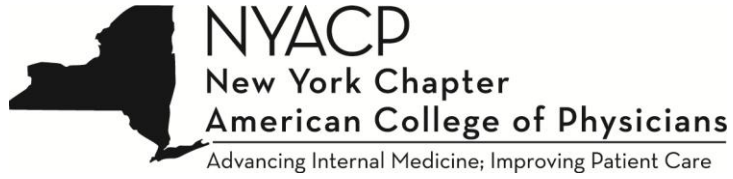


**New York Chapter ACP  
Resident and Medical Student Forum**

**Saturday, September 6, 2014**

**Regina M. McGinn Medical Education Center**

**Staten Island University  
475 Seaview Avenue  
Staten Island, NY 10305**



**New York Chapter ACP**  
**Resident and Medical Student Forum**

**Medical Student Clinical Vignette**

**Regina M. McGinn Medical Education Center**

**Staten Island University**  
**475 Seaview Avenue**  
**Staten Island, NY 10305**

## Medical Student Clinical Vignette

**Author:** Miruna Carnaru

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**Institution:** SUNY Upstate Medical University

**Title:** DELAYED DIAGNOSIS OF COMMON VARIABLE IMMUNODEFICIENCY: HOW LONG IS TOO LONG?

Introduction: Common variable immunodeficiency (CVID) can initially be confused with more common infectious, inflammatory, and autoimmune diseases, often contributing to a delay in diagnosis. We present a patient admitted for community acquired pneumonia who had first developed symptoms of CVID three decades earlier. Case: The patient is a 36-year-old male with history of idiopathic hypertrophic pachymeningitis who presented with subjective fevers, chest pain, cough, and malaise. On admission, patient was afebrile without leukocytosis. Chest radiograph showed signs of multifocal pneumonia and bronchial wall thickening. Further history from the patient and past medical records revealed he had been treated for cold-like symptoms, sinusitis, pneumonia, and meningitis at least every two years since the age of five. His first diagnosis of meningitis had been precipitated by otitis media, which left him deaf in one ear. These frequently recurring infections likely contributed to the development of idiopathic hypertrophic pachymeningitis, a rare disease characterized by chronic inflammation and hypertrophy of the dura. Once the diagnosis of this rare type of meningitis was made, subsequent work up and management focused on this disease entity. However, the underlying etiology for the recurrent infections was never elucidated. During this hospitalization for pneumonia, the history of recurrent infections and the presence of classic bronchial wall thickening prompted work up for an immunodeficiency. The patient's total IgG level (327 mg/dL) and IgG subclass levels, IgG1 (174 mg/dL), IgG2 (97 mg/dL), and IgG4 (1 mg/dL), were very low and his IgM level (76 mg/dL) was within the lower limits of normal. He was diagnosed with common variable immunodeficiency (CVID) and was discharged with follow-up for intravenous immunoglobulin infusion.

Discussion: CVID is a syndrome encompassing a diverse collection of disorders that present with recurrent bacterial infections due to impaired B-cell differentiation and thus defective immunoglobulin production. These hypogammaglobulinemias include reduced IgG levels, and reduced or low normal IgA or IgM levels. Patients present between the ages of 20 to 40 with recurrent respiratory tract infections, including sinusitis, otitis media, bronchitis, and pneumonia, as well as bronchiectasis and interstitial lung diseases. Lastly, 73% of patients with CVID acquire structural pulmonary complications, most commonly presenting as bronchiectasis and bronchial wall thickening, the latter being the final piece that triggered work up and diagnosis of CVID in our patient. Although there is an average delay of 6-8 years from symptom onset to diagnosis, this patient experienced a 31-year delay with frequent hospitalizations and extensive work-ups.

Conclusion: Unexplained recurrent infections, especially of the respiratory tract, should suggest an underlying immune-deficient state. Careful history and a broad differential is key to diagnosis; confounding disease processes should not serve as distracters.

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**Institution:** NY Methodist Hospital

**Title:** HYPERTENSIVE EMERGENCY IN A YOUNG ADULT WITH A SIGNIFICANT FAMILY HISTORY

A 28-year-old obese African American male with no significant past medical history presents with headache, non-productive cough and shortness of breath for the past 5 weeks. Patient's mother reports that her grandmother, mother and all her siblings have been diagnosed with hypertension between the ages of 20-30 years. On physical exam patient's blood pressure (BP) is 246/126 and BMI is 44.35. Fundoscopic examination reveals grade IV hypertensive retinopathy. Lab results show hemolytic anemia, hypokalemia and CR levels of 6.72. UA shows granular casts and his 24h urine shows proteinuria of 3.78g. Aldosterone/Renin ratio is 0.89. ADAMST13, Cortisol, TSH, Hba1c, lipid profile and urine metanepines is WNL. Renal Artery Doppler scan is inconclusive due to body habitus. A captopril nuclear medicine study is negative for Renal Artery Stenosis. A diagnosis of malignant hypertension with secondary hemolytic anemia and renal failure is made. The two main differential diagnoses for hypertension with hypokalemia are Renal Artery Stenosis (RAS) and Primary Aldosteronism (PA). Pheochromocytoma, hyperthyroidism and Cushing's syndrome also need to be considered. A strong family history suggests monogenic syndromes of hypertension, therefore taking a family medical history is integral. A Doppler scan is the method of choice to screen for RAS. Aldosterone/Renin ratio is the diagnostic test for PA and a ratio > 20 confirms the diagnosis. High renin levels and normal TSH, cortisol, urine metanephrine levels rule out secondary causes and favors the diagnosis of essential hypertension with malignant changes.

Initial treatment of malignant hypertension is parenteral nitroprusside or labetalol with a reduction of BP, by no more than 25% of the initial value, within the first 24 hours. Among patients who develop resistant hypertension the preferred three-drug regimen is an angiotensin-converting enzyme inhibitor, a long-acting calcium channel blocker and thiazide diuretic. Minoxidil and potassium sparing diuretics can be added if the hypertension persists despite the regimen. Apart from medications, out patient follow up with Ophthalmology and Nephrology, including life style modifications and weight loss is essential in the long-term management of hypertension.

Genes associated with monogenic syndromes of hypertension have been identified and studied. However, the genetic basis of essential hypertension is recently becoming accessible. Based on Ehret and Caufield's review of the current genome-wide association studies for essential hypertension, the magnitude of the missing heritability could suggest that there is a yet unrecognized, but major, mechanism that remains to be discovered. This is an interesting case with an atypical presentation of essential hypertension leading to multi-organ failure. Further exploration of identified genetic variants and additional loci would be beneficial in understanding BP pathophysiology and treatment.

