history of right eye ptosis without any complaints of blurry vision. Exam was remarkable for medial opthalmoplegia with eye deviated out but not down, and right pupil greater and sluggish than left pupil. The neuro imaging including CT head, CTA, MRA were unremarkable. The anisocoria noted initially led to carotid angiography that was negative for aneurysm. Finally the symptoms were attributed to uncontrolled diabetes. The insulin regimen was optimized and patient was discharged. Follow-up after 3 months revealed better controlled diabetes (HbA1c: 7%) with resolution of eye symptoms.
DISCUSSION:
3rd nerve palsy with pupillary involvement often raises the suspicion of neurological emergencies such as intracerebral aneurysm or subarachnoid hemorrhage while diabetic 3rd nerve palsy is usually considered to be pupil sparing. The incidence of pupillary involvement in 3rd nerve palsy caused by diabetes is more common than has been recognized in past years. About one fourth of patients with diabetic 3rd nerve palsy has some degree of anisocoria and is usually < 1mm. The associated sluggish response of affected pupil differentiates diabetic pupillary involvement from the aneurysmal compression of 3rd nerve that causes unresponsive pupil. The opthalmoplegia in diabetic 3rd nerve palsy is usually complete with affected eye â€œdown and outâ€• and it resolves earlier than anisocoria. But in few instances, it presents as incomplete external dysfunction evident as medial opthalmoplegia with eye only â€œout without being downâ€• (as in our case). The exact mechanism for pupillary involvement (internal dysfunction) in DM is unclear but it may be attributed to autonomic neuropathic effects of diabetes on Edinger- Westphal nucleus in midbrain. The atherosclerotic effect of diabetes involving vasa nervosum in 3rd nerve explains the external dysfunction seen in diabetic 3rd nerve palsy. The optimal control of diabetes is mainstay of treatment that results in resolution of symptoms in 3 months.
LESSONS LEARNED:
1. Pupillary involvement in diabetic 3rd nerve palsy is more common than assumed in past.
2. The autonomic neuropathy and atherosclerotic effect of diabetes are considered possible pathological mechanisms.
3. Optimal diabetic control is the cornerstone of treatment.
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**Author: Anu Neerukonda MBBS**
Additional Authors:Fengshuo Lan, Theodore Gabig

Institution: SUNY Stony Brook University Hospital

**Title:REFRACTORY ADULT PRIMARY AUTOIMMUNE NEUTROPENIA THAT RESPONDED TO ALEMTUZUMAB**

INTRODUCTION
Primary autoimmune neutropenia (P-AIN) has been reported in few adult patients, but limited data exists regarding effective treatment strategies for patients failing conventional treatment.
CASE DESCRIPTION
A 35 year-old man in good health presented with gingival and rectal pain and was found to have a rectal fissure on exam. Laboratory testing revealed an absolute neutrophil count (ANC) of zero and he was treated with granulocyte-colony stimulating factor (G-CSF) with a transient response. He was readmitted multiple times thereafter with acute febrile illnesses and ANC of zero treated with G-CSF with only a transient response each time. Diagnostic evaluation revealed negative serologies for Hepatitis B, Hepatitis C, Epstein-Barr virus, Cytomegalovirus, Lyme disease, and HIV1 and 2. Flow cytometry of peripheral blood leukocytes was negative. Cytoplasmic-antineutrophil cytoplasmic antibody as high as 1:1280 was detected consistent with autoimmune neutropenia. Bone marrow aspirate and core biopsy revealed normal karyotype, normal cellularity with maturation arrest at the myelocyte stage and occasional hypolobulated megakaryocytes that did not meet the criteria for myelodysplastic syndrome.
Over the next 3 years, his neutropenia transiently improved with a variety of agents including Rituximab, Cyclosporine, methylprednisolone, intravenous immunoglobulin (IVIG), and Methotrexate with G-CSF. Splenectomy was then performed for intractable left upper quadrant pain and surgical pathology revealed non-specific reactive lymphoid hyperplasia. Post-splenectomy, his neutrophil counts rose transiently for 4 months only.
Based on the study by Willis et al., in which Alemtuzumab was used intravenously in the treatment of autoimmune cytopenias, a decision was made to administer a modified regimen of Alemtuzumab 10mg fixed dose subcutaneously for 5 days a week for 2 weeks. Neutrophil counts rose and the patient continued to have a sustained response for over seventeen months until his death from a motor vehicle accident. In the 17 months post Alemtuzumab, he was hospitalized 4 times for febrile illnesses compared with 9, 5 and 6 times in the three individual prior years.
DISCUSSION
In contrast to P-AIN occurring in childhood, the disease in adults has a chronic course and spontaneous remission is unusual. Alemtuzumab is a humanized IgG1k monoclonal antibody that recognizes the CD52 antigen on human lymphocytes, monocytes, macrophages, eosinophils, dendritic cells and natural killer cells. Alemtuzumab causes cell lysis via complement or antibody-dependent cellular cytotoxicity as well as by directly acting on T lymphocytes, which play an important role in controlling expansion of antibody producing autoreactive B-cell clones. Based on our experience with this patient, Alemtuzumab was effective in maintaining stable neutrophil counts and reducing neutropenia related hospitalizations. It is potentially a novel option for the treatment of refractory p-AIN but should only be considered in patients who have failed multiple conventional, less toxic treatments due to high risk of infectious complications.

**Author: Chukwudi Obiagwu MBBS**
Additional Authors:Sameer Chadha MD, Kunal Teli MD, Guy Kulbak MD, Gerald Hollander MD, Jacob Shani MD.

Institution:Maimonides Medical Center

**Title:Carfilzomib-Induced Flash Pulmonary Edema**

Introduction: Carfilzomib (CFZ) is a chemotherapy agent used in the treatment of relapsed and refractory multiple myeloma. We present the case of a woman with flash pulmonary edema and ST segment changes after treatment with this agent.
Case Presentation: A 54 year old woman presented to our emergency department with sudden onset shortness of breath and chest pain. The patient had been diagnosed with multiple myeloma (MM) 5 years ago and had suffered relapse after two prior chemotherapy regimens which included bortezomib. Her home medications were gabapentin, bupropion, and hydromorphone. A day prior to presentation, she received a first dose of CFZ from her oncologist. On physical examination, heart rate was 121/min, respiratory rate was 26/min, blood pressure was 116/83 mmHg, temperature was 36.8 C, and SpO2 was 91%. ECG revealed ST depressions in leads I, aVL, V5, and V6 and minimal ST elevation in inferior leads. Blood tests were unremarkable except for hemoglobin of 9.8 g/dL and slightly elevated cardiac enzymes. CT angiogram of chest revealed pulmonary edema with no emboli. The patient was in severe respiratory distress and had to undergo endotracheal intubation. Coronary angiogram showed normal coronaries. The patientâ€™s symptoms improved with diuretic therapy. A 2-D echo showed a LVEF of 35-40. She was successfully extubated on day 3, and was subsequently discharged on day 9 following full recovery.
Discussion: CFZ is a second generation proteasome inhibitor for the treatment of relapsed and refractory multiple myeloma (RRMM) patients who have received at least 2 prior therapies, including bortezomib and an immunomodulatory agent, and have demonstrated disease progression on or within 60 days of the completion of the last therapy. CFZ primarily inhibits the chymotrypsin-like site of the proteasome (1). It acts by inhibiting proteasomes, which are responsible for maintaining cardiac myocyte protein quality, cellular mass and sarcomere quality. Therefore the cardiac ubiquitin-proteasome system is functionally degraded, a finding present in heart failure (2). In phase 2 clinical trials of CFZ, cardiac failure events were reported in 7% of patients, and were mainly grade 3 to 4 severity (3).
Conclusion: This case to the best of our knowledge is the first report of CFZ causing an adverse effect after administration. We recommend that physicians should monitor patients started on CFZ closely and also elicit a detailed medication history.
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**Author: Chukwudi Obiagwu MBBS**
Additional Authors:Vishesh Paul MD, Sameer Chadha MD, Gerald Hollander MD, Jacob Shani MD.

Institution: Maimonides Medical Center

**Title:Acute Pulmonary Edema Secondary to Hyperbaric Oxygen Therapy**

Introduction: Hyperbaric oxygen therapy (HBO) has been shown to be effective in the treatment of diabetic ulcers, air embolism, carbon monoxide poisoning, and gas gangrene. It is usually well tolerated with very few side effects.
Case Presentation: An 80 year old male with Ischemic Cardiomyopathy (ejection fraction - 25%), diabetes mellitus and peripheral vascular disease was admitted because of severe dyspnea. He was getting hyperbaric oxygen treatment for a non-healing ulcer on his foot. His vitals were stable and he was breathing comfortably before the start of therapy. Towards the end of treatment, he developed rapidly worsening dyspnea. EMS was called and he was brought to the hospital on 100% oxygen via non-rebreather mask. His severe respiratory distress and physical examination findings including diffuse inspiratory and expiratory crackles required him to be intubated and mechanical ventilation started. EKG did not show any ischemic changes. Cardiac biomarkers were negative, but his BNP was significantly elevated at 1568 pg/mL. There were pink frothy secretions in the endotracheal tube and chest X-ray showed severe pulmonary edema. He was admitted to the cardiac ICU for acute respiratory failure secondary to pulmonary edema. He received ventilator care and intravenous diuretics, and was successfully extubated 3 days later.
Discussion: HBO therapy has been shown to improve the rate of healing of diabetic foot ulcers possibly by improving wound tissue hypoxia, enhancing perfusion, and down-regulation of inflammatory cytokines. Some side effects of HBO include otic barotrauma, visual changes and possible CNS oxygen toxicity. Very few cases of pulmonary edema due to HBO treatment have been described. Weaver et al described three cases in 2001- all of them had pre-existing cardiac disease, and two of them were diabetic. Yildiz et al demonstrated that HBO treatment led to increase of N-terminal pro-B-type natriuretic peptide (NT pro-BNP) levels in diabetics by mean of 100 pg/mL.
HBO can precipitate acute pulmonary edema in patients with pre-existing heart disease via the following possible mechanisms: HBO-induced hyperoxia leading to increased peripheral vasoconstriction and thus cardiac afterload, increased oxidative myocardial stress, decreased LV compliance by oxygen radical-mediated reduction in nitric oxide and increased pulmonary capillary permeability. Treatment remains primarily supportive with diuretics, supplemental oxygen and occasionally ventilator support.
Conclusion: Acute pulmonary edema is a rare but serious side effect of hyperbaric oxygen therapy in patients with pre-existing heart disease. Thus caution should be observed in treating patients with heart disease with hyperbaric oxygen therapy.

**Author: Won Jun Park M.D.**
Additional Authors:Won Jun Park, MD, Pawan Sarda, MD, Kevin Luke Tsai, MD, Ciprian Nedelcu, MD

Institution:The Brooklyn Hospital Center

**Title:Regadenoson (Lexiscan)-Induced Left Bundle Branch Block (LBBB) which Reversed with Administration of Aminophylline**

Regadenoson is a pharmacologic stress agent indicated for radionuclide myocardial perfusion imaging (MPI) in patients unable to undergo adequate exercise stress testing. It is a modified form of the adenosine molecule with an additional side chain (1, 2, 6) and works by activating the A2A adenosine receptor, which produces coronary vasodilatation and increases coronary blood flow (1, 3, 4). Adenosine receptor agonists, including regadenoson, can depress the SA and AV nodes and can cause first-, second-, or third- degree AV block and sinus bradycardia (5). Until now, a new-onset LBBB secondary to the administration of regadenoson has not been described.
This is a case report of an 84-year-old obese African-American woman with coronary artery disease status post (s/p) percutaneous coronary intervention with stent placement, severe peripheral vascular disease s/p stent placement, hypertension, type 2 diabetes mellitus, chronic kidney disease stage 2, and dyslipidemia who received a radionuclide MPI as an outpatient. She had no known allergies and took aspirin, clopidogrel, metoprolol, quinapril, simvastatin, furosemide, nifedipine (extended release), and insulin (NovoLog Mix) at home. Her echocardiogram showed concentric left ventricular hypertrophy (LVH) with normal LV systolic function and an EF of 65-70%, moderate aortic stenosis, mild mitral regurgitation and tricuspid regurgitation, and mild-to-moderate pulmonary hypertension. Her electrocardiogram (ECG) showed sinus rhythm with minimal voltage criteria for LVH and normal QRS duration and morphology (Figure 1). The test was initiated and the patient was injected with 0.4 mg of regadenoson. Her baseline ECG showed a heart rate of 63 beats per minute (BPM) (Figure 1). Ten minutes into the test, the patientâ€™s ECG showed a LBBB pattern (Figure 2). Her peak heart rate was 83 BPM during this episode but had decreased to 65 BPM while the LBBB pattern persisted, before aminophylline administration (Figure 3). She was asymptomatic during this period. Our initial suspicion was between rate-related LBBB and regadenoson-induced LBBB. 50 mg of aminophylline was administered and an ECG performed immediately after aminophylline administration did not show the LBBB pattern and showed that the electrical activity of her heart had returned to her narrow-complex baseline with the same heart rate (Figure 4). This supported our suspicion that regadenoson was the causative agent of this new-onset LBBB.
It is well established that the adverse effects from regadenoson administration, in terms of arrhythmias, are AV block and sinus bradycardia; not bundle branch block (5). The exact mechanism of this phenomenon is not known. From this case report, further studies can be pursued to determine the mechanism of effect of regadenoson on the cardiac conduction system and to reveal whether the quality and/or quantity of A2A adenosine receptors in the cardiac conduction system play a significant role.

**Author: Eugene Pashkovetsky**
Additional Authors:Robert Graham, MD

Institution: North Shore LIJ Lenox Hill Hospital

**Title:The “Whoosh” • In My Ears**

Introduction:
Intracranial Hypotension is a benign condition characterized by an orthostatic headache, that is, one that occurs or worsens with upright posture. In addition to headaches, patients may experience nausea, dizziness, horizontal diplopia, changes in hearing, or radicular symptoms involving the upper limb, all of which are orthostatic in nature.
Case:
A 31 y/o female 37 weeks pregnant initially presented to the hospital for cesarean section for delivery of twins. The delivery was complicated by significant post-partum bleeding, requiring multiple units of blood transfusions, and subsequent supracervical hysterectomy. She presented the following day with hyperacusis and â€œechoingâ€ • symptoms that were unbearable in tolerating everyday sounds. Patient at the time denied hearing loss, vertigo, diplopia, or headaches, and stated that the auditory changes came on the same evening after the surgery was done. Of note, patient stated that she had an epidural anesthesia two days ago prior to her Cesarean section but stated that she had no complications after it was done. On physical exam, patientâ€™s vital signs were stable and there were no pertinent findings on the patientâ€™s neurologic exam including conductive and sensorineural hearing loss. Patient was sent for an MRI/MRA with and without contrast of her brain and was found to have meningeal thickening (in the convexity of bilateral cerebral hemispheres) and diffuse dural enhancement. This study was followed up by a lumbar spine MRI to look for a CSF leak but the study was unremarkable. Given the findings of the brain MRI, and that a CSF leak could not be ruled out, a blood patch was recommended as a therapeutic and diagnostic for intracranial hypotension. A few hours post blood patch, patients symptoms improved with complete resolution within 48 hours.
Discussion:
The incidence of intracranial hypotension has been estimated at 5 per 100,000 per year, with a peak around age 40 years and a female to male ratio of 1.5 to 1. Most cases result from a persistent CSF leak, such as after dural puncture for a lumbar puncture, myelography, or spinal anesthesia.
The diagnosis of intracranial hypotension can be confirmed by demonstrating decreased CSF opening pressure, often less than 60 mm H20, on performing an LP. Imaging can also be used for diagnosis. Cranial MR with gadolinium can show diffuse thickening of the pachymeninges, engorgement of venous sinuses, subdural fluid collections, and downward displacement of the brain. Intracranial hypotension can result in several complications including subdural hematoma, subarachnoid hemorrhage, dural venous sinuses thrombosis, and stupor resulting from sagging of the brain. Treatment for ICH involves conservative management and bed rest. Laying supine reduces CSF pressure at the site of leakage and therefore allows healing of the meningeal defects.

**Author: Hardik Patel MD**
Additional Authors:Pramod Theetha Kariyanna, MD; Tamera Akarah, MD

Institution:Brookdale University Hospital and Medical Center

**Title:MYOCARDIAL ISCHEMIA FOLLOWING INTRACAVERNOSAL PHENYLEPHRINE INJECTION FOR PRIAPISM SECONDARY TO TAMSULOSIN**

Objective:
1. To appreciate priapism as a rare side effect of Tamsulosin
2. To appreciate myocardial infarction as a potential side effect of intracavernosal Phenylephrine
Case Description:
A 71-year-old black male presented to the ER with painful priapism of 6 hours duration, which was not relieved by ice-pack application. He had a past medical history of BPH, hypertension, and diabetes for which he was on Tamsulosin (for 4 years), Enalapril, and Metformin respectively. He stopped taking all his medications 6 weeks before the symptom onset, as he felt â€œasymptomaticâ€• . However, upon relapse of BPH symptoms he took double (0.8 mg) the regular dose of Tamsulosin the night before the onset of priapism. Physical examination was remarkable for tender, rigid penile erection without any signs of trauma. Right corpus cavernosum aspiration was attempted with 18 G needle, which helped to provide brief relief. Then, he was given intracavernosal Phenylephrine injection, which helped to achieve detumescence. However, within an hour of phenylephrine administration, he developed palpitations. EKG revealed sinus tachycardia with mild ST segment depression in leads II and V6. Laboratory findings demonstrated significant elevation of Troponin-I. He was admitted with the diagnosis of NSTEMI. Medical management followed by successful PCI and stent placement in the obtuse marginal branch of left circumflex artery was performed. He was then discharged on day 3 of hospitalization.
Discussion:
Tamsulosin is a selective a1A antagonist that is prostate specific, but is known to have effect on corporal smooth muscles. Alpha-blockers (ABs) directly inhibit sympathetic stimulation for detumescence. Tamsulosin is the only AB know to improve sexual function, priapism is viewed as one end of this effect. Priapism in patients on Tamsulosin usually follows a high dose or with concurrent use of medication that inhibits its metabolism. Phenylephrine, a pure alpha agonist is the drug of choice, as it has minimal cardiovascular effects. Myocardial infarction following Phenylephrine administration is believed to be due to systemic vasoconstriction causing reflex bradycardia and low cardiac output, which in turn reduces coronary blood flow, especially in elderly patients with pre-existing CAD. Furthermore, peripheral vasoconstriction increases afterload and hence increases myocardial oxygen demand. This phenomenon could have led to the precipitation of myocardial infarction in this patient. This case is remarkable as ischemic priapism is very uncommon side effect of Tamsulosin. In addition, a literature review revealed no previous reports of myocardial ischemia following intracavernosal Phenylephrine injection. The case exemplifies unique challenges that are encountered even with the use of common medications. It is also an eye-opener that necessitates vigilance during the use of drugs that can precipitate myocardial ischemia, especially in high-risk elderly patients.

**Author: Karishma Patel MD**
Additional Authors:Karishma Patel, M.D., Samantha Smalley, Pharm.D., BCPS, Harish Patel, M.D., Joseph Paul, M.D.

Institution: Kingsbrook Jewish Medical Center

**Title:CASE REPORT: SEVERE RHABDOMYOLYSIS WITH ACUTE KIDKEY INJURY SECONDARY TO SEASONAL INFLUENZA A INFECTION IN A YOUNG ADULT MALE**

Rhabdomyolysis is an uncommon but serious complication of influenza. We discuss the presentation and management of a 32-year- old male diagnosed with seasonal influenza A and severe rhabdomyolysis. While previously published case reports reveal creatinine phosphokinase (CPK) levels typically < 50,000 units/L, our patient presented with CPK levels greater than 1,500,000 units/L and acute kidney injury (AKI) requiring hemodialysis. To our knowledge, this is the highest CPK level secondary to influenza infection reported to date.
Our case is notable in several ways. While pH1N1 is notorious for causing severe infections in previously healthy, young or middle age adults, influenza A infections typically cause the most severe complications in very young children, the elderly, and those with significant co-morbid disease states. Our patient was 32 years old, with no significant co-morbidities (except mild MR) and within one day of MICU admission, required intubation and ventilatory support due to respiratory failure. Furthermore, he had severe renal morbidity. Overall, renal complications secondary to influenza A are uncommon. They are even more uncommon in association with influenza A as compared to pH1N1. Our patient presented with extremely severe rhabdomyolysis and AKI. Maximal CPK levels reported range between 522 â€“ 1,150,000 units/L. Our patient had an initial CPK of at least 1,557,174 units/L (our systems used would not quantify higher numbers), which is slightly higher than the highest reported CPK level caused by influenza A infection. By hospital day two, this had only deceased to around 780,000 units/L. Furthermore, our patient required intermittent hemodialysis for one monthsâ€™ duration. Higher CPK levels have been associated with worse outcomes and propensity to develop AKI. Based on this rationale, it is unsurprising that our patient would develop AKI without initial recovery despite aggressive therapy. This patient was also noted to have mild MR. It is possible that he was not able to recognize symptoms of influenza as early as other patients. The CDC recommends that patients with MR receive the influenza vaccination yearly as these patients are more prone to influenza-associated complications.
This case demonstrates that severe renal complications can occur in young, previously healthy patients secondary to seasonal influenza A infection. While rhabdomyolysis and AKI in young patients are typically see with pH1N1, clinicians should be cognizant of this diagnosis in any patient presenting with flu-like symptoms and rhabdomyolysis. CPK levels can increase to levels even higher than reported previously and could potentially be a signal for more prolonged or severe AKI. Furthermore, caretakers of patients with MR should make the extra effort to ensure vaccination of these patients, as MR patients may not be able to readily seek help for their symptoms.

**Author: Eshan Patel MD**
Additional Authors:Galyna Ivashchuk BS, MSc; David Conner, MD

Institution:New York Methodist Hospital

**Title:Unusual case of Superior gluteal artery injury from low energy buttock contusion**

INTRODUCTION

Superior gluteal artery injury is a known but rare complication, occurring most commonly after hip fractures and penetrating abdominal injury, although it has been known to occur after total hip arthroplasty, intramuscular injection, iliac crest bone grafting, and L2 spine fracture. Its origin in the sciatic notch makes it vulnerable to shearing forces of blunt and penetrating trauma. We present a case of superior gluteal artery injury after a mechanical fall.

CASE REPORT

An 83 year old male with medical history significant for hypertension, COPD, sick sinus syndrome, s/p permanent pacemaker placement presents to the hospital after a fall from lower stairs in his house, witnessed by family member. On admission patientâ€™s vitals signs were within normal limits, physical exam was positive for bruises over right forearm and hip. On palpation, there was tenderness of right hip, with decreased range of motion. No numbness, tingling, sensory or motor deficits were noted. No hematomawas palpated. X-Ray of pelvis and CT of brain didnâ€™t show any evidence of fractures or bleeding. Next day, patient complained of increasing pain and decreased range of motion in the right hip. Hemoglobin dropped from 11.1 g/dL upon admission to 8.4 g/dL, blood pressure decreased from 101/64 to 93/54 over 24 hour of period.Medical team also noticed bruise on hip was expanding. CT scan of hip was urgently performed and showed large hematoma within right gluteal musculature. Angiogram showed right superior gluteal artery injury feeding the hematoma. Interventional radiology coil embolized the artery, after which patientâ€™s hemoglobin stabilized and BP returned to 129/65.

CONCLUSION

Superior gluteal artery injury is a rare complication of traumatic abdominal and pelvic injuries. Case reports predominantly describe immediate hemodynamic instability following traumatic injury, although some authors report delayed hemodynamic changes after injury (after 48 hours). Delayed hemodynamic changes may be due in part to injury of smaller branches of superior gluteal artery, leading to a slower, prolonged bleeding. Bleeding can be extensive enough to cause compartment syndrome of the buttock, presenting with unrelenting pain resistant to analgesics and swelling. Treatment usually includes arterial embolization. If compartment syndrome is present, fasciotomy may also be considered in the treatment plan to prevent muscle and nerve necrosis, along with rhabdomyolosyis and kidney injury. Other known complications of delaying decompression of the gluteal compartment are increased risk of infection and permanently weakened lower extremity. Our case is unique because the cause was identified before the patient became hemodynamically unstable and patientâ€™s only complaint was pain. Our patient did not have any type of severe trauma usually associated with superior gluteal artery rupture, nor did he have the typical symptoms of compartment syndrome.

**Author: Siddharth Raghavan M.D.**
Additional Authors:Siddharth R. Raghavan1 MD (ACP member), Sahar Ahmad2 MD. 1Department of Internal Medicine, Stony Brook University Hospital, Stony Brook, NY. 2Department of Internal Medicine, Division of Pulmonary and Critical Care Medicine, Stony Brook University Hospita

Institution: Stony Brook University Hospital

**Title:CASE REPORT: SEVERE VITAMIN C DEFICIENCY VEILED BY PSEUDOVASCULITIS**

Background: Scurvy, first described in the Ebers papyrus in 1500 BC, is now an uncommon disease process. It results from ascorbic acid (vitamin C) deficiency, and in modern times, at risk groups include alcoholics, malnourished, and economically disadvantaged persons. We report a unique case of scurvy masquerading as vasculitis in a patient with severe food intolerance.

Case Presentation: A 23-year-old man with irritable bowel syndrome presented with lightheadedness and syncope. One month prior to presentation the patient reported myalgia, arthralgia, fatigue, and a rash over his lower extremities. Two weeks later he became increasingly lightheaded and on the day of admission, he lost consciousness while ascending stairs at home.

On presentation, his temperature was 38.1oC. Orthostatic vital signs were BP 114/62, pulse 100 (supine) and BP 82/49, pulse 117 (standing). Physical examination revealed erythematous gums; tenderness and swelling in the right thigh, diffuse perifollicular petechiae over the lower extremities, and corkscrew hairs. Initial laboratory findings were WBC 2.86 x 109/L, Hgb 5.4 g/dl, platelet 381 x 109/L, MCV 87.4 fL, ESR 67 mm/hr, INR 1.2, total bilirubin 1.9 mg/dl, and direct bilirubin 0.6 mg/dl. Peripheral smear demonstrated normocytic, normochromic anemia without evidence of hemolysis. Computerized tomography scan demonstrated a thigh hematoma measuring 3.7 x 7.0 cm.

The patient received six units of PRBCâ€™s over the next three days with an inadequate response (Hgb 7.2). The diagnosis of vasculitis was suspected; an autoimmune workup revealed a low titer ANA 1:40 (speckled) with negative ANCA and extractable nuclear antigen antibodies. Skin biopsy of the rash demonstrated perifollicular hemorrhage and hemosiderin deposits but was negative for vasculitis. Esophagogastroduodenoscopy revealed gastritis and multiple intramucosal hemorrhages within the gastric antrum. Workup for nutritional deficiency revealed an undetectable plasma vitamin C level (< 0.08 mg/dl). The patient was started on oral vitamin C 1000 mg daily and midodrine for vasomotor instability. Hgb trended upward to 11.2 and he was discharged soon thereafter. The patientâ€™s rash disappeared quickly and he had complete resolution of his other symptoms one month after discharge.

Discussion: Scurvy is a clinical syndrome classically characterized by sacral osteopenia, petechial rash, and spontaneous bleeding late in the disease course. Presenting symptoms vary and can mimic other rheumatologic disorders, often resulting in delayed diagnosis. Our patient had completely restricted dietary consumption of fruits and vegetables for eight months prior to admission in an effort to eliminate symptoms of abdominal discomfort and bloating. He required multiple hospitalizations and was misdiagnosed with leukocytoclastic vasculitis before severe vitamin C deficiency was discovered. This case highlights the absolute importance of a detailed history and the danger of anchoring heuristics. Additionally, it underscores the timeless relevance of poor dietary intake and chronic malabsorption as risk factors for scurvy.