## Medical Student Clinical Vignette

**Author:** Thomas Franzon Medical Student IV  
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### **Title: THE UNMASKING OF AUTOIMMUNE DISEASE IN PREGNANCY**

#### Introduction

Thrombocytopenia is found in 7-8% of all pregnancies. The mechanism for thrombocytopenia in pregnancy is most often secondary to an increased activation or destruction of platelets, such as in gestational thrombocytopenia, preeclampsia, and rarely, immune thrombocytopenic purpura (ITP). We present a rare case of thrombocytopenia in a post-partum female.

#### Case

A 26 year old female gravid 2 para 2 with a history of preeclampsia presented to the hospital two weeks after delivery, with progressively worsening arthralgias involving the small joints in her hands, wrists, elbows, knees, and ankles bilaterally. She reported fevers, malaise, and swelling of her joints that she treated with ibuprofen at home. She denied morning stiffness, but reported some bruising on her toes, right knee, and a few episodes of epistaxis.

Her vital signs were within normal range. A detailed physical exam revealed point tenderness of the small joints in her hands, wrists, elbows, knees, and ankles bilaterally. There was minimal bruising of her right knee and great toes bilaterally. Her lab workup revealed anemia and thrombocytopenia. A hematologic workup revealed elevated LDH, low haptoglobin, normal fibrinogen, and normal ADAMTS13 activity. Direct and indirect Coombs' tests were negative and a peripheral blood smear did not show evidence of schistocytes.

Rheumatologic work up showed elevated antinuclear antibody, anti-dsDNA, anti-smith, anti-RNP, anti-scl-70, and anti-histone levels and low C3 and C4 complement levels. The patient was diagnosed with ITP secondary to systemic lupus erythematosus (SLE). Her platelet count subsequently improved with steroids and the patient was discharged on prednisone with close rheumatological follow up.

#### Discussion

Most causes of thrombocytopenia during pregnancy are due to gestational thrombocytopenia, preeclampsia, or ITP. Although the pathophysiology of ITP is not completely understood, the destruction of platelets is related to specific IgG autoantibodies directed against platelet membrane glycoproteins such as GIIb/IIIa. The majority of ITP (80%) is primary and is not associated with a precipitating condition. Secondary ITP is associated with a precipitating condition such as autoimmune disease, viral illness, or malignancy. SLE has been attributed to 2-5% of all cases of ITP. This patient likely experienced a pregnancy-induced flare-up of SLE, causing her thrombocytopenia and arthralgias. The hormonal and biochemical changes during pregnancy are thought to exacerbate lupus, sometimes causing a patient's first presentation of lupus. SLE should be included in the differential diagnosis of ITP, especially in the pregnant and post-partum female.

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### **Title: AN UNUSUAL CASE OF MENORRHAGIA**

#### Case Description:

45 years old female, Taiwanese Buddhist nun, presented to emergency room for menorrhagia which has lasted for 3 days. She has been feeling weak and fatigue for the last 3 months, and 1 month prior to presentation, she had menorrhagia which lasted for 6 days. She had another menorrhagia which started 3 days before admission. Her complaint was associated with weakness, fatigue, lightheadedness, palpitation and lower abdominal discomfort. In the emergency room, her complete blood count with differential and smear revealed; white blood cell count 11,900/UL, hemoglobin 4.6 g/dl, platelet 26,000 /UL, MCV 72.2 FL, neutrophils 26%, monocytes 31%, metamyelocytes 1%, myelomonocytes 6%, promyelocytes 1%, and blast cells 2%. During hospital course, total 4 units of packed red blood cell and 2 units of platelet transfusion along with intravenous iron and folic acid were given to her. Bone marrow aspiration showed focal area with blasts reaching 20% of the cell, flow cytometry showed increased number of early myeloid cells and mild monocytosis, fluorescence in situ hybridization analysis was negative for acute myeloid leukemia with recurrent abnormalities, and cytogenetic analysis showed an abnormal female karyotype with t(3:12)(q26:p13).

#### Discussion:

This case is a rare type of myelodysplastic/myeloproliferative neoplasm that presented with menorrhagia, anemia and thrombocytopenia. Bone marrow aspiration and biopsy showed focal area with blasts more than 20% of the cells, but fluorescence in situ hybridization analysis was negative for acute myeloid leukemia and cytogenetic analysis showed an abnormal female karyotype with t(3:12)(q26:p13), which has been described in a setting of myeloid neoplasm and is associated with an unfavorable progression. These findings are those of chronic myelomonocytic leukemia (CMML-2) with progression to acute myelomonocytic leukemia (AMML).

## Medical Student Clinical Vignette

**Author:** Galyna Ivashchuk BS, MS  
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**Institution:** New York Methodist Hospital  
**Title:** Hemichorea Hemiballismus in Hyperglycemia

HEMICHOREA HEMIBALLISMUS IN HYPERGLYCEMIA  
Galyna Ivashchuk BS, MS, Todd Simon MD, FACP  
New York Methodist Hospital, Brooklyn, N.Y.  
INTRODUCTION

Hemichorea hemiballismus is a clinical condition that has been used to describe a spectrum of disorders including uncontrollable, random, variable amplitude jerking, flinging, or kicking motions involving the distal or proximal parts of the limbs. This syndrome is a rare but known complication of non-ketotic hyperglycemia, especially in poorly controlled diabetic patients, and has a rapid onset.

### CASE REPORT

An 88 year old Guatemalan female with medical history significant for hypertension, osteoarthritis, rheumatic fever, asthma, diabetes mellitus presents to the hospital after waking up four days prior with uncontrolled movement in her right leg. She doesn't complain of any weakness, numbness or muscular paralysis. Movement disappears when the patient is sleeping, and starts again when she is awake. The movement has gotten worse since it started, with the patient unable to sleep well or ambulate since the onset of symptoms. The movement has progressed to the right arm and right side of the neck, although in lesser severity. History is notable for an admission to hospital a month and a half ago for elevated sugar readings at home, with glucose on admission of 568 mg/dL, HbA1C 14.8% and urinary ketones of 5mg/dL. Physical examination now was remarkable for choreiform, circular-like movements of right lower extremity, with no sensory deficits. Motor strength couldn't be assessed due to uncontrolled movement. Laboratory findings demonstrate elevated urinary ketones (10mg/dL) and elevated HbA1C (9.5%). CT of brain without contrast showed no evidence of an acute event. T1 weighted MRI showed increased signal in left putamen. Patient was treated with Haldol as an inpatient, and continues to take tetrabenazine at home. Although the patient's symptoms have improved, they have not fully resolved and have left her non-ambulatory.