**Author: Mohammed Samannodi MBBS**
Additional Authors:Mohammed Samannodi, Yaser Nemshah, Kevin Shiley.

Institution:University at buffalo/ Catholic Health System

**Title:Rapidly progressive Plesiomonas Shigelloides bacteremia and septic shock in a post-splenectomy patient with hemochromatosis: A Case report.**

Introduction:
Plesiomonas shigelloides infection in immunocompromised individuals is associated with high mortality rates, especially in aspleinc patients. Sepsis secondary to this organism has also been reported in patients with hemochromatosis. We report a fatal case of Plesiomanas septicemia despite early aggressive antimicrobial therapy.
Presentation:
A 43 year-old Caucasian male patient was brought to the hospital with a three hour history of fever, chills, and confusion. The patient had no history of recent travel or sick contact. He had a new puppy at home that bit and scratched him occasionally. The patient had eaten clams which were prepared at home one week prior. In addition, patient swam numerous times in Lake Erie over the past several weeks. The beach he swam from was closed several times by the local health department during the preceding weeks due to high coliform counts. On exam, the patient was hypotensive, tachycardic, tachypneic and confused. Laboratory evaluation revealed leukocytosis with bandemia, macrocytic anemia, renal and liver failure and lactic acidosis. A diagnosis of septic shock was made. Empiric coverage with ceftriaxone and intravenous vancomycin was initiated along with aggressive volume resuscitation. He subsequently developed respiratory failure and later underwent hemodialysis due to worsening renal failure. On day two, gram negative rods were reported from blood cultures. Disseminated intravascular coagulation developed with worsening multi-organ failure. Levofloxacin and tobramycin were subsequently added to the empiric antimicrobials as the patientâ€™s condition worsened. On day three, the final blood culture identification was made as Plesiomonas shigelloides . Despite aggressive resuscitation measures the patientâ€™s condition worsened and he expired.
Conclusion:
Plesiomonas shigelloides normally resides in fresh water environments; including the Great Lakes. Plesiomonas species rarely cause significant disease in people with normal immune systems. However, a rapidly progressive septic picture can evolve in asplenic patients with Plesiomonas infections. Early diagnosis of septic shock with appropriate antimicrobials and volume resuscitation is recommended. In addition, systemic corticosteroid administration may also be beneficial for refractory shock as Plesiomonas septicemia has been associated with adrenal insufficiency secondary to adrenal infarction in the setting of septic shock.

**Author: Gisell Yuriana Sanchez MD**
Additional Authors:Samuel Bavli MD FACE.

Institution: St Johns Episcopal Hospital

**Title:A CASE OF A MALIGNANT HYPERFUNCTIONING (â€œHOTâ€ • ) THYROID NODULE**

Background: Most thyroid nodules are benign, and only 10 to 15% are malignant. When confronted with a patient having a thyroid nodule, it is incumbent on the physician to the likelihood of malignancy.If the patient is hyperthyroid, a thyroid scan is done to confirm that the nodule is hyperfunctioning (a â€œhotâ€ • nodule). Since thyroid cancer in a â€œhotâ€ • nodule is rare, biopsy of such a nodule generally is not indicated.
Case: A 68-year-old woman presented in early October 2006 after discovering a mass in the left side of her neck, and later that month she started having hoarseness. CT scan showed a 4.6 x 4.0-cm soft-tissue mass inseparable from the left thyroid lobe, and multiple hypodense nodules in both thyroid lobes. TFTs showed mild hyperthyroidism, and thyroid scan confirmed a hot nodule in the left thyroid lobe. She was treated with 131I and subsequently reported some shrinkage of the mass, but hoarseness did not improve. She then consulted a different doctor, who performed laryngoscopy, laryngeal biopsy, and thyroid fine-needle aspiration (FNA) biopsy. Both the laryngeal and the thyroid biopsies showed papillary thyroid carcinoma. Total thyroidectomy was performed, but invasion of the larynx, cricoid cartilage, and sternocleidomastoid muscle precluded complete resection of the cancer. Post-surgical131Iwhole-body scan showed two foci of uptake in the neck, probably lymph nodes, and she was treated with 220 mCi of 131I. Pathology revealed invasive papillary carcinoma with focal oncocytic/Hürthle cells, follicular, and papillary patterns involving the entire gland. Due to the aggressive nature of her disease, she also received external beam radiation from November 2007 to February 2008, but serum thyroglobulin never declined to zero. Because of a relentlessly increasing thyroglobulin, she was treated twice more with 131I: 318 mCi in 2010, and 268 mCi in 2012. In 2010, 131I scan using 5 mCi of 131I failed to show any uptake, but whole body scan after the 318-mCI treatment dose showed diffuse uptake in the liver. Despite treatment, TG continue to rise, reaching 85.2 ng/ml (reference range 2.0-35.0) in July 2013. Chemotherapy with sorafenib was discussed with her, but she switched doctors in late 2013, because of insurance concerns, and she was lost to follow-up.
Discussion: Although hyperfunctioning thyroid nodules are almost invariably benign, the possibility of thyroid cancer should still be entertained in the presence of certain findings such as recent onset of hoarseness, a hard nodule on palpation, or fixation to adjacent tissue. In such cases, FNA biopsy should be performed.

**Author: Disha Shah**
Additional Authors:Szuhuei Wilson, M.A., Roxana Lazarescu, M.D.

Institution:New York Hospital Queens

**Title:Antibiotic â€“ associated diarrhea â€“ Is this Clostridium difficile colitis?**

Introduction: Patients with Inflammatory Bowel Diseases (IBD) may be at increased risk for colitis secondary to Staphylococcus aureus, especially in the setting of a history of antibiotic therapy.
Case Presentation: This is an 81 year-old female with a history of Ulcerative Colitis (on Mesalamine), Coronary Artery Disease, Hypertension, and Hypothyroidism who presents with voluminous, bloody diarrhea, nausea, vomiting, and inability to tolerate oral intake. She was hospitalized for generalized weakness a week prior to admission and was found to have a Urinary Tract Infection and colitis at that time. She was sent home on Levaquin and Flagyl. On physical examination, the abdomen was soft, non-tender, non-distended, and bowel sounds were present in all four quadrants. CT scan of the abdomen with contrast performed on admission showed descending and sigmoid colitis. Stool studies, including culture, ova and parasites, and Clostridium difficile toxin were sent. Stool culture was found to be positive for Staphylococcus aureus and the remaining studies were negative. Initially, patient was continued on Flagyl but when stool culture results were noted, she was started on oral Vancomycin 125 mg every 6 hours. Patient had significant symptomatic improvement with the initiation of oral Vancomycin. She was discharged home on a 2-week regimen of Vancomycin.
Discussion: Staphylococcal enterocolitis is usually associated with prior use of antimicrobials, recent abdominal surgery, prior proton pump inhibitor therapy, and immune-compromising conditions. In this context, IBD patients on immunosuppressive therapy and recent antimicrobial use may have an increased risk in developing Staphylococcal colitis. This patient population has been described in various case reports without sufficient large trial studies to provide concrete recommendations for treatment. In a comparative analysis, Asha and colleagues found C. difficile toxin to be sixty times more common than S. aureus on stool examination. In 1960s and 1970s, the use of Lincomycin and Clindamycin, which effectively inhibits S. aureus but has limited activity against C. difficile, led to the dominance of C. difficile associated diarrhea. The recent increase in the use of Metronidazole which has activity against C. difficile but has poor activity against S. aureus, may be a contributory factor to the reemergence of staphylococcal enterocolitis. Physicians should consider S. aureus associated colitis as an alternative diagnosis to C. difficile colitis in patients with prior antibiotic use and IBD, and they should be aware that treating a patient with Metronidazole for C. difficile colitis might predispose the patient to S. aureus colitis.

**Author: Suvash Shrestha MBBS**
Additional Authors:Israel Jacobowitz, MD, Department of Cardiothoracic surgery, Yisachar Greenberg, MD, Department of Cardiac Electrophysiology,
Felix Yang, MD, Department of Cardiac Electrophysiology, Maimonides Medical Center

Institution: Maimonides Medical Center

**Title:BOWEL LOOPS IN PERICARDIUM: AN UNUSUAL COMPLICATION OF CONVERGENT ABLATION OF ATRIAL FIBRILLATION**

Introduction
Convergent ablation has high success rate for treating persistent and permanent atrial fibrillation. It combines epicardial and endocardial ablation of left atrium. It is minimally invasive, and has limited reported complications: pericardial effusion, new third degree AV block, and esophageal fistula.[1,2] We present a previously unreported case of pericardial hernia presenting eight months after the ablation.
Case Description:
A 60 year old apparently healthy lady was being treated for recurrent paroxysmal atrial fibrillation (AF) with beta blockers and anti-arrhythmic medications without satisfactory control of her symptoms. She underwent ablation of her AF using endocardial approach, yet continued to have AF and atrial flutter post ablation. She therefore underwent convergent ablation. The cardiothoracic surgeon, through a trans-diaphragmatic pericardial access, created linear epicardial lesions along the posterior region of the left atrium. Subsequently, the electro-physiologist, through endocardial approach, created endocardial lesions to complete the ablation.
The procedure went well without any complications and she was discharged with rivaroxaban and amiodarone, which were discontinued after three months. She did not have any more symptoms of AF. However, eight months after the procedure, on a regular follow up, she complained of constant bloating. It was not severe enough for her to seek medical attention but had been present for a few months.
On examination, chest was normal with regular heart sounds and adequate air entry over all the lung fields. Abdomen was soft and non tender. Routine labs were within normal limits, as were her ECG and Chest X-ray. However, her echocardiography showed an echogenic mass anterior to her heart and computerized tomography of her chest showed diaphragmatic hernia with portions of greater omentum and transverse colon extending into the pericardium. Laparoscopic diaphragmatic hernia repair was performed and the pericardial opening was closed with figure of 8 sutures. She was discharged a day later without any complications. She has been doing fine without any symptoms four months after the surgery.

Figure 1. CT chest showing pericardial hernia in transverse view
Discussion:
Herniation of the bowel into the pericardial space is a rare complication of convergent ablation procedure. Factors which may have contributed to this complication include the size and location of the pericardial access as well as the position and size of the liver. A more posterior pericardial access may reduce the incidence of this complication. Some operators also choose to close the pericardial window at the end of the procedure.
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**Author: Sapna Siddharth MD**
Additional Authors:TS Dharmarajan MD, MACP
Institution:Montefiore Medical center, Wakefield Campus

**Title:POST-HEMORRHAGIC ANEMIA IN AN OLDER ADULT WITH ABDOMINAL AORTIC ANEURYSM, ANOREXIA AND FATIGUE: ADDRESSING TRIVIAL CLUES HELP DIAGNOSIS AND OUTCOMES!**

Introduction:
Lethargy and anorexia are common non-specific complaints; however, they could make a difference when addressed in an older adult with Abdominal Aortic Aneurysm ( AAA)
Case:
An 89 year Spanish speaking functionally independent community female with hypertension and AAA for 7 years presented with anorexia, fatigue, melena and weight loss of 25 pounds over two weeks. Labs: Hb/Hct-7.4/23.0, a drop from 9.5/29.7 one month ago. BUN 47 mg%, creatinine 2.9 mg%. The patient demonstrated orthostatic hypotension (BP lying, standing). Physical exam confirmed a pulsatile abdominal mass.No blood pressure difference between both arms was appreciated. Stool was positive for occult blood. Chest X ray was normal. Based on these, the likelihood of a dissecting aneurysm was low. While in hospital, she complained briefly of new onset chest pain. EKG: ST segment depression in infero-lateral leads; troponins trended up. Echocardiogram: large apical infarction, ejection fraction 34%. CT abdomen and pelvis: AAA 6.4 cm X 6.2 cm with possible intramural hematoma. She received transfusions; anticoagulation for the NSTEMI was held in view of the hematoma. Endoscopic intervention was deferred in view of no active bleed. The hemoglobin remained stable. The patient and family decided against surgery for the AAA and opted to go home with possible Hospice care.
Discussion:
This elderly patient presented with extreme fatigue of recent onset due to a cause, in this case, likely anemia. The cause of anemia was likely to be a bleed from an enlarging AAA, previously known to be stable for 7 years. The patient and her familyâ€™s attitude was a lack of concern for the AAA as a possible source of the problem given its known stability in the past and being painless at present. The patient also dismissed her complaint of chest pain as insignificant compared to the fatigue she was experiencing. Addressing all patient comorbidities, however insignificant, resulted in the early recognition of a new coronary event and also addressing the matter of the AAA, although the patient only had little by way of complaints other than fatigue.
Lessons learnt
-Manage patients in the context of entire comorbidity, even if some disorders appear chronic or stable.
-Although AAAs are typically longstanding or stable, rupture is unpredictable and can occur, resulting in catastrophe.
-The AAA here was large, warranting consideration for correction.
Reference:
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**Author: Gurpreet Singh PGY-2**
Additional Authors:Anand Kumar MD (member); TS Dharmarajan MD, MACP

Institution: Montefiore Medical Center (Wakefield), Bronx, New York.

**Title:A RARE CAUSE OF MYOCARDIAL INFARCTION IN THE YOUNG: POST-PARTUM SPONTANEOUS CORONARY ARTERY DISSECTION**

Introduction
Acute myocardial infarction (MI) is uncommon under age 40, especially in women. Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS) in the peri-partum period, as demonstrated in this case, where emergent coronary artery bypass grafting (CABG) was required.
Case
34 years old female presented to ER with severe sub-sternal chest pain that began 2 days back as intermittent, pressure like, exertional and worsened overnight to 10/10 intensity, associated with dyspnea. She was 3 weeks post-partum after an uncomplicated pregnancy and delivery. Patient denied smoking, alcohol intake and family history of cardiac disease. Physical exam was unremarkable. Labs: Hemoglobin 11.5 g/dl, Troponin-T 0.38 ng/ml (normal range: 0.00- 0.10 ng/ml), CPK 743 U/L. EKG: T wave inversion in antero-lateral leads and QTc 515 ms. Unfractionated heparin drip was started. Left heart catheterization revealed left main stem (LM) dissection with occlusion of true lumen by false lumen resulting in 60% LM stenosis with intramural hematoma, extending into left anterior descending (LAD) resulting in mid-LAD 95% stenosis. Ventriculography showed ejection fraction (LVEF) 40% with anterior and infero-apical wall severe hypokinesia. She underwent 3-vessel CABG with good results and uneventful post-op recovery. Echocardiogram after one month showed normal LVEF without wall motion abnormalities. She was asymptomatic on follow up.
Discussion
Prior to advent of cardiac catheterization, SCAD was often missed, barring autopsy diagnosis. Hormonal, hemodynamic and morphological changes in vasculature during pregnancy may be pathogenetic. Most cases occur in late third trimester to early post-partum period and involve LM and LAD. Classic CAD risk factors are not the predispositions, as the coronaries are usually normal. The presentation is that of ACS or sudden death. Management relates to coronary angiography findings: conservative treatment with medications, percutaneous coronary intervention (PCI) and CABG. Thrombolysis is relatively contraindicated (risk of propagation of dissection). PCI is the choice in SCAD with involvement of a single vessel with ongoing ischemia whereas CABG is indicated in patients with LM dissection (as in our case), multi-vessel dissection or in patients with failed PCI. Short-term mortality in SCAD approaches 50%, but early survival equates good long-term outcome.
Learning Points:
- A high index of suspicion of SCAD is warranted in peri-partum females presenting with chest pain, in the context of absent cardiac risk factors.
- Urgent coronary angiography and appropriate management are the key to favorable outcome.
Reference
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**Author: Aisha Siraj PGY-3, Internal medicine**
Additional Authors:Neyman,Michael.M.D, Nannaka,Varalaxmi.M.D,.Lvovsky,Dmitry.M.D.

Institution:Bronx Lebanon Hospital

**Title:A RARE CASE OF ACUTE MYOCARDIAL INFARCTION DUE TO CORONARY VASOSPASM IN A 27 YEAR OLD PREGNANT WOMAN WITH GESTATIONAL TRANSIENT THYROTOXICOSIS**

Introduction
Acute myocardial infarction (AMI) in pregnancy is rare. It is exceedingly uncommon to be caused by a coronary vasospasm, a case which we describe below.
Case Report
A 27 year old nine weeks pregnant woman presented to our hospital complaining of fatigue for three week duration and nausea with vomiting and atypical chest pain for two weeks.
She was hemodynamically stable with unremarkable physical examination. Her initial EKG showed normal sinus rhythm with ventricular rate of 72. Her labs were remarkable for hypokalemia (2.4mEq/L), metabolic alkalosis (Bicarbonate: 30mEq/L, pH: 7.59) and elevated transaminases (ALT: 549 unit/L, AST: 285unit/L). Urine toxicology was positive for cannabinoids. Patient was admitted to the intensive care unit with a working diagnosis of Hyperemesis Gravidarum. Twelve hours later after admission to ICU, patient complained of worsening of chest discomfort. Repeat EKG showed 3mm ST segment elevations in leads II, III and AVF. Intravenous heparin, beta-blocker, aspirin, clopidogrel and statin were started. Echocardiogram showed left ventricular ejection fraction (LVEF) of 48% with inferior and inferoseptal akinesis. Chest computed tomography with contrast did not reveal pulmonary embolism or aortic/ coronary dissection. Additional labs revealed troponin T 1.410(ng/ml) peaking at 3.600(ng/ml) after 12 hours, TSH of <0.07mIU/L; T4 24.8µg/dl; and T3 377µg/dl. Workup for Graveâ€™s disease was negative. She was diagnosed with gestational hyperthyroidism and received dexamethasone prior to cardiac catheterization which revealed right coronary artery vasospasm. The initial finding of long segment narrowing of the right coronary artery reverted back to normal lumen size after intracoronary nitroglycerine injection, confirming diagnosis of Prinzmetal angina. She underwent elective termination of pregnancy. Two days after coronary angiogram, patient developed ventricular fibrillation with cardiac arrest, which responded to cardioversion. LVEF decreased to 37% on repeat ECHO. The patient was transferred to another institution for cardiac MRI and ICD placement.
Discussion
AMI in pregnancy is rare and occurrence of Prinzmetalâ€™s angina is less known. It accounts for 2% of all cases of angina. The etiology of the vasospasm is unknown; however, it has been associated with hyperthyroidism in some cases. Gestational hyperthyroidism occurs approximately 1-2 cases/1000 pregnancies and usually is mild not requiring treatment. Studies suggest treatment with vasodilators and achievement of euthyroid state has been associated with resolution of vasospasm; however this was not curative in our patient.
Conclusion
This is a rare case of prolonged coronary vasospasm leading to AMI and cardiac arrest despite optimal medical therapy in a patient with gestational transient hyperthyroidism. This case highlights the importance of considering gestational transient hyperthyroidism in the differential diagnosis of coronary artery spasm.

**Author: Michael Tanoue MD**
Additional Authors:Monika K. Shah, MD

Institution: New York Presbyterian Hospital - Weill Cornell

**Title:Local Action of Nebulized Ipratropium Bromide Causing a Unilateral Fixed Dilated Pupil**

We report a case of unilateral mydriasis as a result of ocular exposure related to mask nebulization of Ipratropium Bromide. In this case, the patient had no cognitive impairment and was unaware of the unilateral mydriasis as she had chronic blurred vision related to distant chorioretinitis. A CT of the head was performed emergently and was unremarkable. Further ophthalmologic exam demonstrated a chorioretinitis scar on the right eye without evidence of vascular abnormalities or findings indicative of a vasculitic or infectious process. Pilocarpine test was negative. Nebulizer treatment was subsequently discontinued with resolution of mydriasis within 24 hours. This case-report illustrates unilateral ocular mydriasis as a direct consequence of local action of Ipratropium Bromide on cholinergic receptors of the eye. It also sheds light on less common pharmacologic causes of mydriasis, which should be investigated in patients with normal mental status and a new finding of unilateral fixed mydriasis prior to embarking on more invasive and/or costly interventions.

**Author: Hassan Tariq M.D.**
Additional Authors:Bibi Ayesha, MD1; Karen M. Weidenheim, MD2; Giovanni Franchin, MD1.
Department of Medicine1, Bronx-Lebanon Hospital Center1, Department of Pathology2, Montefiore Medical Center2.

Institution:Bronx Lebanon Hospital Center

**Title:ETANERCEPT-INDUCED MYOSITIS: DO WE HAVE TO STOP IT? A SURPRISING OUTCOME.**

BACKGROUND
Antiâ€“tumor necrosis factor alpha (anti-TNF) agents are widely used to treat a variety of rheumatic and autoimmune diseases including psoriasis. In rare cases, these agents can induce inflammatory myositis which is usually managed by cessation of the anti-TNF agents and may require additional therapy with corticosteroids and immunosuppressive medications to manage the underlying disease.
CASE
Our patient presented here is a 47 year old man diagnosed with severe psoriasis and psoriatic arthritis at age 40 who was refractory to methotrexate, leflunomide and systemic steroids. He had recurrent skin and arthritic flares requiring frequent hospitalizations and intravenous steroid therapy. Etanercept 50mg subcutaneously once weekly was started, resulting in nearly complete resolution of psoriatic plaques and marked improvement of arthritis during a short period of time. Six months after initiation of anti-TNF therapy, patient presented complaining of generalized myalgia, progressive weakness in upper and lower extremities and unstable gait of 2 weeks duration. On examination, vitals were normal, musculoskeletal exam revealed synovitis of left wrist and bogginess of right hand proximal interphalangeal joints. Upper and lower proximal muscle strength was decreased to 3/5 and patient had an unstable gait. Deep tendon reflexes were normal. Laboratory tests showed elevated transaminases with aspartate transaminase of 173 U/L, alanine transaminase 497 U/L. The serum creatine kinase was found to be 5666 U/L, lactate dehydrogenase 1265 U/L, C-reactive protein 51.69 mg/dL, ESR 112 mm/hr, and antibody to JO-1 was negative. A muscle biopsy was performed and showed perifascicular muscle fiber atrophy with perimysial inflammatory activity .This presentation was suggestive of Etanercept-induced myositis. The patient was advised to stop Etanercept and was started on prednisone 60 mg once a day. His muscle strength fully recovered, gait improved with no imbalance and serum creatine kinase normalized within three months. Despite our recommendation to stop Etanercept, patient continued using it due to his concern of experiencing a possible flare of psoriasis. Oral steroids were subsequently tapered off in the next couple of months and patient has remained stable on Etanercept.
DISCUSSION
Etanercept is used in the treatment of moderate to severe psoriasis and psoriatic arthritis. There has been rare cases of anti-TNFâ€“induced inflammatory myositis. Cases described in the literature usually have a good outcome after discontinuation of anti-TNF, however a challenge remains to treat the underlying auto-immune disorder, psoriasis or psoriatic arthritis. This is the first description to our knowledge of an anti-TNF-induced myositis responding favorably to a short course of steroids despite continuation of anti-TNF.

**Author: Augusta Uwaje MD**
Additional Authors:Anthony Iluyomade,Anthony Umoh,Saira Shahab

Institution: St Johns Episcopal Hospital

**Title:TWO CASES OF LIFE THREATENING INVASIVE THORACIC CAVITY INFECTIONS SECONDARY TO STREPTOCOCCAL ANGINOSUS**

Streptococcal anginosus, a sub group of streptococcal viridans, consists of three distinct streptococcal species: Strep. anginosus, Strep. intermedius and Strep. constellatus. These organisms are normal commensals of the human oral cavity but they also possess the ability to cause unusual life-threatening invasive pyogenic infections which are often difficult to treat and may require interventional procedures.
Case 1: A 59 year old male with medical history of HTN, DM, A-fib, COPD, chronic back pain and Peripheral vascular disease was sent from NH due to two weeks history of palpitations and cough. Physical examination was remarkeable for edentulous patient with Oxygen saturation of 85% , temp of 102F. Labs showed WBC- 13,000 /cubic metre with 80.5% neutrophils. Echo revealed pericardial tamponade with no evidence of endocarditis. CT scan of chest and abdomen was unremarkeable for deep seated abscesses. Emergency pericardial window was performed. Pericardial fluid analysis revealed wbc of 5550, neutrophil of 94% and pleural fluid culture grew Strept anginosus which was sensitive to Ceftriaxone. Patient was later discharged after significant improvement and antibiotic therapy.
Case 2: A 64 yr old woman with medical history of HTN, DM, Hypothyroidism, coronary artery disease, schizophrenia and dementia was admitted from nursing home due to fever, lethargy and AMS. Vitals were temp of 97F, HR: 101BPM, BP: 149/77, RR: 18CPM and oxygen saturation of 98%. Physical examination was remarkable for poor dentition and markedly reduced breath sounds on the left lung field. Labs in the ER revealed WBC of 14,300 / cubic meter with neutrophil count of 87.5%. Blood culture grew streptococcus epidermis. Chest X-ray showed opacification of the left hemi-thorax and CT chest revealed large left pleural effusion with ipsilateral compressive atelectasis. Abdomino-pelvic and neck CT was negative for abscess. Echocardiography showed no evidence of endocarditis.
Percutaneous left sided chest tube was placed draining 1200cc of brownish purulent fluid. Pleural fluid analysis revealed WBC of 361,111 with 87% neutrophils, total protein of 3.0, albumin: 0.9, LDH: 181, Glucose: 66. Pleural fluid culture grew streptococcus anginosus.
She was treated for pneumonia with Moxifloxacin and Vancomycin. After one week of antibiotics and chest tube drainage, repeat CT scan revealed persistent pleural effusion which prompted thoracotomy and decortications. She felt better and was later discharged.
DISCUSSION:The unique characteristic of the Strep. anginosus group that sets them apart from other pathogenic streptococci, such as S. pyogenes is their ability to cause deep seated abscesses .To the best of our knowledge, only few cases of life-threathening invasive intrathoracic infections involving the pericardium and pleural space necessitating urgent medical and open surgical interventions have been described. Therefore Strep. anginosus group should be considered true pathogens in a symptomatic individual if isolated.

**Author: Neil Vyas**
Additional Authors:Rafael A. Ching Companioni, MD
Melik Tiba, MD
Loveleen Sidhu, MD
Christopher Tomaino, MD
Aaron E. Walfish, MD

Institution:Elmhurst Hospital Center

**Title:Pegaspargase induced severe pancreatitis. Friend or foe?**

Introduction: Pegaspargase (Oncaspar) is a modified version of L-asparaginase conjugated with polyethylene glycol. In leukemic cells, asparaginase hydrolyzes L-asparagine to ammonia and L-aspartic acid leading to depletion of asparagine. Leukemia cells need exogenous asparagine, thus asparagine depletion in leukemic cells leads to inhibition of protein synthesis and apoptosis. Despite its potential benefits there are a wide range of side effects. One rare but potentially deadly complication is pancreatitis.
Case: 24 year old Mexican male with a history of acute T-cell lymphoblastic leukemia (ALL) on recent chemotherapy including pegaspargase, admitted for abdominal pain, found to have acute pancreatitis secondary to hypertriglyceridemia. On examination, tachycardia of 127, decreased air entry in right lung bases, and a severely tender abdomen. Laboratory tests were remarkable for elevated liver enzymes ALP 360 U/L, AST 310 U/L, GGT 216 U/L, ALT 44 U/L, LDH 829 U/L, elevated lipase 228 U/L, and hypertriglyceridemia >3,000 mg/dl. Abdominal CT showed pancreatitis with necrosis; peripancreatic, intraperitoneal and retroperitoneal fluid. Subsequently his severe pancreatitis was associated with acute kidney injury and respiratory failure which is illustrated by his BUN 22 mg/dl, creatinine 2.16 mg/dl, and persistent hypoxia. According to the Atlanta classification, patient is classified under severe acute pancreatitis. In addition, patientâ€™s BISAP score was 3, which establishes the high mortality risk. Despite appropriate treatment for pancreatitis according to current recommendations, he expired.
Discussion: Pegaspargase is used for treatment of ALL and is gaining in popularity over asparaginase therapy due to it having fewer incidences of hypersensitivity reactions and because of its long half life (367 hours) allowing dosing every 14 days as opposed to asparaginase which is dosed daily(1). Pegaspargase definitely has its benefits but we canâ€™t lose sight of one of its rare, but potentially deadly complications, pancreatitis. In one study nine of the 50 patients (18%) with ALL treated with pegaspargase were diagnosed to have pancreatitis. In contrast, only one out of 52 (1.9%) ALL patients who received native E. coli L-asparaginase during the same time period developed pancreatitis (1). One proposed mechanism of this drug-induced pancreatitis is hypertriglyceridemia, which is seen in our case. It is suggested that apolipoprotein E polymorphism may influence the development of hyperlipidemia in patients receiving pegaspargase(2).
We report a case to increase the awareness of higher incidence of pegaspargase-induced pancreatitis, which is a rare but potentially deadly complication. Clinicians should monitor triglycerides while on treatment and suspect pancreatitis if patient develops abdominal pain. If pancreatitis occurs, therapy should be stopped and not reinstituted. For patients with hypertriglyceridemia without pancreatitis, discontinuation of therapy should be considered.