### DISCUSSION

Hemichorea hemiballismus (HH) is a rare manifestation of uncontrolled diabetes mellitus. Other potential causes include CVA, neoplasm, SLE, Wilson's disease, and thyrotoxicosis. Pathophysiology is thought to relate to hyperglycemic impaired cerebral autoregulation, causing hypoperfusion and depletion of GABA in neurons, leading to uncontrolled movement. CT scans typically show hyperintense signal within basal ganglia, but unlike hypertensive hemorrhage, there's no mass effect or edema. Characteristically, on T1-weighted MRI, there's hyperintensity in the basal ganglia, consistent with ischemia. Treatment involves aggressive glucose control and postsynaptic D2 receptor blockers, such as haloperidol and tetrabenazine. Recognition of this condition is important, as prompt glycemic control usually leads to resolution of symptoms. Our case is unique as the patient didn't have marked hyperglycemia at the time of admission, and her symptoms didn't fully resolve.

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**Institution:** Jacobi Medical Center

**Title:** A unique presentation of takotsubo cardiomyopathy related to illness anxiety

An 87 year-old woman presented to the ER left-sided chest pain radiating to the neck accompanied by nausea and diaphoresis not relieved by nitroglycerin.

In the ED, the patient was hypotensive to 80/40 mmHg and pulse of 61. Physical exam was significant only for tenderness and palpable abdominal mass in the left lower quadrant. There were no cardiac murmurs, rubs or gallops, JVD, or lower extremity edema. Lungs were clear to auscultation. EKG showed ST elevations in V2, ST depressions in V4-V6, and T-wave inversions in V1-6. Troponins were elevated to 0.3 ng/ml. She was given aspirin 325 mg, Plavix 600 mg, heparin 3000 units, morphine 4 mg IV and transferred for catheterization, which showed severe hypokinesis in anterolateral, apical and mid diaphragmatic segments with 90% diffuse LAD disease. An echocardiogram showed an ejection fraction of 30%, decreased from 75% one-month prior. Left ventricle dysfunction was determined to be out of proportion to CAD and the patient was diagnosed with Takotsubo cardiomyopathy. She was discharged six days later.

Although both the patient herself and her daughter denied any recent deaths in the family or acute emotional hardship, the patient was extremely anxious about a large, palpable left sided abdominal mass that had been present for at least three years. Repeated CT scans of the abdomen showed a soft-tissue density mass measuring 5.7 x 1.9 cm representing a torn muscle that exhibited no signs of growth over four years. A biopsy of the lesion three years prior was read as benign connective-fat-muscle tissue. Despite constant reassurance, the patient expressed concern that it was cancerous and persistently complained about its presence; notes from both her regular primary care physician and gastroenterologist made note of her fixation on the mass.

Discussion: Takotsubo cardiomyopathy is triggered by stress-induced catecholamine release and frequently associated with emotional trauma. The absence of any acute hardship in this case suggests patient anxiety concerning her abdominal mass contributed to cardiomyopathy. Although it has been frequently postulated that Takotsubo may result from a patient's concern about physical malady, to this author's knowledge no cases have been formally described.

## Medical Student Clinical Vignette

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**Institution:** SUNY Upstate Medical University

### **Title: UNMASKING PROSOPLEGIA**

Neurologic complications such as prosoplegia, or facial nerve palsy, can be the sole manifestation of Epstein-Barr virus (EBV) infection of the CNS in up to 5.5% of cases.

Although this association is more common than is generally appreciated, it may be difficult to discern from a number of diagnostic possibilities. We present one such case of cranial nerve VII palsy that was challenging due to the patient's existing diagnosis of HIV/AIDS.

A stroke code was initiated in the emergency department for a 74 year old male with HIV/AIDS who presented with moderately severe neurologic impairment. His symptoms included left-sided facial weakness, photophobia, occipital headaches and transient LUE weakness. He was evaluated for similar symptoms 1 week prior to hospital admission. The day before presentation, he was seen at his HIV clinic where he was found to have persisting symptoms and a few inconspicuous healing blisters on his lips. Acyclovir and gabapentin were prescribed for suspected herpes zoster.

The patient was afebrile and lacked lymphadenopathy, tonsillopharyngitis or splenomegaly. His medical history was significant for adrenal insufficiency, stage 3 chronic kidney disease, stage 1 hypertension, CMV retinitis and toxoplasmosis. His last CD4 count was 222/mm<sup>3</sup> despite adherence to his antiretroviral therapy. On initial examination, he was found to have left-sided weakness of the frontalis muscle, flattening of the nasolabial folds, drooping of the corner of the mouth and dysguesia. He was able to close his eyes completely but the left eye was easily forced open. Additionally, there were no obvious labial blisters. The physical exam suggested peripheral facial nerve palsy.

Further workup was as follows: MRI of the brain did not show acute infarct or mass. CSF analysis revealed: glucose 88mg/dL, LD 23U/L, protein 47mg/dL, WBC 36/mm<sup>3</sup>, monocytes 100% and RBC 1/mm<sup>3</sup>. This monocytic pleocytosis led to a viral encephalitis work up. DNA PCR for adenovirus, VZV, CMV, HSV-1, HSV-2, HHV-6, Enterovirus RNA PCR, fungal culture, gram stain and culture, VDRL and SPEP for oligoclonal bands were all negative. Toxoplasma IgG was positive, but IgM was negative. Lyme serology was also negative. HIV-1 RNA was undetectable and CD4 count was 179/mm<sup>3</sup>. However, the EBV DNA PCR was positive. Prior to these findings, presumptive treatment with acyclovir and gabapentin was initiated with symptomatic improvement observed during the hospital course.

Bell's Palsy is 100 times more frequent in the HIV-1 infected population. However, one must remember that Bell's Palsy is a diagnosis of exclusion. An alternative etiology can often be unmasked. Thus, severe immunocompromise should invoke a meticulous work up. Finally, isolated, unilateral and reversible cranial nerve VII palsy secondary to acute EBV infection can occur without the classic clinical presentation of heterophile positive mononucleosis.

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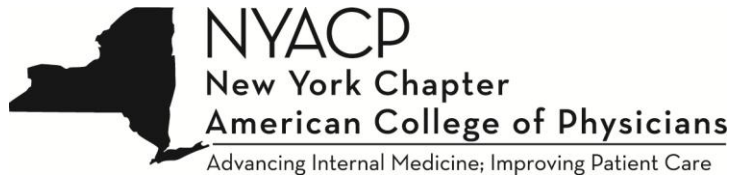
### **Title: CHILL OUT: HYPOTHERMIA IN SYSTEMIC LUPUS ERYTHEMATOSUS**

**Introduction:** Systemic Lupus Erythematosus (SLE) is an elusive autoimmune disorder with its ability to imitate other medical conditions with its differing manifestations. Since no definite cure has been established, flare-ups are commonly treated with corticosteroid therapy. However, corticosteroids are notorious for their many adverse effects. This is a case of a patient experiencing hypothermia after the administration of corticosteroids for a SLE flare.