**Author: Rahul Yadav MBBS**
Additional Authors:Dr. William Pascal, Department of Pulmonary Disease, Maimonides Medical Center

Institution: Maimonides Medical Center

**Title:PNEUMOCYSTIS PNEUMONIA IN A PATIENT TREATED WITH TEMOZOLOMIDE FOR MALIGNANT OLIGODENDROGLIOMA**

A 45 year old Asian female with hypertension presented to our ER with fever for 3 day along with cough and lightheadedness. She was diagnosed with Grade III anaplastic oligodendroglioma 6 months ago and had received her fourth cycle of Temozolomide about 10 days prior to presentation. Upon presentation, she was febrile with a temperature of 101.9° F, tachycardic and tachypnic. Her Oxygen saturations were 80 % on room air and 90 % on 4L nasal canula. She was in obvious respiratory distress, had tubular breath sounds in right upper lobe and scattered fine crackles in all lung fields. She had 4200 K/UL WBC with 78 % neutrophils, hemoglobin of 11.4 gm/dl and platelets of 154 K/UL. Her serum chemistries showed calcium of 8.2 mg/dl. Her blood gas analysis revealed hypoxemia with an A-a gradient of 45 mmHg. Serum LDH was elevated at 550 IU/L. Serum 1, 3 beta-D-glucan was elevated at >500 pg/ml. Chest x-ray suggested CHF. CT chest showed interstitial edema, bilateral air space disease and nonspecific ground glass opacification.
The patient was admitted for sepsis presumed due to health care associated pneumonia complicated by acute hypoxemic respiratory failure. She was started on broad spectrum antibiotics and intravenous Sulfamethoxazole-Trimethoprim for Pneumocystis pneumonia along with steroids. During her hospital stay, she refused any mechanical ventilation and was maintained on nasal canula and face mask. Upon symptomatic improvement, she was discharged with oral antibiotics and a tapering dose of oral prednisone.
Oligodendroglioma is a type of glial tumors. Temozolomide is an antineoplastic agent approved by FDA for treating newly diagnosed glioblastoma multiforme (GBM). It is recommended that prophylaxis against Pneumocystis pneumonia is required for all patients receiving concomitant Temozolomide and radiotherapy for the 42-day regimen. It is postulated that Temozolomide causes lymphopenia, specifically CD4 helper cells. Our patient had hypoxemic respiratory failure with elevated A-a gradient, elevated LDH and 1, 3 beta-D-glucan and a CT scan showing ground glass opacities. Blood tests did not reveal any evidence of any invasive fungal infections, which could falsely elevate 1, 3 beta-D-glucan. To our knowledge, this is the first case of Pneumocystis pneumonia in a patient treated with Temozolomide in the continental North America. Our patient survived this episode without any need for ventilatory support and resumed her treatment course. This highlights the importance of maintaining high clinical suspicion in patients receiving Temozolomide, giving prophylaxis while on therapy and prompt referral of the patient to a hospital with ICU facilities.

**Author: Rahul Yadav MBBS**
Additional Authors:Sunita Verma MBBS, Renu Muttana MD, KC Janga MD
Institution:Maimonides Medical Center

**Title:'TREATMENT OF HIGH OUTPUT FAILURE IN A HEMODIALYSIS PATIENT BY RADIO-CEPHALIC FISTULA REVISION AND GRAFT**

An 82 year old Caucasian male with end stage renal disease (ESRD) on hemodialysis for the past 8 years, presented to the ER with weakness and difficulty breathing during hemodialysis leading to premature termination of multiple dialysis sessions. He has had multiple CHF exacerbations and pleural effusions prior to this admission. Physical exam, laboratory tests and radiographic exams were consistent with CHF exacerbation, pleural effusion and rapid atrial fibrillation. He had a left arm radio-cephalic AV fistula that was clean, dry and without any evidence of infection. There was normal pulsatility and bruit with good augmentation and collapse with limb elevation. The patient was admitted for acute respiratory failure and inability to perform efficient hemodialysis. Upon hospitalization, he was treated for pneumonia and atrial fibrillation. Temporary central venous access was established to maintain continuity of hemodialysis. Duplex study showed a fistula with flow (Qa) = 7000 ml/min and highly tortuous anatomy with aneurysmal dilatation (pic -1). Patient underwent resection of aneurysm and placement of a 7mm PTFE interpositioning graft at the level of anastomosis, functionally converting AV fistula into AV graft. He was discharged and later seen in vascular clinic after 3 months. Repeat duplex scan showed successful AV fistula revision and reduction of the flow, Qa = 2600 ml/min. Subsequently he resumed dialysis through the revised AV fistula, which had been transformed into a graft with limited flow, resulting in resolution of atrial fibrillation and recurrent CHF exacerbations and hospitalizations. The end point of progressive chronic renal failure is renal replacement therapy. Hemodialysis through an arterio-venous fistula (AVF) is the most preferred option due to its efficacy, practicality, minimal potential for infection and durability. High Output Cardiac Failure (HOCF) is a rare but serious complication of AVF. Male sex, preexisting heart disease, upper extremity fistula, Qa > 2000 ml/min, ratio of Qa to Cardiac output > 0.3 increase risk of HOCF. Although medical management is tried first, many patients eventually need surgical intervention. The existing options include banding, RUDI (revision using distal inflow), abandonment of access and creation of a new access or changing the modality of renal replacement to either peritoneal dialysis or renal transplant. We used a novel approach, resulting in resolution of heart failure by controlling the blood flow in the fistula. With CHF and ESRD frequently coexisting in daily practice, it is important to recognize HOCF early in its course. KDIGO recommends monthly surveillance to identify risk factors. Our approach is new and advantageous in terms of preventing complications, improving morbidity and mortality and preserving the original access and does need to be tested in larger, well designed studies.

**Author: Mazen Zaarour MD**
Additional Authors:Chanudi Weerasinghe MD 1, Joseph Mosak MD 2
1 Department of Medicine, Staten Island University Hospital, Staten Island, NY
2 Division of Rheumatology, Staten Island University Hospital, Staten Island, NY
Institution: Staten Island University Hospital

**Title: MAS: A Can't Miss!**

Macrophage activation syndrome (MAS) is a hyper-inflammatory condition characterized by a cytokine storm. Cytokines act on macrophages causing hemophagocytic conversion and expansion with multi-organ infiltration. Patients typically present with acute febrile illness, diffuse lymphadenopathy, hepatosplenomegaly, liver dysfunction, low ESR, and extremely high ferritin levels. MAS is a variant of Hemophagocytic Lymphohistiocyosis (HLH) that is described in patients with rheumatologic disorders such as systemic juvenile inflammatory arthritis, rheumatoid arthritis, and SLE. It is a rare, yet rapidly progressive and life-threatening condition that requires a low threshold of suspicion, with early diagnosis and prompt initiation of therapy being essential for improved survival.

We describe a 62yo white female with a history of long-standing rheumatoid arthritis who presented with shortness of breath over three days. Additional medical history was significant for hypertension. On admission, patient was tachycardic and tachypneic with wheezing in her bilateral lung fields. Non-invasive mechanical ventilation was initiated, upon which shortness of breath improved. Chest X-ray demonstrated bilateral pulmonary nodules of unclear etiology. CT chest/abdomen/pelvis confirmed the presence of pulmonary nodules as well as hilar and inguinal lymphadenopathy. The patient's condition deteriorated over the next twenty-four hours requiring mechanical ventilation and the use of vasopressors. Work-up for sepsis including bronchoscopy was negative for infection. Her subsequent hospital course was significant for further multi-organ failure as evidenced by oliguric acute kidney injury requiring CVVH, transaminitis of unclear origin, and refractory pancytopenia requiring multiple transfusions. Given the multi-system involvement, including the pancytopenia, in a patient with underlying rheumatoid arthritis, the diagnosis of MAS was strongly considered. This suspicion was confirmed by the presence of marked hyperferritinemia (36,260 ng/ml) in the setting of low ESR (10 mm/hr). Therefore, treatment with etoposide and dexamethasone was started. To further support our diagnosis, bone marrow biopsy was performed which demonstrated the presence of histiocytes with features suggestive of hemophagocytosis. Also in favor of MAS, additional lab work came back showing elevated serum levels of IL-2 receptor. Despite the early initiation of therapy the patient expired five days later.

As described in our case, MAS presents an extreme diagnostic challenge. Given its tendency to present similarly to sepsis with a rapid progression to multi organ failure, a high index of suspicion is warranted. The combination of unexplained pancytopenia and extremely high ferritin levels in patients with underlying rheumatologic disease should make the diagnosis of MAS a strong consideration and treatment should be initiated immediately, irrespective of confirmatory diagnostic testing.

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**Renaissance Westchester Hotel**
**80 West Red Oak Lane**
**West Harrison, NY 10604**

Author: **Ayelet Hilewitz, MD**

Additional Authors: Andrew Miller, MD; Robert E. Graham MD, MPH

Institution: Lenox Hill Hospital

## Title: Guidelines? Depends on who's asking: C. difficile testing

Purpose:
Clostridium difficile infection (CDI) in hospitalized patients has become a large burden on the healthcare system. Despite wide distribution and promotion of clinical practice guidelines, adherence is sub-optimal. To improve adherence to guidelines, an analysis of barriers to implementation is advocated. Our objective was to identify the rate of adherence and compliance to our institution's recommendations compared to a national guideline.
Methods:
A retrospective observational pilot study of all in-patients from 12/5/13 through 1/5/14. Patients were identified by either C. difficile antigen or PCR orders. Results for each patient were noted. The institution and the American College of Gastoenterology (ACG) guidelines were applied to each patient and percentage compliance was calculated.
Lenox Hill Department of Epidemiology guidelines (adapted from several sources):
1) only diarrheal specimens
2) clinical suspicion should drive lab testing
3) single antigen stool test and wait for result, PCR for negative antigen only; treat only with positive result; antigen testing not exceeding three samples in three days
4) testing for cure should not be performed
5) all subsequent specimens after the first requires a written order by an LIP
ACG guidelines:
1) only diarrheal specimens
2) PCR is superior to EIA antigen testing
3) Antigen testing can be used in a 2 or 3 step screening algorithm: antigen being the initial test followed by antigen-negative specimens get no further testing, antigen positive testing undergo additional testing either by PCR or EIA or by EIA followed by PCR if EIA results are discordant
4) repeat testing is discouraged
5) test for cure should not be done
Results:
Thirty patients were included in the study. Of the 30 patients observed, 7% (2/30) adhered to the institution guidelines. 30% (10/30) adhered to the ACG guidelines. The most common barrier in testing was made in repeat testing, which amounted to 60%. Of note, 1 patient who had repeat testing was positive for CDI. The second most common error was ordering PCR and antigen testing simultaneously.
Conclusions:
As the barriers largely differ within guidelines, tailored and barrier-driven implementation strategies focusing on repeat testing recommendations are needed to improve adherence in practice. The lack of unified and clear guidelines make it difficult to assess the â€œappropriate useâ€• of the tests. We believe that this study should trigger changes in the institution guidelines including clarifying the recommendations, as well as instituting an IT hard stop with repeat testing from the laboratory, with the hopes of decreasing morbidity, mortality, length of stay and ultimately cost.

Author: **Clara Kwan, MD**

Additional Authors: Yu Shia Lin MD, Peter Homel PhD, Mary Rojas PhD, Vijay Shetty MD, Edgar Lichstein MD

Institution: Maimonides Medical Center

## Title: Barriers to care in Chinese patients with Heart Disease

Purpose: Chinese Americans are one of the largest and fastest growing cultural groups in the United States. It is documented that people with limited English proficiency are less likely to make use of health services when in need. This study identified the barriers preventing the Chinese population from obtaining prompt medical care when they experienced cardiovascular complaints. Our hypothesis was that the barriers are more than just language alone. We believe that cultural practices, beliefs, together with the types of cardiovascular diseases also play a role.
Method: The data were collected from 100 Chinese patients with symptoms of cardiovascular disease presenting to the Maimonides Medical Center, a community hospital located in the borough of Brooklyn, New York between October 2013 and June 2014. A cross-sectional interview identified eligible patients who were approached for participation during their hospital stay. Data was collected through in person interviews by a bilingual physician administering a structured questionnaire. Twelve questions were included to evaluate gender, age, country of origin, time living in the United States, religious background, spoken English fluency, preference in seeking care verses self-treatment, and length of time waited at home before arriving to the emergency room.
Results: Pearson correlation and regression analysis were utilized. Cultural Barriers were encountered with (86 %) reporting at least one cultural barrier, followed by administrative barriers (77 %), personal barriers (64 %), and circumstantial barriers (50%). Having language difficulties was the most common barrier reported (82 %). Negative correlations were found between total barriers and wait time for patients with syncope ($r = -0.47$), indicating that the more barriers these patients encountered, the sooner they came to the hospital. In contrast, patients with congestive heart failure showed a significant positive correlation between total barriers encountered and wait time ($r = 0.62$, $p = 0.01$). This indicated that the more barriers they had experienced, the longer they waited.
Conclusion: Cultural incompatibility contributed a significant obstacle when presenting to the hospital. Patients who presented with more barriers may have acute fear and viewed syncope or having a transient loss of consciousness as a more detrimental health concern compared to shortness of breath. In contrast, the opposite was true for patients with congestive heart failure or shortness of breath. This may have been due to having more barriers, these patients may be less educated about their illness, and therefore, they may have preferred to try herbal medications before seeking medical help. By exploring the factors associated with the delayed decision to visit the hospital we can develop strategies to overcome these barriers and can deliver better medicine for the Chinese population living in Brooklyn.

Author: **Jarred Marshak, MD**

Additional Authors: Ritu Saha, MD; Karl Ziermann, DO; Jennifer Slane, DO; Rose Calixte, PhD; Peter F. Malet, MD

Institution: Winthrop University Hospital

## Title: Birth Cohort Screening Rates for Hepatitis C in an Internal Medicine Clinic: Before and After an Educational Intervention

Background:
Hepatitis C Virus (HCV) affects an estimated 3.4-4.9 million people in the United States. Persons born during 1945â€“1965 account for 75% of chronic HCV infections in the United States. Approximately 50-75% of those with chronic HCV infection remain unaware of their condition. In August 2012, the Center for Disease Control (CDC) advised one-time testing for HCV in adults born from 1945-1965 without prior ascertainment of HCV risk.

**Aim Statement:**
This is a quality improvement project with the aim of raising awareness of birth cohort screening through education of resident and attending physicians. The objective was to increase the screening rate for HCV in an outpatient Internal Medicine practice.

**Methods:**
We first determined baseline birth cohort screening rates in a large Internal Medicine practice. This was accomplished by retrospectively measuring the number of HCV antibody assays ordered for screening purposes only during a 5 month period from January to May 2013. Eligible patients were those new to the practice and born between 1945 and 1965. Patients who had a prior diagnosis of HCV were excluded. We subsequently devoted one month in June, 2013 to educate the internal medicine housestaff and attendings of the clinic on the updated CDC screening recommendations. This was done through a series of lectures and handouts. We then prospectively measured the number of HCV antibody assays ordered during a 5 month period from July to November 2013. The two periods were compared to determine if education had any effect on screening rates.

**Results:**
In the pre-intervention period, we examined 139 patient records and 5 were excluded due to prior history of HCV. In the remaining 134 patients, 3 HCV antibody assays were ordered for a screening rate of 2.2%. In the post-intervention period, 106 patient records were examined and 7 were excluded due to prior history of HCV. In the remaining 99 patients, 9 HCV antibody assays were ordered, a screening rate of 9.1% which was significantly increased compared to the pre-intervention screening rate (P-value= 0.0193).

**Conclusion/Discussion:**
We found a 4-fold increase in screening rates for HCV after our educational intervention. However, further intervention may be necessary as the majority of eligible patients were still not screened for HCV. Screening is important in detecting patients with HCV who are unaware of their condition so they may be referred for further evaluation and treatment. Approximately 75%â€“85% of persons with HCV are chronically infected conferring a higher risk for liver cirrhosis and hepatocellular carcinoma (HCC). Benefits associated with diagnosis and treatment have been linked with significant reductions in HCC and all-cause mortality.

Author: **Minhaj Musa, MD**

Additional Authors: Saqif Hasan, MD (Associate Member);
Marufa Khatun, MD (Associate Member);
Rajat Mukherji, MD (Fellow Member);
Roya Mukhterzad, DO ;
Lim Josephine, MD

Institution: KINGSBROOK JEWISH MEDICAL CENTER

## Title: QUALITY IMPROVEMENT PROJECT TO REDUCE URINARY CATHETERIZATION

**Goal of Project:**
Urinary tract infection (UTI) is one of the leading causes of hospital acquired infection and leads to increased morbidity and health care costs. We postulate that, under many circumstances, catheterizations are done without any clear indication. Patients catheterized in the Emergency Room (ER) sometimes remain catheterized and this leads to Healthcare associated UTI. Our goal was to reduce the number of unnecessary urinary catheterizations in the ER.

**Methods:**
We used Centers for Medicare & Medicaid Services (CMS) guidelines to define the criteria for urinary tract catheterization. Our approach from the start was a multipronged campaign, directed towards staff education. As a first step, we discussed the proper technique of catheterization with the ER staff. Next, we began an educational in-service program involving all ER nurses and Physicians. Posters regarding the proper indication for catheterization were placed in different areas of the ER. Secondly, the electronic audit system in the ER was updated with the help of the Information Technology (IT) department, to require every physician to provide the indication for catheter placement whenever the catheterization order was placed.
Data were collected from all patients who visited Kingsbrook Jewish Medical Center ER and were given a urinary catheter. We tabulated our findings according to the number of urinary catheters inserted every month as well as the indications for catheterization. The tabulated results for each month were then compared to see if educating the ER staff including nurses and physicians had an impact on the number of urinary catheters inserted in the ER.

**Results:**
We found that, prior to our education project in ER the total number of urinary catheters inserted in March, 2014 was 52 (1.57% of the ER visit ) . After our intervention described above, the total number of catheters inserted in April, 2014 were 36 (1.1%) and with a further drop to 26 (0.7 %) in May, 2014. The data indicate that a formal educational effort and adjustment of electronic ordering system can lower the catheterization rate in patients admitted to hospital.

**Conclusion:**
Catheterization rates in the hospital can be reduced by an educational campaign in the ER as well as by adjusting the electronic ordering system. Making an adjustment to electronic order writing, such as, by mandating the placement of an indication for catheterization, can also results in a dramatic reduction in the rate of catheter placement.

Author: **Daniel Rodriguez, MD**

Additional Authors: Geurys Rojas-Marte MD, Jordana Cheta MD, Karolina Ubogaya NP, Prabhsimranjot Singh MBBS,

Institution: Maimonides Medical Center Hospital

**Title: CLINICAL SIGNIFICANCE OF A VALIDATED SCORING SYSTEM FOR PREDICTING 30-DAY READMISSIONS IN ELDERLY PATIENTS WITH DECOMPENSATED HEART FAILURE**

Purpose: Approximately one million patients are hospitalized each year for Heart Failure (HF) in the United States; almost 25% of them are readmitted within 30 days. The Centers for Medicare & Medicaid Services (CMS) have started to financially penalize hospitals with excessive risk-standardized 30-day all-cause readmission rates in patients with HF. Efforts are needed to reduce the number of readmissions in individuals with HF. The Yale Readmission Score (YRS) is a scoring system developed and validated using retrospective data from Medicare patients for predicting 30-day readmissions in patients with HF. We conducted a study to assess the usefulness of the YRS when calculated prospectively. Additionally, we sought to determine whether the difference between the score calculated at admission and discharge is predictive of readmission.
Methods: During a nine-month period, patients 65 years or older admitted with a diagnosis of decompensated HF were prospectively enrolled in the study; informed consent was obtained from all patients. The YRS was calculated at admission and before discharge using an online calculator (http://www.readmissionscore.org/heart_failure.php). The difference between the two scores (Delta score) was computed. Patients were contacted 30 days after discharge to determine whether they had been re-admitted. An analysis was conducted to assess for independent variables that might be used to determine which patients might be at risk of readmission.
Results: 201 patients were included in the final analysis. The mean age was 81.8 years (± 8 years SD); 56.6 percent of the patients were female and 82 percent were Caucasian. The mean length of stay was 7.9 days (± 5.9 days SD). The unadjusted readmission rate was 26 percent. The YRS calculated at admission and discharge were both predictive of 30-day readmission, however, when calculated at discharge, the readmission score was more predictive (P= 0.01 and 0.001, respectively). The difference between the readmission score at admission and discharge (Delta score) was not predictive of readmission (P= 0.48). Other variables found to be associated with 30-day readmissions were, low serum sodium at discharge (P= 0.03), low hemoglobin concentration and hematocrit at discharge (P= 0.01 for both) and not being discharged on a diuretic medication (P= 0.02).
Conclusion: Our study showed that an increased YRS on admission or discharge was associated with higher rate of readmission at 30 days. However, an increased score at discharge was more predictive of readmissions. The Delta score was not predictive of readmissions. Other variables were associated with higher risk of readmission, including, low serum sodium and hemoglobin concentration at discharge, and not being discharged on a diuretic medication.

Author: **Vahe Shahnazarian,**

Additional Authors: Eric Karu MD (Non-Member), Parag Mehta MD (FACP)

Institution: New York Methodist Hospital

**Title: BABY BOOMERS BEWARE: HEPATITIS C IN A BROOKLYN COMMUNITY HOSPITAL**

Purpose: To increase the quality of patient care by providing appropriate Hepatitis C testing as described by the Centers for Disease Control, United States Preventive Services Task Force and New York State Law.
**Methods**: Approval of the Institutional Review Board was obtained to begin this study. In conjunction with the Information Technology Services Department, two separate workflows were designed: one for all patients admitted to any service in the hospital, and the other for patients visiting our primary care ambulatory clinics. If the patient is a â€œBaby Boomerâ€• (born between 1945-1965), their Hepatitis C testing status is checked. After that, there are constant reminders to order the testing for qualified patients, or to never offer testing again if the patient refused or has been tested in the past. This was implemented on Tuesday, April 15, 2014.
Results: A total of 9349 patients were included in the study in a seven-month period. In December 2013, one month prior to the enactment of the Hepatitis C testing law in NY State, 47.2% of our patients were tested. In the month after the law was implemented, we still had a 46.7% rate for patient testing. However, in the month after our testing protocol was implemented, that percentage went up to 56.1% of patients either being tested or being offered testing. In subsequent months, the percentages were 54.4%, 56.8%, 58.9%, and 55.3%. When comparing the December group with the January group, there was no statistically significant difference. When comparing the December group to the intervention groups, an increase was noted with a strong statistical significance. The incidence of new monthly diagnoses were also noted to be the following: 1.6%, 1.7%, 2.8%, 3.9%, 3.7%, 3.5%, and 4.5%, respectively. When comparing the December group with the January and May groups, no statistically significant change was noted. However, when comparing the December group to the groups after May, there was a statistically significant increase noted in the incidence.
**Conclusion:** Since the implementation of our protocol, we have seen a statistically significant increase in the number of patients being offered Hepatitis C testing, as well as the incidence of positive tests. The protocol has already undergone a number of Plan-Do-Study-Act (PDSA) cycles, and we anticipate more to come. With these cycles, we have made numerous adjustments, which are reflected positively in our data. We are also in the midst of ensuring proper notification and follow-up, as well as testing a new change to the protocol that should greatly increase our testing-offered percentages. Numerous advancements remain to be made, which will all be guided by PDSA cycles that we have not yet foreseen.

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# New York Chapter ACP

# Annual Scientific Meeting

# Resident/Fellow Research

**Renaissance Westchester Hotel**
**80 West Red Oak Lane**
**West Harrison, NY 10604**

Author: **Andres Carmona-Rubio, MD**

Additional Authors: Stefan Puchner, MD; Ashley M Lee, BS; Ting Liu, MD; Godtfred Holmvang, MD; Brian Ghoshhajra, MD, MBA; Udo Hoffmann, MD, MPH; Suhny Abbara, MD and Umesh C Sharma, MD, PhD

Institution: University at Buffalo

**Title: Utility of Cardiac Magnetic Resonance Imaging and Follow-Up of Defibrillator Events to Identify the Etiology and Natural History of Sudden Cardiac Arrest**

Background: In patients with sudden cardiac arrest (SCA) cardiac MRI (CMR) can be useful to evaluate ischemic, inflammatory, infiltrative and degenerative processes. Correlation of initial CMR findings with future events recorded by automatic implantable cardiac defibrillator (AICD) can characterize the natural history of these life-threatening cardiac conditions.
Methods: We examined the CMR studies of 83 patients with SCA. In all patients, initial cardiac work-up was non-revealing for potential etiology, and CMR with late gadolinium enhancement (LGE) was performed. Following CMR, most of these patients underwent clinically indicated AICD placement. The interrogated AICD events were followed up for 1-4 years to monitor significant arrhythmias.
Results: Of the 83 patients resuscitated from SCA of otherwise unknown etiology, CMR identified a possible substrate in 41%. Presence of major diagnostic findings in CMR in SCA patients had 67% sensitivity for significant AICD-events during the follow-up.
Conclusions: Beyond standard diagnostic algorithm, CMR can identify potential cause of SCA in over 40% of the patients. Presence of LGE or other major diagnostic findings on CMR can uniquely identify patients with significant AICD events.

Author: **SHILPA DESHMUKH, MD**

Additional Authors: Shilpa Deshmukh MD, Neville Jadeja MD, Tasneem Zahra MD

Institution: Lincoln Medical and Mental Center

**Title: HIV DISEASE, RHEUMATOID ARTHRITIS, PNEUMONIA AND UNRUPTURED CEREBRAL ANEURYSM ARE ASSOCIATED WITH SIADH IN PATIENTS WITH INTRACEREBRAL HEMORRHAGE. FINDINGS FROM THE NATIONWIDE INPATIENT SAMPLE (NIS) 2011 DATABASE**

Intracerebral hemorrhage has the highest mortality of all stroke subtypes with a 30-day mortality ranging from 35-52%. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a known complication in these patients.
Introduction: To investigate the association of neurological and endocrinological factors with SIADH in hospitalized patients with intracerebral hemorrhage from a large nationwide registry.
Methods: All hospitalized patients included in the nationwide inpatient sample (NIS) 2011 database with a confirmed discharge diagnosis of intracerebral hemorrhage, as per the ICD-9-CM code 431 were identified. NIS is the largest all-payer inpatient database in the United States. Multivariable logistic models were used to determine the associations of HIV, Rheumatoid arthritis (RA), pneumonia and nonruptured cerebral aneurysm with SIADH in these patients.
Results: From the 8,023,590 admissions in the NIS 2011 database, 21,031 patients with intracerebral hemorrhage were identified. Patients had a mean age of 67.15 (+ 17.10) years, 49.5% (10,411) were women. We used weighted analysis to estimate the national values. 98,823 patients with intracerebral hemorrhage were thus analyzed with SIADH identified in 1383 (1.39%) patients. The model was adjusted for age, sex, race, median income, subarachnoid hemorrhage (SAH), nontraumatic extradural hemorrhage (NTEDH), subdural hemorrhage, HIV, anemia, RA, chronic heart failure (CHF), chronic lung disease, diabetes type 2, cocaine intoxication, hypertension, liver disease, obesity, chronic renal failure (CRF), acute renal failure (ARF), peptic ulcer disease, coagulation disorder, ischemic stroke, malignancy, pneumonia, hypothyroidism, hypertensive encephalopathy, nonruptured cerebral aneurysm, cerebral arteritis and brain tumor. SAH (OR 1.859), NTEDH (OR 4.427), HIV (OR 10.598), RA (OR 1.689),hypertension (OR 1.178), pneumonia (OR 1.521), nonruptured cerebral aneurysm (OR 1.791) and brain tumors (OR 2.076) were independently associated with increased risk of SIADH ($P<0.05$). We also found that low median income group, chronic lung disease, diabetes, CRF, ARF and ischemic stroke were associated with decreased risk of SIADH ($P<0.05$).
Conclusion: In this large national database we found HIV, Rheumatoid arthritis, pneumonia and nonruptured cerebral aneurysms to be independently associated with increased risk of SIADH in patients with intracerebral hemorrhage in addition to other known factors associated with SIADH. Further prospective studies are needed to understand more about these associations.

Author: **Ahmad Farooq, MD**

Additional Authors: Anaum Maqsood, Ahsan Farooq, Wajahat Mehal

Institution: University at Buffalo , Catholic Health System

**Title: Unexpected effects of amino acids and NMDA receptor in the treatment of Acute liver failure and acetaminophen hepatotoxicity.**

ABSTRACT BODY: Background: TLR4 and NLRP3 inflammasome activation are responsible for many inflammatory liver disease but little is known about their regulation. The NMDA receptor is known to be present on macrophages and its role in immune regulation has not been investigated. We used the NMDA ligand aspartic acid (AA) to test the role of NMDA activation in liver inflammation.
Aims: To test if AA can modulate TLR4 and NLRP3 inflammasome signaling and liver injury via its known NMDA receptor.
Methods: The NLRP3 inflammasome was activated by LPS and ATP in primary mouse macrophages, Kupffer cells and human peripheral monocytes in the presence and absence of AA and production of pro-Il1 beta and IL-1 beta assayed. NMDA receptor and beta-arrestin 2 dependence of AA effects was examined in the RAW 264.7 cells using siRNA knockdown. AA was supplemented in vivo in the presence or absence of beta-arrestin 2 knockdown in the LPS/d-Gal hepatitis and acetaminophen hepatotoxicity. Liver tissue was examined for injury and inflammation by histological grading and serum transaminases.
Results: AA suppresses in vitro TLR4 and NLRP3 inflammasome dependent inflammation in human peripheral monocytes, mouse peritoneal macrophages and Kupffer cells as assessed by levels of pro-Il1 beta and IL-1 beta. AA immunosuppressive effects require NMDA and beta-arrestin 2. In vivo AA supplementation decreases liver inflammation and injury in the LPS/d-GalN hepatitis (hemorrhage 1.03 /- 0.3 versus 3.89 +/- 0.2, ALT 744 +/- 406 versus 12560 +/- 5295, P < 0.05), and acetaminophen hepatotoxicity (necrosis 0.1 +/- 0.1 versus 1.4 +/- 0.1, hemorrhage 1.77 +/- 0.2 versus 2.5 +/- 0.6, liver transcript for pro-IL-1 beta and Nlrp3 caspase 1 and serum IL-1 beta release, P < 0.01). AA induced in vivo protection is dependent on NMDA and beta-arrestin 2.
Conclusions: Aspartic Acid acts through NMDA and beta-arrestin 2 to suppress TLR4 and NLRP3 mediated pro-inflammatory signaling and hepatitis. Aspartic acid has potential as a therapeutic agent in the treatment of acute liver failure.