**Case Presentation:** The patient is a 23 year-old Bangladeshi female with past medical history significant for SLE and bipolar disorder, who presented with complaints of fever associated with night sweats and chills for the past month. The patient had been diagnosed with SLE 4 years ago and bipolar disorder 1 year ago, and has had one flare up per year since diagnosis. During previous flare-ups, mild fevers were accompanied with severe myalgias, and arthralgias, however the patient denied any in this episode. Vital signs on admission revealed rectal temperature of 103F and tachycardia at 118bpm. On exam, patient was alert, awake, oriented x 3, but lethargic. There were chronic hyperpigmented areas on upper/lower limbs, but no other acute rash was evident. There were no signs of active synovitis. On labs, ANA and anti-dsDNA were positive, complement levels were low (C3-28.1, C4<1.5, CH50 <10), and ESR (46) was high, consistent with an active flare up. The patient was administered 60mg IV Solumedrol every 8hrs. She received 2 doses before developing hypothermia at 92F. She received another 3 doses and then was switched to Solumedrol 20mg PO. The patient was treated with warming blankets until core temperature was consistently between 97-98F 24 hours later. EKG performed during hypothermic episode showed no Osborn waves. Other known causes of hypothermia were excluded, including hypothyroidism and sepsis.

**Discussion:** Hypothermia is a condition in which core body temperature decreases below 95F. Etiology of hypothermia is multifaceted, occurring due to exposure to extreme cold environments, or as sequelae to severe medical conditions such as malnutrition, hypothyroidism, and sepsis.

Hypothermia is not a typical feature of SLE. We believe this to be the first reported case of hypothermia in a patient with SLE treated with corticosteroids in the United States over the past thirty years. Older case reports have noted patients with a similar presentation and concluded that hypothermia may develop as a neuropsychiatric expression of SLE, and steroid therapy may have a role in the etiology. Also, neurotransmitter dysfunction involved in neuropsychiatric SLE has been speculated to cause dysregulation of thermogenesis. However, the exact pathophysiology of steroids and neurochemistry modulating thermoregulation is still unclear.



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# Medical Student Research

**Author:** Maria Battaglia

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**Institution:** Albany Medical Center

**Title: Categorizing Neonatal Deaths: A Cost-efficiency Analysis of Resource Expenditure in U.S. and Filipino Neonatal Intensive Care Units (NICUs)**

The Neonatal Intensive Care Unit is an environment where some of the most extreme measures are taken to treat sick newborns. While this often results in newborns getting the expensive life-saving treatments that allow them to thrive, it is also a place where countless resources may be spent without success. This project examined the cost efficiencies of two culturally disparate NICUs (in Albany Medical Center (AMC, Albany, United States) and Corazon Locsin Montelibano Memorial Regional Hospital (CLMMRH, Bacolod, Philippines)), comparing patterns of resource expenditure. Both NICUs are of similar capacity and structure, but are very different in terms of available resources, funding, as well as cultural and ethical perspectives. This is a cross-cultural comparative study using retrospective chart review over a two-year period, examining medical records from 2011 and 2012 deaths. We categorized the deaths based on cause and mode of death, using cost of medical care and length of stay as primary outcome measures. In addition, information was collected on who made the important end-of-life decisions on behalf of the babies.

The mean length of stay for NICU deaths was 13 days at AMC versus 3 days at CLMMRH. The NICU mortality rates were 6% at AMC compared to 35% at CLMMRH. At AMC, 5% of infants greater than 32 weeks gestation died compared to 45% at CLMMRH. Sepsis was the main cause of death for CLMMRH and the second top cause of death for AMC, and these deaths were the most costly for both NICUs, respectively. The lack of ventilator accessibility at CLMMRH and the course of antibiotic administration are two specific cost items that we noted to be extremely important factors to concentrate on in order to make future improvements in care. Last, CLMMRH was found to have significantly less resource availability but demonstrates their initiative towards resource efficiency. Two examples include utilizing used NCPAP prongs instead of new ones as well as offering ambu-bagging as an alternative to ventilator support. In this way, CLMMRH offers an alternative to families who cannot pay for more optimal treatments. Both hospitals used the strategy of joint decision making by both health care professionals and families. We found that an intrinsic ethical dilemma exists when resources have the ability to control the degree of treatment given to neonates who are often in life or death situations. Though the two NICUs are equipped with very different proportions of resources, serving very different communities, a lot can be learned for the future not only between the two NICUs but to other units as well. The results of this study have implications for strategic improvements to neonatal care and reducing neonatal mortality in different clinical settings and with varying resource constraints.

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**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 plaque'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 symptomatology – 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 Farneback'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.



# Medical Student Research

**Author:** Sarah Lander  
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**Institution:** SUNY Upstate Medical University

**Title: THE BURDEN OF DEPRESSION AND ANXIETY ON THE QUALITY OF LIFE OF MEDICAL STUDENTS**

**Purpose:**  
We screened medical students (MS) for generalized anxiety disorder (GAD) and major depression and compared these results to the prevalence of these diseases in the general population.

**Methods:**  
An anonymous, voluntary online survey was completed by the MSs at SUNY Upstate Medical University incorporating the Patient Health Questionnaire (PHQ-2) and the generalized anxiety disorder scale (GAD-7), two widely used screening tools for depression and GAD, along with additional questions on stressors and academic performance. Data was analyzed using Microsoft Excel 2010 and SPSS v19.

**Results:**  
Our study population included 336 MS analyzed by age group, gender, and race. Age groups; 18 to 24 = 46.4%, 25 to 30 = 45.2% and >30 = 8.4%. Females were 51.2%. Caucasians were 67.6%. 16.4% and 20.3% of MS were found to have a positive screen for depression and GAD, respectively. In the US population the prevalence of depression and GAD are 6.7% and 3.1%, respectively. The rates of positive screens for major depression (PHQ-2  $\geq$  3) were higher in MSI (16.5%) and MSII (24.4%) than MSIII (12.3%) and MSIV (13%). However, medical students in the first three years had higher rates of positive screens for moderate to severe anxiety (GAD-7  $\geq$  10) than fourth year medical students {(MSI-MSIII = 22.5%) vs (MSV = 14.1%)}. 32% of MSs believe there is a significant impact of depression or anxiety on their academic performance, and a positive screen for depression was significantly associated with a positive GAD screen ( $p < 0.05$ ). While the majority of MS deal with their stress with exercise or talking to friends, 21% use substances such as alcohol or tobacco for stress relief. MS III-IV experienced most stress with surgery and least stress with psychiatry and family medicine rotations. Attendings and residents contributed to their most stressful experiences.

**Conclusion:**  
Medical Schools recruit high achieving individuals into a long, arduous and stressful training process, which may predispose these students to psychopathological disorders. Rates of positive screens for major depression and GAD in MS were significantly higher than the general U.S. population. Further studies are needed to identify personal and institutional related aspects that contribute to depression and anxiety and further explore the relationship between these stressors and academic performance.