Author: **Shipra Gandhi,**

Additional Authors: Additional Authors: Neha Gupta, Sidra Anwar, Katy Wang, Roberto Pili, Yashodhara Satchidanand

Institution: State University of New York at Buffalo

**Title: Impact of palliative care clinic referrals on pain control in genitourinary cancer patients: Retrospective analysis at Roswell Park Cancer Institute.**

Purpose:
Many cancer patients with genitourinary (GU) cancer suffer from uncontrolled pain, and may benefit from more focused palliative care. We assessed the frequency of specialist palliative care clinic (PCC) referrals in our GU Medical Oncology Clinic (GUMOC), and analyzed the impact of PCC referrals on their pain management.
Methods:
239 consecutive patients were collected from a retrospective review of GUMOC records from 12/1/2013 to 2/28/2014. This group of patients was used to assess the frequency of PCC referrals. Patients were divided into two arms- Arm A: GUMOC patients referred to PCC; Arm B: GUMOC patients not referred to PCC. To be able to detect a 15% between the two arms at 95% significance, 37 additional patients (who were already being seen at GUMOC) were collected from retrospective review of PCC records over 9/1/2013 to 2/28/2014. Total 276 patients were divided into Arm A (n=49), Arm B (n=227 patients). Arm B included 12 patients from GUMOC records and 37 patients from PCC records. Data for baseline pain score and 4-week follow up pain scores were collected. A palliative care screening tool (retrieved from Center to Advance Palliative care [CAPC] website) was used to assign a palliative care screening score (PCSS) to all study patients. Chi-square test and T-test were used respectively for categorical variables and numerical variables.
Results:
Among all types of cancer, prostate cancer was the most frequent (53%), followed by renal cancer (25%), bladder cancer (14%), testicular cancer (7%), and penile cancer (1%). Out of the 239 initially collected GUMOC patients, 5% were referred to PCC. 10% (n=24) had PCSS score of = 4, and 33% patients with PCSS = 4 were referred to PCC. Baseline symptoms, ECOG status (2-3) and cancer stage (locally advanced or stage 4) were more advanced in the Arm A vs. Arm B ($p=0.02$, $p<0.01$, $p<0.01$ respectively). On comparing the symptom score change from baseline to 4-week follow-up, significant improvement occurred in Arm A (vs. Arm B) in the pain score (Arm A vs. Arm B -2.74 vs. -0.13; $p<0.01$).
Conclusion:
GU cancer patients who are referred to PCC from medical oncology clinic have significant decrease in pain symptoms. Frequency of PCC consultation is still low in comprehensive cancer institutes, and not in congruence with the available palliative care screening tools criteria suggested by CAPC. Standardized tools should be developed to guide PCC referrals, and routine use of these tools will significantly help in pain control by seeking specialist palliative care.

Author: **Carlos Gongora, MD**

Additional Authors: Carlos A. Gongora MD1,2, Masahiko ShibuyaMD1, Kamal Ramzipoor4, Chang Lee4, Jenn McGregor BS1,Yanping Cheng MD1, Armando Tellez MD3, Jose V. Narvaez MD1, Edward A. Estrada4, Juan F. Granada MD FACC1, Greg L. Kaluza MD Phd FACC1
1Skirball Center for Cardiovascular Research, Cardiovascular Research Foundation, New York, NY; 2Mount Sinai St. Lukeâ€™s Roosevelt Hospital, New York, NY; 3Alizee Pathology, Thurmont, MD; 4Amaranth Medical, Inc; Mountain View, CA.

Institution: Mount Sinai St. Luke's Roosevelt Hospital

**Title: A LONG TERM LUMEN REMODELING ANALYSIS OF A NOVEL NON-DRUG ELUTING BIOABSORBABLE STENT IN PORCINE CORONARY ARTERIES**

Background: Degradation of bioabsorbable stents (BAS) has been shown to allow restoration of the treated segmentâ€™s plasticity and reactivity to those resembling a non-stented artery, a desirable feature unattainable with metallic stents (BMS).
Objective: We aimed to evaluate by optical coherence tomography (OCT) the long term dynamic changes in arterial geometry in response to a novel non-drug eluting BAS as compared to BMS.
Methods: Thirty-five coronary arteries of 13 swine received BAS (Amaranth Medical, Mountain View, CA n=22) or BMS (Liberte® Boston Scientific, Natick, MA n=13). Optical coherence tomography (OCT) was done at day 0, day 28 (BAS, n=22; BMS, n=13), 1 year and 2 years (BAS, n=6; BMS, n=4).
Results: The lumen areas of reference segments increased over time, but less for the BMS-caged segments (Day 0=6.1±1.8mm2, 2 Years=10.6±2.9mm2) than for the BAS-treated arteries (Day 0=6.7±1.9mm2, 2 Years=12.1±0.4mm2). BMS showed lumen area loss at 28 days as expected due to neointimal formation, and then minimal lumen area variance over time up to 2 years (Day 0=6.79±1.7mm2, 2 Years=6.78±2.25mm2). In contrast, in BAS the early lumen area loss present at 28 days inverted into lumen gain that corresponded with the scaffold mechanics paralleling the artery growth and allowing the treated segment to remodel (Day 0=7.31±0.9mm2, 2 Years=9.71±1.58mm2, Figure).
Conclusion: Restoration of the treated segmentâ€™s ability to grow and remodel, with late lumen area gain as a consequence, appear to be a reproducible and inherent pattern of non-drug eluting bioabsorbable stent behavior in the porcine coronary model.

Author: **Matthew Gorgone, DO**

Additional Authors: Matthew Gorgone, DO
Michael Apostolakos, MD
Michelle Malnoske, MD

Institution: University of Rochester

**Title: THE NATIONAL EARLY WARNING SCORE (NEWS) IN ITS PREDICTION OF DETERIORATING FLOOR PATIENTS**

Purpose
To evaluate the ability of the National Early Warning Score(NEWS) to predict outcomes in deteriorating floor patients.
Methods
A retrospective chart review of 60 rapid responses was performed with specific attention paid to the vital signs documented just prior to the triggering of the rapid response. The clinical course following the rapid response was noted, with documentation of whether an ICU or ICU step-down transfer occurred, as well as if the patients were intubated, coded, or died during current hospitalization. Each patientsâ€™ NEWS was calculated based on the vital signs, including systolic blood pressure, heart rate, respiratory rate, temperature, oxygen saturation and supplemental oxygen usage, as well as level of consciousness, with more abnormal findings being given higher scores. A score of 3 for any one category is the maximum that could be assigned, with the exception of supplemental oxygen usage, which presence alone, if greater than a patientâ€™s baseline, is assigned a value of 2. Patients were then stratified into 3 groups: Low (0-4), moderate (5-6, or any single physiologic parameter measuring 3) and high (7+) risk. Results were obtained from a previous chart review of the same subset of patients with risk stratification based on the Modified Early Warning Score(MEWS) for comparison.
Results
There were zero patients with a â€˜lowâ€™ score that ended up being intubated, coded, or dying, whereas both moderate and high score appeared to be associated with those outcomes. Of patients with a high NEWS stratification, over 50% transferred to a higher level of care.

Conclusion
Moderate to high NEWS stratification was associated with increased transfer to a higher level of care, as well as increased risk of death during current hospitalization. Due to its simplicity, it would be feasible to implement a change to the electronic medical record so that the score is reflexively calculated and high scores trigger evaluation by the rapid response team in hopes of identifying these deteriorating patients earlier in the course of their decompensation. Before this is implemented, more data will need to be reviewed in order to calculate a statistical significance as opposed to a trend.

Author: **Sarah Guigui, MD**

Additional Authors: B Condon, RI Cohen

Institution: North Shore University Hospital at Forest Hills

**Title: The use of Ivacaftor in cystic fibrosis mutations with residual functioning protein**

Objective: Ivacaftor, a cystic fibrosis (CF) potentiator, is approved in patients with Class III gating-type mutations and demonstrated significant improvements. It may also be effective in CF mutations with residual functioning protein (Class IV and V mutations). A pre-specified subgroup analysis of patients with the class IV mutation R117H and more advanced lung disease revealed a mean absolute treatment difference in FEV1 of 5.0 %. We report the use of ivacaftor in 4 CF adults with various Class IV mutations.

Methods: Ivacaftor was prescribed to 4 patients whose clinical status was deteriorating over the previous year. We obtained pre and post administration FEV1, weight, sweat chloride (in 3 out of 4), and CFQ-R.

Results: Patient 1 is a 26 year-old woman. CF genotype of 2 class IV mutations (R347P and L1065P). She had four acute pulmonary exacerbations, three of which required IV antibiotics. Her FEV1 was maintained between 80 to 85% but dropped to 75%. Six months following ivacaftor, FEV1 improved to 81% and she gained 10 lbs. Sweat chloride decreased from 95.9 to 85.9 moles/L and CFQ-R respiratory increased from 55 to 100. She reported almost complete disappearance of cough and remained free of exacerbations. When ivacaftor was stopped due to lack of coverage, there was marked worsening in all parameters within one month. FEV1 dropped to 68%, weight dropped by 3 lbs, sweat chloride increased to 93.6 moles/L and CFQ-R respiratory dropped to 44.6.

Patient 2 is a 61 year-old male with W1282X mutation and the class IV mutation D1152H. The year preceding ivacaftor, he had three acute exacerbations requiring IV antibiotics. FEV1 dropped from 95% to 67%. Six months into invocator, FEV1improved to 87% and he gained 17 lbs. Sweat chloride decreased from 29.8 to 22 moles/L and CFQ-R respiratory improved from 55 to 100 points. He reported disappearance of cough and had one pulmonary exacerbation, treated with oral fluoroquinolone.

Patient 3 is a 35 year-old male with the class IV D579G mutation and S912X. Following ivacaftor, FEV1 improved from 27 to 31%. He gained 7 lbs. His sweat chloride decreased from 91.2 to 74.3 moles/L and CFQ-R doubled.

Patient 4 is a 72 year-old male with CF genotype G542X and the class IV mutation D1152H. Following ivacaftor, FEV1 improved from 39 to 51%. He gained 7 lbs and CFQ-R doubled. He did not undergo sweat chloride testing. He reported disappearance of cough and wheezing.

Conclusions: We noted improvement in all patients and in all parameters at 6 months compared to baseline. One patient was retested at one month after stopping ivacaftor with worsening parameters. These data provide evidence for the beneficial effects of CFTR potentiators in CF mutations producing residual functioning protein.

Author: **Sabeeda Kadavath, M.D.**

Additional Authors: S. Chittalae1, O. Nidal Shuaib1, K. Soon Goh2,M. Tosic,Matt Briggs1, J. Giles3, P. Efthimiou1,4
1New York Methodist Hospital, 2Rheumatology fellow,Temple University 3Columbia University, 4Weill Cornell Medical College, NY, United States

Institution: LINCOLN MEDICAL AND MENTAL HEALTH CENTER

**Title: 14-3-3 ETA PROTEIN : AN EMERGING BIOMARKER IN THE DIAGNOSIS OF RHEUMATOID ARTHRITIS**

Background:The diagnostic utility of Serum 14-3-3 eta has been extensively studied and in established RA an association has been established between serum levels of this marker and the degree of joint damage. The ? isoform is expressed in much higher concentration than the ? isoform or MMP 1 and 3 levels in the serum and synovial fluid of patients with RA compared to the normal population. Rheumatoid factor (RF) is known to be sensitive and antiCCP highly specific for RA, however the number of patients remaining seronegative is substantial.There is a growing need for a biomarker to prevent underdiagnosis of this patient subset.

Objectives:The purpose of the study was to investigate if serum 14-3-3 ? enhanced the detection of RA over RF or antiCCP in RA patients. We also studied the utility of 14-3-3 eta as a diagnostic test by comparing presence of this protein in RA v/s non-RA patients.

Methods:A retrospective chart review study was conducted in RA patients at an outpatient rheumatology clinic in an inner city population at a community teaching hospital serving a large immigrant population. 132 RA patients were identified who satisfied the 2010 ACR diagnostic criteria and 37 non RA patients seen in the clinic for other rheumatologic conditions were chosen as the control group. Serum 14-3-3? protein was measured by ELISA. The positive threshold range using Quest Diagnostic for RF was 15 IU/ml, antiCCP was 20 Units and for 14-3-3 eta was 0.2 ng/mL. The chi-square test was used to analyze the frequency of RF and antiCCP positivity in both RA and non RA patients while kappa was calculated to compare the RA and non RA patients.

Results:Of the 132 RA patients, 75.8 % were females and mean age was 58 (range 28- 90) years. The population was predominantly Hispanic (75%). In the non-RA group, 9% had psoriatic arthritis and 14% lupus, 73% were females,76% Hispanic and the mean age was 54 (range 19-93) years. In the RA population, 61 were eta positive and 71 were eta negative.8.2% of the RF- and 6.56% of the antiCCP- patients were eta positive .63.4% of the RF- and 64.8% of the antiCCP- patients were eta negative.In the non RA subset, 92.6% of the RF- and 88.9% of the antiCCP- patients were negative for eta.When comparing RA vs. non-RA, the kappa calculated was 0.016 (95% confidence interval: -0.047 to 0.078), the strength of agreement is considered to be poor. Chi â€“ square computed in the RA patients was 1.86E-011 for RF and 9.34E-013 for antiCCP.

Conclusion:Measurement of 14-3-3? complements RF and antiCCP antibody tests in RA and may improve diagnostic sensitivity. Used in combination with other serological markers,14-3-3 eta can increase identification of patients with RA.

Author: **Huijuan Liao,**

Additional Authors: Daniel Lorber, Ellen Cohen

Institution: New York Hospital Queens

## Title: The Etiology and Risk Factors Analysis in Hypercalcemic Crisis

Hypercalcemic crisis is a rare but life-threatening condition involving the decompensation of hypercalcemia (usually when serum calcium > 13-15 mg/dL) with significant disturbance to cardiac, renal, gastrointestinal and neurological function. Although major textbooks have thorough and detailed reviews of hypercalcemia, there are no articles elaborating specifically the etiology and pathophysiology of hypercalcemic crisis. The goal of this study was to identify the etiologies and risk factors of hypercalcemic crisis. We performed a retrospective review from 01/2012 to 05/2014 of patients with hypercalcemia at our tertiary care center and analyzed their characteristics. Sixty-two patients with severe hypercalcemia (adjusted serum calcium level by albumin = 13.5 mg/dL) were identified from 262 of hypercalcemia. Demographic and clinical characteristics, such as age, gender, race, etiologies (e.g. primary hyperparathyroidism (pHPT), malignancy, or other causes), serum calcium level, clinical manifestations including gastroenterology, renal, cardiovascular, altered mental status, EKG changes, precipitating factors (dehydration, acute kidney injury (AKI), infection) were evaluated. Our study revealed that there were no differences in the etiologies between hypercalcemic crisis (pHPT/malignancy/others: 15%/60%/25%) and severe hypercalcemia without crisis (pHPT/malignancy/others: 7.1%/64.3%/28.6%, P = 0.617). Compared with severe hypercalcemia without crisis, the serum calcium level was significantly higher in hypercalcemic crisis (16.9±1.8 mg/dL vs 14.8±1.1 mg/dL, P < 0.001). However, no differences in serum calcium level were observed among the subgroups of different etiologies in hypercalcemic crisis (P = 0.662) or severe hypercalcemia without crisis (P = 0.423). The logistic regression analysis showed that serum calcium level and age were independent risk factors for hypercalcemic crisis. Specifically in our risk-prediction model, 1 mg/dL increase serum calcium concentration increases 2.7 times the odds of hypercalcemia crisis; one year increase in age increases the odds of hypercalcemic crisis by 61%. The multivariate linear regression analysis showed that significant predictors of serum calcium level in hypercalcemic crisis were age and AKI. To our knowledge, this is the first and most comprehensive study to investigate the etiology and risk factors of hypercalcemic crisis. Our constructed risk-prediction model makes possible the rapid and easy calculation of risk for hypercalcemic crisis. The accurate assessment of risk before investigating etiology has an important place in hypercalcemic crisis screening. The implementation of our risk-prediction model is expected to improve clinical and critical care practice in hypercalcemic crisis.

Author: **Jason Ling, MD**

Additional Authors: Timothy Miller, Sandeep Mallipattu, MD

Institution: Stony Brook University Hospital

## Title: THE ROLE OF KRÜPPEL-LIKE FACTOR 15 IN GLOMERULAR KIDNEY DISEASE

Background:
In the United States, 8.3 million individuals are affected by chronic kidney disease (CKD), resulting in significant morbidity and accounting for nearly 25% of the Medicare budget. The primary etiology of CKD is a direct consequence of initial glomerular dysfunction and injury. The glomerular basement membrane is lined by podocytes, or terminally differentiated epithelial cells, whose major function is the maintenance of the renal filtration barrier. Podocyte injury is implicated in many primary glomerulopathies, including Minimal Change Disease (MCD) and Focal Segmental Glomerulosclerosis (FSGS). In many of these diseased conditions, the podocyte loses specific markers of differentiation, characteristic morphologic features, and the functional capacity to maintain the glomerular filtration barrier. Glucocorticoids (GCs) are the first line of immunosuppressive therapy in the treatment of many primary glomerular diseases, but their mechanism of action remains unclear. In a recently published study, we characterized the role of Krüppel-Like Factor 15 (KLF15), a kidney-enriched zinc-finger transcription factor, in podocyte injury. Our in vitro (human podocyte cell culture) and in vivo (mouse model) studies revealed that KLF15 is required for recovery from podocyte injury, that a loss of KLF15 increases the susceptibility to kidney injury, and that GCs induce KLF15 expression.
Objective:
To determine whether the expression of KLF15 is a prerequisite for glucocorticoid responsiveness in glomerular injury.
Methods:
We reviewed archival renal biopsies performed between 2002 and 2012 at Stony Brook University and quantified the level of KLF15 expression (by immunofluorescence) in control subjects (healthy donors) and patients with biopsy proven primary glomerulopathy. Baseline KLF15 expression at the time of the biopsy were correlated with the specific glomerular disease, response to GCs, and renal function.
Results:
We identified donor nephrectomies (control) (n=19), patients with GC-responsive (GC-R) (n=30), and GC-nonresponsive (GC-NR) primary glomerulopathy (n=10) biopsy specimens and observed that the podocyte-specific expression of KLF15 (using WT1- a known podocyte localization marker) was significantly reduced in patients with GC-nonresponsive glomerulopathy as compared to GC-responders and healthy subjects.
Conclusion:
Glomerular disease is a major cause of chronic kidney disease and targeted therapy is unfortunately very limited. Alternative treatments are not even considered unless patients fail GC therapy. Through our database of renal biopsy specimens, we identified that KLF15 is a prerequisite for the salutary effects of GC in primary glomerulopathies. This identified role of KLF15 is promising as it helps target treatments in patients with primary glomerulopathy and brings new insight into podocyte pathophysiology.

Author: **Mansi Nabar, MD, MBI**

Additional Authors: Mansi Nabar, MD, Norbert Alex Seidenschwartz MD, Rishikesh Morey, MD, Prasanta Basak MD, FACP, Stephen Jesmajian MD, FACP

Institution: Montefiore New Rochelle Hospital and Albert Einstein College of Medicine, New Rochelle, NY

## Title: PROTON PUMP INHIBITOR INDUCED HYPOMAGNESEMIA

Background
Hypomagnesemia occurs in up to 12 percent of hospitalized patients. Since 2006, various case reports have been published citing instances of proton pump inhibitor (PPI) induced hypomagnesemia. Patients can present with profound muscle weakness, twitching, and cardiac dysrhythmias that may prompt the diagnosis of hypomagnesemia. Studies suggest that magnesium levels should be measured in patients receiving PPIs, particularly in those with malabsorption syndromes, kidney diseases, or on medications inducing renal loss of magnesium.
Methods
We conducted a retrospective chart review for hospitalized patients who were on PPIs prior to admission. The magnesium levels on these patients were noted. 300 charts were reviewed and 55 patients included for the final analysis.
Results
42 percent patients using PPI were noted to hypomagnesemic. Females were twice more likely to develop hypomagnesemia compared to males. Omeprazole was the most commonly used PPI and had the highest association with hypomagnesemia compared to other PPIs. Hypomagnesemia was associated with hypokalemia and hypocalcemia in all cases. 34% of hypomagnesemic patients were also on diuretics in addition to PPIs. Hypomagnesemia was more common in patients using diuretics and PPI than those using PPI alone. Admission notes documented that 62% patients did not have any identifiable reason to be on a PPI.
Discussion
PPI use is associated with hypomagnesemia. The bulk of magnesium is passively transported and absorbed in the small bowel. Active transport mechanisms in the small bowel are now recognized, including transient receptor potential melastatin TRPM6 and TRPM7. Patients who have a homozygous mutation of these pathways actually have significant isolated hypomagnesemia without hypocalcemia. It is not known if PPIs affect active or passive transport of magnesium, or whether PPI use may trigger hypomagnesemia in patients with TRPM deficiency. Prior studies indicate that in hospitalized patients, about 25 percent did not respond to magnesium repletion until the PPI was stopped. These patients then reabsorbed normally and their magnesium levels normalized. Over the counter (OTC) PPIs are marketed for the treatment of frequent heartburn. OTC PPIs are labeled for 14 days of use, and this treatment course may be repeated every 4 months up to 3 times per year. PPI induced hypomagnesemia is important especially in patients using diuretics or having coronary artery disease. The concomitant use of diuretics in our patients could be a possible confounder. Further consideration is needed for checking magnesium level before initiating and during the course of PPI therapy. Also, PPIs should be discontinued once the indication for its use ceases to exist.

Author: **Armaghan Soomro, MBBS**

Additional Authors: Sainath Gaddam, Vratika Agarwal, Bhavi Pandya, James Lafferty

Institution: Staten island University Hospital

## Title: MitraClip Acute Outcomes are Similar in Functional vs. Degenerative Mitral Regurgitation: A Meta-analysis of Observational Studies

Introduction:
Percutaneous mitral valve repair using MitraClip (MC) is recommended by 2014 AHA/ACC valvular heart disease guidelines, only for inoperable symptomatic chronic degenerative MR (DMR) patients on optimal medical therapy (Class IIb). However outside USA, MC is extensively utilized for severe functional MR (FMR), with some recent registries suggesting increased success with FMR when compared to DMR. Randomized control trials to evaluate MC success in functional MR are ongoing.
Methods
PubMed, EMBASE, Google scholar database and international meeting abstracts were searched for all observational MC studies. Studies which did not specify type of MR or where post-procedural results were not delineated between the types of MR were excluded from the analysis. We defined acute procedural success (APS) in our analysis as decrease in MR severity to = 2 at the time of discharge or within 30 days post procedure and MACE as composite 30 day myocardial infarction, stroke, and all-cause mortality.
Results
We included 8 observational studies, with total of 1452 patients, with 806 patients in FMR vs. 552 patients in DMR. Meta-analysis was performed with intention to treat principle. Pooled analysis using random effects model (Mantel- Haenszel statistics) was performed for acute procedural success and results were similar for both FMR and DMR (OR=0.93, p=0.82). Short term MACE events, including death were similar in both groups (OR=0.57, p=0.15). Cumulative procedural success rates with MC were 89% for both FMR and DMR.
Conclusion:
To date, this is the first meta-analysis suggesting MitraClip therapy to be of equal efficacy and safety at short term follow up for both functional and degenerative MR.

Author: **Michael Tanoue, MD**

Additional Authors: Dmitriy N. Feldman, MD, Konstantinos Charitakis, MD, Robert M. Minutello, MD, S. Chiu Wong, MD, David C. Yang, MD, Min Kyeong Lee, DMD, Harsimran S. Singh, MD, MSc, Shirley Xu, Geoffrey Bergman, MD, Luke K. Kim, MD, Rajesh V. Swaminathan, MD

Institution: New York Presbyterian Hospital - Weill Cornell

**Title: National Trends in Real-World Utilization of Percutaneous Ventricular Assist Devices and In-Hospital Outcomes**

Abstract:
Background: Percutaneous ventricular assist devices (PVADs) are increasingly being utilized over the traditional intra-aortic balloon pump (IABP) in various settings including acute myocardial infarction (MI), cardiogenic shock (CS), and high-risk non-emergent (HRNE) percutaneous coronary intervention (PCI) with limited clinical data. We assessed national trends in real-world utilization of PVAD vs. IABP and associated in-hospital outcomes.
Methods and Results: We analyzed all patients undergoing PVAD (n=4,924) vs. IABP (n=215,283) placement from 2008-2011 in the Nationwide Inpatient Sample database. Temporal trends as well as in-hospital outcomes (death, stroke, bleeding, vascular complications, and acute renal failure (ARF)) were examined for all indications. Multivariate propensity-score adjusted analysis was performed to adjust for selection bias. PVAD utilization increased 4-fold (from 470 to 1,949 devices), whereas IABP use decreased by 11% (from 57,875 to 51,415). PVAD patients were more likely to be older, female, have chronic renal failure, and undergo PCI. IABP patients were more likely to present with CS and AMI. After adjustment, PVAD utilization was associated with increased mortality (OR 1.25, 95%CI 1.05-1.48), bleeding (OR 1.32, 95%CI 1.04-1.66), vascular complications (OR 1.94, 95%CI 1.30-2.90), and ARF (OR 1.39, 95%CI 1.18-1.63). However, in the HRNE PCI group (PVAD, n=1,632; IABP, n=6,301), the adjusted risk of death was significantly lower with PVAD vs. IABP (OR 0.41, 95%CI 0.21-0.82) without differences in other in-hospital complications. Conclusions: PVAD utilization in the United States is increasing without a clear clinical benefit over less costly IABP devices. In patients undergoing HRNE PCI, however, PVAD use is associated with improved survival.

Author: **Yefei Zhu, MD, PhD**

Additional Authors: Ilya Yakhnenko, Zhigang Zhou, Praveena Satti, Kaushal Sondarwa, Michelle Dahdouh

Institution: St. Barnabas Hospital

**Title: Prevalence of High Vancomycin Minimal Inhibitory Concentration (MIC) Isolate Infections among Patients with MRSA and MSSA bacteremia**

Purpose: With the increased prevalence of methicillin-resistant Staphylococcus aureus continues to increase worldwide, there is a concern about an increase in vancomycin Minimal Inhibitory Concentration (MIC) for S. aureus strains. Vancomycin MIC has been shown to affect the clinical outcome of both methicillin-resistant S. aureus bacteremia and methicillin-sensitive S. aureus bacteremia. In this study, we evaluate the prevalence of high vancomycin MIC isolate infections in a community hospital in Bronx, New York, and risk factors and its effects on clinical outcomes.
Methods: All patients who were hospitalized in a single community hospital from January 2012 to December 2012 with S. aureus bacteremia was included in this study. We analyzed the prevalence of high vancomycin MIC S. aureus strains in Methicillin resistance S. aureus (MRSA) and Methicillin sensitive S. aureus (MSSA). The clinical features and outcome for these patients were recorded. High vancomycin MIC S. aureus strain is defined as MIC =2 µg/mL, whereas low vancomycin MIC strain is defined as MIC <2 µg/mL.

Results: We analyzed 70 patients with S. aureus bacteremia, there are 34 (48.6%) infected with MRSA with 36 (51.4%) with MSSA. Among the 34 MRSA isolates, 20 (58.8%) had a vancomycin MIC = 2 µg/mL and 14 (41.2%) had a vancomycin MIC < 2 µg/mL. In-hospital mortality was 20% (n = 4/20) in patients with a high vancomycin MIC and 21.4% (n = 3/14) in those with a low vancomycin MIC. Patients with MRSA bacteremia who had a history of HIV/AIDS or those who is on hemodialysis more likely to be infected with strains of high vancomycin MIC MRSA ($p < 0.05$). Among the 36 MSSA isolates, 41.6% had a vancomycin MIC = 2 µg/mL, 58.3% had a MIC < 2 µg/mL. The mortality rate is the same (33.3%) between high vancomycin MIC and low vancomycin MIC group. Different from MRSA group, there is no difference in the rate of infection with high vancomycin MIC between the patients with HIV/AIDS history or hemodialysis.
Conclusions: Patients with MRSA bacteremia with a history of HIV/AIDS and hemodialysis more likely to be infected with S. aureus strains with high vancomycin MICs. Unexpectedly, the in hospital mortality rate was the same among patients infected with these strains compared to patients infected with low MIC strains. This outcome can be explained with awareness of the high vancomycin MIC and early use of Linezolid and daptomycin on the patients.