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**Institution:** Metropolitan Hospital

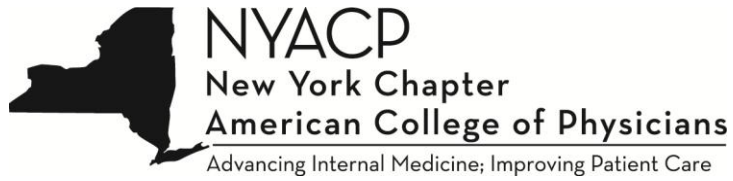
**Title: COCHLEAR IMPLANT OUTCOMES IN THE GERIATRIC POPULATION**

**PURPOSE:** This study evaluates the audiologic benefit of cochlear implantation in the elderly population.

**METHODS:** This is a retrospective chart review of patients age 65 or older that presented to an academic referral center between April 1993 to December 2013. Inclusion criteria were patients who had received at least a unilateral cochlear implant and a 3-month post implant evaluation. Preoperative and postoperative consonant-nucleus-consonant (CNC) word recognition scores at 50 dB were collected.

**RESULTS:** 118 patients were included in the study. The average age of the patients was 72 (range of 65-88 years at age of implantation). The average age of hearing loss onset was 49 (range of 0-78 years). Patients in this study experienced a significant improvement in audiologic performance as seen with word recognition scores ( $p < 0.00001$ ). The mean post implant score at 6 months was 52% and at 24 months was 58% on CNC scores at 50 dB compared to average preimplantation aided score of 5%. There was no correlation with age of hearing loss onset and performance ( $R^2 = .0062$ ). There were very few surgical complications (two wound infections) and six patients had minor postoperative complications (dizziness and hyperacusis). Two patients had the implant removed and were reimplanted due to device failure.

**CONCLUSIONS:** Our findings suggest that cochlear implantation in the geriatric population is a safe and effective way to improve speech scores. It was previously thought that this procedure would be of little aid to older candidates, due to age-related degeneration of the central and peripheral auditory system causing limited improvement. In addition, there is an increased likelihood of surgical and anesthetic complications in the elderly, as well as coexisting physical and cognitive deficits that may have an impact on recovery postoperatively. When we stratified our age groups into ages 65-74 and 75+, there was no statistical difference in CNC word scores at 3 months post implant. The same trend was also seen at 12 months post implant. This suggests that the 75+ group did not require a prolonged time to acclimate to the cochlear implant technology. Furthermore, previous studies have found that a shorter percentage of life spent with hearing loss carries a greater predictive value in determining postoperative performance than age. In our study, we did not observe such a clear trend.



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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Molham Abdulsamad M.D <b>Additional Authors:</b> HASHMI HAFIZ Chaitanya Chandrala Sridhar Chilimuri <b>Institution:</b> Bronx Lebanon Hospital Center</p> <p><b>Title: Late onset posterior reversible encephalopathy syndrome (PRES) following an uneventful pregnancy</b></p> <p><b>Introduction:</b> PRES is commonly seen in pregnant patients with eclampsia and hypertensive encephalopathy. We report an unusual case of PRES occurring in a patient with uneventful pregnancy.</p> <p><b>Case Description:</b> A 21-year old woman G1P1 presented to our hospital with multiple episodes of generalized tonic clonic seizures. She complained of headache with no focal neurological deficits. She had an uneventful pregnancy and uncomplicated cesarean section 10 days prior to her presentation. She had a history of intermittent bronchial asthma. Her physical exam was unremarkable with normal blood pressure. Her labs were unremarkable and urine analysis showed no proteinuria. CT scan of the head was normal and CSF studies to exclude meningitis were normal. Due to recurrent seizures and development altered mental status, an MRI with contrast was done which showed moderate sized patchy areas of flair hyperintensity involving the cortical bilateral frontal, parietal, occipital and temporal lobes along with the bilateral basal ganglia in a pattern most suggestive of posterior reversible encephalopathy syndrome. Despite lack of clinical evidence of eclampsia she was started on magnesium drip with significant improvement of her mental status and seizures.</p> <p><b>Discussion:</b> PRES is a radiographic diagnosis in patients with complicated pregnancies and neurological manifestations. Most commonly it presents with headache, seizures and neurological deficits in patients with eclampsia and hypertensive encephalopathy. MRI remains the modality of choice in diagnosing PRES. Our case represents a rare presentation of PRES with remarkable improvement with magnesium in the absence of eclampsia. In postpartum women with neurological manifestations, PRES should be considered despite of normal blood pressure and lack of proteinuria.</p>	<p><b>Author:</b> Munjid Al Harthy MD <b>Additional Authors:</b> Steven Bernstein, MD and Peter A. Kouides, MD</p> <p><b>Institution:</b> Rochester General Hospital</p> <p><b>Title: ATRAUMATIC WARFARIN-RELATED SPLENIC HEMATOMA</b></p> <p><b>Case:</b> An 83-year-old man with atrial fibrillation on chronic anticoagulation with warfarin, presented to the emergency department with a complaint of progressively worsening lightheadedness, fatigue and exertional dyspnea for one month, and a 10 pound weight loss over 2 months. Laboratory evaluation showed a hemoglobin (Hgb) of 8.5 g/dL, down from 13.3 g/dL 6 months prior. Platelets were 355 000/uL. The international normalized ratio (INR) was 4.3, with a previous value being 2.6 twelve days prior. Further work up revealed a lactate dehydrogenase (LDH) of 638 (RR: 120-246), ferritin of 1285 (RR: 22-322), C-reactive protein (CRP) of 236.5 (RR: 0-10), and an erythrocyte sedimentation rate (ESR) of 91 (RR: 0-23). A basic metabolic panel and electrolytes were within normal limits. CT abdomen and pelvis with contrast revealed splenomegaly with diffuse heterogeneous enhancement, measuring approximately 20 cm in length. Given the elevated inflammatory markers and splenomegaly, there was a strong suspicion for a lymphoproliferative neoplasm as a cause of his anemia, and a bone marrow biopsy was performed. The bone marrow aspirate revealed a relatively hypocellular marrow with a fat: cell ratio of 95:5, and normal M:E ratio. All hematopoietic elements were present with normal maturation. Flow cytometry revealed findings consistent with a monoclonal B cell population, with a non-specific immunophenotype. He was referred to a surgeon for evaluation for a splenectomy. A pre operative PET CT performed for further staging in the setting of possible lymphoma revealed findings consistent with a sub capsular hematoma in an otherwise normal appearing spleen.</p> <p><b>Discussion:</b> Atraumatic splenic rupture (ASR), sometimes also referred to as spontaneous splenic rupture, is an ill-defined term that is used to describe cases of splenic infarction and hematoma without a clear antecedent history of trauma, in a grossly and histopathologically normal spleen. The causes of ASR include infectious, neoplastic, autoimmune, benign hematologic, inflammatory, drug or toxin related, and iatrogenic causes. Surprisingly, only a hand full of cases of ASR in patients on anticoagulants have been described, with very few occurring in patients on warfarin. Our patient was referred to a surgeon for a splenectomy due to the elevation in inflammatory markers, LDH, heterogenous enlargement of the spleen and monoclonal B cell population, that all seemed to point towards a possible lymphoproliferative process. A pre-surgical PET CT however, proved that the splenomegaly was infact due to a subcapsular hematoma that was missed on initial CT. It appears that the elevated LDH was secondary to the hematoma, and the monoclonal B cell population was merely a "red herring" termed clonal lymphocytosis of undetermined significance. His anticoagulation was discontinued, and a follow up CT scan 3 months later demonstrated a return of the spleen size to normal, and a resolving hematoma.</p>
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## Resident / Fellow Clinical Vignette

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**Institution:** Montefiore New Rochelle

### **Title: HERPES SIMPLEX ESOPHAGITIS IN COPD EXACERBATION**

**Introduction:** Herpes simplex esophagitis (HSE) is usually seen in immunocompromised or severely ill patients, and rarely in healthy individuals. We describe a case of HSE, developing after initiation of a short course of high dose steroid therapy for acute COPD exacerbation.

**Case report:** A 70 year old female with a history of COPD came to the ER for pleuritic chest pain and worsening dyspnea for a week. She denied cough, fever, chills, weight loss or leg swelling. She was on azithromycin, fluticasone-salmeterol inhaler and prednisone 40 mg daily, since the last 3 days. She had bilateral wheezing and chest radiography revealed hyperinflated lungs consistent with COPD. She was started on bronchodilators and methylprednisone 125 mg, followed by 60 mg every 6 hours. On the third day of admission, she developed progressive odynophagia and dysphagia limiting her ability to swallow. Subsequent esophagogastroduodenoscopy revealed shallow diffuse ulcerations within the body of the esophagus. Biopsies were consistent with HSE and the diagnosis of HSV type 1 was confirmed with positive viral cultures. HIV tested negative. Treatment with esomeprazole and acyclovir proved effective with gradual improvement of her symptoms.

**Discussion:** Herpes esophagitis is rare in immunocompetent patients, occurring most commonly in solid organ and bone marrow transplant recipients, patients on chemotherapy, and those with HIV infection. HSV type 1 represents the majority of infections, with HSV 2 being occasionally reported. Patients usually present with odynophagia and/or dysphagia, retrosternal pain and heartburn. The diagnosis is based on endoscopic findings of well circumscribed ulcers (<2cm) usually with a "volcano-like" appearance. Histology findings include multinucleated giant cells with ground-glass nuclei and eosinophilic Cowdry type A inclusion bodies. Histochemical staining or viral cultures are confirmatory. HSE has been described in patients on chronic steroid therapy for COPD or asthma. A recent report described an elderly woman developing HSE within days of initiation of high dose steroids for COPD exacerbation. Patients with COPD exacerbations treated with low dose oral corticosteroids have been reported to have outcomes similar to those treated with more costly and invasive high dose intravenous corticosteroid therapy. Our case is potentially part of an increasing body of evidence that high dose short course steroid therapy can induce HSE. We recommend avoiding high doses of steroids and a high degree of clinical suspicion for HSE, in patients being treated for acute COPD exacerbations who complain of odynophagia.

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### **Title: AEROCOCCUS VIRIDIANS: AN UNUSUAL CAUSE OF INFECTIVE ENDOCARDITIS**

**Aerococcus viridans** is a rare microorganism causing invasive infections in humans. It has been associated with bacteremia, septic arthritis, urinary tract infection, meningitis and endocarditis. They are catalase negative, Gram positive cocci that resemble staphylococci on Gram stain. It is generally considered as a contaminant in clinical cultures, but occasional reports have noted clinically significant roles for this organism in systemic infections.

A 54 year old male complained of intermittent fever, chills, malaise and headaches over 1 month. 2 weeks ago he developed a cough and went to his physician for evaluation. He was given a levofloxacin for 1 week, and felt better. 2 days later, he again developed fever and chills. Blood cultures done at his PMDs office grew *Aerococcus viridans*. An echocardiogram revealed a small echodensity on the posterior mitral valve leaflet. He was sent to the ER for further management. The patient had earlier been in good health, denied any intravenous drug use, and had his last dental cleaning 3 months ago.

Blood cultures were resent from the ER. He was started on IV penicillin 18 million units in divided doses plus gentamicin 1 mg/kg, given 8 hourly. A transesophageal echocardiogram showed normal left ventricular size and systolic function and a mobile echo density approximately 1.3 to 1.4cm on the right coronary cusp of the aortic valve with associated moderate to severe aortic regurgitation. There also was a small echodensity on the mitral valve leaflets with associated trivial mitral regurgitation. No abscess was identified. Both sets of blood cultures sent from the ER grew *Aerococcus viridans*, sensitive to penicillin. A PICC line was then placed and the patient discharged on IV antibiotics for a total of 6 weeks.

*Aerococcus viridans* was first described as a potential human pathogen in 1967. It is isolated as a common airborne organism, and as a marine organism causing fatal disease in lobsters. *Aerococci* can also be found as indigenous inhabitants in the upper respiratory tract and on the skin of normal persons. Infections due to this organism usually occur as a nosocomial infection in association with prolonged hospitalization, antibiotic treatment, invasive procedures, presence of foreign bodies, or neutropenic state. Our patient had no risk factors. *Aerococcus viridans* is usually sensitive to penicillin, although penicillin resistant strains have been isolated. *A. viridans* is rarely associated with human infections, and endocarditis is even rarer. Penicillin should be started empirically pending final sensitivities.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Shirin Attarian  <b>Additional Authors:</b> Sheira Schlair, Santiago Aparo  <b>Institution:</b> Montefiore Medical Center, Albert Einstein College of Medicine</p> <p><b>Title: Cerebral venous sinus thrombosis presenting in pregnancy in a patient with prothrombin gene mutation</b></p> <p>The annual incidence of cerebral venous sinus thrombosis (CVST) is estimated to be 2-4 cases per million adults. Incidence of CVST increases during pregnancy and is about 11.6 per 100,000 deliveries. Although CVST during pregnancy and puerperium is rare, it is a leading cause of morbidity and mortality in this population, and hence its diagnosis and treatment is of particular importance. We report a case of CVST in a young woman during pregnancy. A 27 year old woman (gravida 2, para1) presented with progressive headache in the left parietal region for one day in her 10th week of pregnancy. At the time of presentation, headache was described as throbbing, with severity of 10 out of 10, and associated with nausea, vomiting, and bilateral photo/phonophobia. Physical exam findings including a full neurologic exam were unremarkable. Lumbar puncture was performed, which was unremarkable (WBC=0, RBC=11, Glucose=66 mg/dL), with no growth on culture. Non-contrast brain magnetic resonance imaging/angiogram/venogram showed left sigmoid sinus thrombosis, also including a portion of the left transverse sinus, and the visualized portions of the left internal jugular vein. She was immediately started on low molecular weight heparin with full symptom resolution within 4 days. On further evaluation, she was found to be heterozygous for the G20210A prothrombin-gene. Other thrombophilia work up was negative. Pregnancy was otherwise uneventful, leading to a normal spontaneous vaginal delivery. Anticoagulation was discontinued 6 months after pregnancy without further episodes of venous thrombosis.</p> <p>In pregnancy, the plasma concentration and activity of several proteins involved in blood coagulation and fibrinolysis change. These changes may promote coagulation, decrease anticoagulation, and inhibit fibrinolysis and thus may increase the risk of thromboembolic events, especially among pregnant women who have acquired or genetic risk factors for thrombosis. Among the genetically determined changes, a guanine-to-adenine mutation in the prothrombin gene, associated with elevated plasma prothrombin concentrations and an increased risk of venous thrombosis, has been identified. There appears to be a relation between the presence of the G20210A prothrombin gene mutation and pregnancy related alterations in coagulation or fibrinolysis that lead to thrombosis in women who are carriers of the G20210A prothrombin-gene mutation.</p> <p>The most common pathogenesis of recurrent thrombosis in this population includes hypercoagulable states such as pregnancy and puerperium and the use of oral contraceptive pills (OCP). Cases of CVST in the setting of prothrombin gene mutation are rare, however CSVT carries high morbidity. Carriers should receive anticoagulation after a first venous thrombosis for a similar length of time as patients with a normal genotype.</p>	<p><b>Author:</b> Varnita BAWEJA MD  <b>Additional Authors:</b> John R. Fudyma, MD, Chief and Clinical Associate Professor, General Internal Medicine, University at Buffalo          Jyotsna Bhatnagar, MD, Clinical Assistant Professor, University at Buffalo</p> <p><b>Institution:</b> University at Buffalo</p> <p><b>Title: Primary Mucosal Melanoma Presenting as Recurrent Epistaxis</b></p> <p>Background: Melanomas of mucosal origin are rare compared to their cutaneous counterparts. They account for 0.03% of all new cancer diagnoses and 1.3% of all melanoma cases. Primary mucosal melanomas of the head and neck comprise 55% of all mucosal melanomas. They carry a poor prognosis with less than 25% overall survival rate, and present treatment dilemmas given the higher incidence in the elderly population. Few descriptive studies exist for this rare condition; further studies such as this would thus aid in a better understanding of its presentation.</p> <p>Case Presentation: An 81 year old Caucasian female presented post fall; she reported fatigue, decreased oral intake and weakness in the days preceding the fall. She reported head trauma during the fall but denied loss of consciousness. Review of systems revealed recurrent epistaxis and inability to breathe through the left nostril over the previous six months. A small amount of blood was noted in the left nare with no other evidence of bleeding or bruising. Computerized Tomography (CT) of her head showed a large elongated soft tissue mass approximately 6.3cm in greatest dimension, obstructing the left nasal cavity and extending posteriorly to the left nasopharynx, left Eustachian tube, and maxillary sinus. There was evidence of lymphadenopathy, and perineural tumor spread along the trigeminal nerve with extension into the trigeminal cave, cavernous sinus, and medial aspect of the left middle cranial fossa. There was no evidence of intracranial hemorrhage, midline shift or mass effect. Diagnostic biopsy of the mass revealed a malignant melanoma. With no known history of melanoma and no melanotic lesions identified on detailed physical exam, the patient was diagnosed with a Primary Mucosal Melanoma of the nasal cavity. She was a poor surgical candidate given her advanced age and died within 2 months post diagnosis.</p> <p>Discussion: Due to its lack of visibility, absence of symptoms during the early stages, and no known risk factors, the diagnosis of mucosal melanomas is often delayed. Epistaxis, nasal obstruction, and headache are the most common presenting symptoms of a Nasal Mucosal Melanoma (NMM) and should lead providers to consider a nasal cavity tumor in the differential. Immunohistochemistry is essential for the correct diagnosis and wide surgical excision is the treatment of choice. The local recurrences are common because of the difficulty in achieving complete tumor removal with wider negative margins at this anatomic site. Adjuvant therapy has not been studied in a randomized fashion because of the rarity of the disease but novel agents are being extensively investigated. The challenges associated with a NMM makes it imperative to recognize it early on. Early diagnosis can lead to appropriate management and provide patients and their families realistic expectations of available treatment options and prognosis.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Max Benson MD <b>Additional Authors:</b> Devarapally S, Afzal A, Patel V. <b>Institution:</b> New York Methodist Hospital</p> <p><b>Title: Spontaneous Coronary Artery Spasm: A Case Report</b></p> <p><b>Introduction:</b> Transient coronary artery spasm due to mechanical stimuli in the treated vessel during percutaneous coronary intervention is common, particularly after balloon dilation or stent placement. It usually responds to intra-coronary vasodilators (i.e. nitroglycerin) without any complications. Persistent coronary artery spasm is uncommon and can lead to fatal arrhythmias as well as cardiogenic shock. We report a case of a patient who had severe, extended, generalized spasm of the entire left coronary system during a cardiac catheterization which was not responsive to medical therapy and resulted in cardiogenic shock.</p> <p><b>Case:</b> A 70 year old man with known coronary artery disease presented for elective left heart catheterization after having unstable angina and a positive nuclear stress test at another facility. Patient admitted prior smoking history, cocaine abuse, and denied any allergies. There was no JVD and pulmonary, cardiac, and abdominal exam were otherwise within normal limits. Femoral approach was used and initially there was a patent left main, left anterior descending (LAD), and left circumflex (LCX), and an 80% stenosis in the ostial D1. On second injection of contrast with a different view there was a severe diffuse multi vessel spasm not responsive to intra-coronary nitrates which ultimately resulted in cardiopulmonary arrest. Rescue measures including ACLS and intubation were undertaken and simultaneously bare metal stent was placed in proximal LAD to facilitate flow to the left ventricle (LV). Patient made a full neurologic recovery and was discharged home on optimal medical therapy.</p> <p><b>Conclusion:</b> Intermittent coronary artery spasm is common during catheterizations. Prolonged generalized spasm in the entire left system during catheterization with resultant pulmonary edema and cardiogenic shock however, has only been reported once before. It is important to realize that this is an uncommon complication of catheterization perhaps in individuals prone to vasospasm and that distal local intracoronary administration of vasodilators via the Rapid Transit catheter has been successful in relieving the spasm in at least one other instance and thus maybe useful for other patients in a comparable situation.</p>	<p><b>Author:</b> Prakriti Bista Associate member <b>Additional Authors:</b> Prakriti Bista MD, Rashmi Banjade MD, Jonathan Ang MD, Prasanta Basak MD, Stephen Jesmajian MD</p> <p><b>Institution:</b> Montefiore New Rochelle Hospital and Albert Einstein College of Medicine, New Rochelle, NY.</p> <p><b>Title: Acute Pancreatitis In Ulcerative Colitis</b></p> <p>Many extra-intestinal manifestations of ulcerative colitis (UC) have been described, but pancreatitis is not a classical manifestation. There are a few case reports of patients undergoing treatment for UC presenting with acute pancreatitis. However acute pancreatitis presenting with the initial manifestation of UC is rare.</p> <p>A 26-year old, previously healthy male, presented with diarrhea for 4 weeks. The diarrhea was initially watery, but gradually became bloody and more frequent. On the day of admission, he also complained of severe epigastric pain, radiating to the back. He denied any fever, chills, night sweats, nausea, vomiting, weight loss, recent travel or sick contacts. He denied taking alcohol or any drugs.</p> <p>In the ED, he had a temperature of 101.4 F. CBC and LFTs were unremarkable. Amylase was 418 IU/L and lipase 1733 U/L. CT scan of the abdomen showed diffuse colitis with no evidence of pancreatitis. US abdomen showed normal gall bladder with no stones. Common bile duct diameter was 4mm. Triglyceride level was 40mg/dl. Clostridium difficile molecular assay was negative. Colonoscopy showed active colitis with friable mucosa that easily bled on contact with the scope. Biopsy showed architectural alteration, cryptitis, focal abscesses and chronic inflammation extending to muscularis mucosa, consistent with UC. He was diagnosed with ulcerative colitis and acute pancreatitis and started on IV hydrocortisone 50mg q 6hrs and PO Mesalamine 1600mg q 8hrs. The rectal bleed decreased gradually and stopped completely in 3 days. Hydrocortisone was changed to PO prednisone. Amylase was 72 IU/L and lipase was 120 U/L on the day of discharge. He was discharged on tapering doses of prednisone and sulfasalazine.</p> <p>Clinical symptoms of IBD-associated acute pancreatitis are found in approximately 2% of IBD patients. Identifiable causes of acute pancreatitis include trauma, infection, biliary tract disease, drugs, hereditary, congenital anomalies, hypercalcemia, hypertriglyceridemia and cystic fibrosis. In our patient no cause was identified. Drugs used in ulcerative colitis and implicated in the etiology include: 6-mercaptopurine, 5 mesalamine, steroids, and metronidazole. Our patient had acute pancreatitis before steroids and sulfasalazine were started. Evidence suggests involvement of proinflammatory cytokines in the development of pancreatitis associated with IBD. The concomitant pancreatic over-expression of IL-1 and TNF-a, in patients with acute pancreatitis is in favor of the existence of a pancreatic inflammatory mechanism mediated by proinflammatory cytokines. Although rare, acute pancreatitis can be a presentation of UC. Gastroenterologists are urged to consider expanding the work-up of acute idiopathic pancreatitis to include screening for ulcerative colitis when management of the condition does not result in clinical improvement.</p>
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## Resident / Fellow Clinical Vignette

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**Institution:** St. John's Episcopal Hospital

### **Title: ACUTE PANCREATITIS COMPLICATED BY SPLENIC INFARCTION**

Splenic Infarction secondary to Acute Pancreatitis is uncommon, having an incidence of 7%. This complication occurs when an inflamed pancreas compresses the splenic vessels, leading to splenic infarction. A 55-year-old man, with past medical history of alcohol use, chronic thrombocytopenia, and cholecystectomy, presented with epigastric pain that was described as dull, constant, 8/10 in severity, and radiating to the back. The pain was associated with nausea and vomiting. Physical exam was remarkable for tachycardia and left-sided costovertebral angle tenderness. Laboratory tests were significant for platelets  $42.8 \times 10^3/uL$ , glucose 157 mg/dL, total bilirubin 2.0 mg/dL, lipase 667 U/L, AST 84 U/L, ALT 64 U/L, and for urinalysis with large bilirubin. Urine toxicology and viral hepatitis profile were non-significant. Abdominal ultrasound revealed diffuse hepatomegaly (18.6 cm) and splenomegaly (13.1 cm), with unremarkable common bile duct, hepatic and portal veins. Non-contrast abdominal CT revealed minimal infiltration adjacent to the pancreas, which may represent pancreatitis. The patient was managed conservatively with NPO and intravenous fluid, with gradual improvement in lipase and hepatic profile. However, patient developed Systemic Inflammatory Response Syndrome with fever of 101.7 deg;F and WBC of  $15.7 \times 10^3/uL$ . Contrast-enhanced abdominal CT revealed a resolved pancreatic inflammation, but spleen increased in size to 14.5 cm with scattered areas of infarction. Blood and urine cultures were negative. CXR was normal. Transthoracic echocardiogram and transesophageal echocardiogram were done, but both demonstrated no evidence of intra-cardiac thrombi or pathologic inter-chamber connections. Gadolinium-enhanced MRA of the aorta with its major branches revealed no significant stenosis and persistent multiple wedge-shaped hypodensities consistent with splenic infarctions. Conservative management was continued, and patient eventually improved. According to outpatient follow-up record, the patient remained afebrile without any pain symptoms, and repeat abdominal CT revealed a complete resolution of the splenic infarction. The close anatomical position of the pancreatic tail to the spleen may result to compression due to pancreatic inflammation, causing splenic infarction. There are two known pathological processes that lead to splenic infarction in acute pancreatitis: compression of the splenic vessels by an extensive inflammatory infiltrate, and thrombosis of splenic vein by direct extension of inflammation and hypercoagulability state induced by acute pancreatitis. Compression of the splenic artery with development of arterial spasm may be an additional factor. The current approach to the management of patients with Splenic Infarction secondary to Acute Pancreatitis is mostly expectant management, necessitating aggressive measures if complications such as abscess formation or splenic rupture occur. Splenic infarction in patients with acute pancreatitis can be diagnostically challenging. Other causes of splenic infarction, particularly systemic thrombosis and septic embolism, must be ruled out to make a diagnosis of Splenic Infarction secondary to Acute Pancreatitis.

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**Institution:** Maimonides Medical Center

### **Title: A rare case of Left Ventricular Non-Compaction Cardiomyopathy**

**Introduction -**

Left Ventricular Non-Compaction Cardiomyopathy (LVNCC) is a rare myocardial disorder which results from failure of left ventricle to compact in embryogenesis. It is characterized by a two-layered ventricular wall, an outer compacted epicardial layer and an inner non-compacted layer composed of prominent trabeculations and deep intertrabecular recesses which communicate with LV cavity. Case Presentation -

A 37 year old female with history of CVA presented to our ER with complaints of progressively worsening shortness of breath and decreased exercised tolerance. The physical exam was significant for bilateral crackles, S3 gallop and pitting pedal edema. Laboratory work up showed elevated BNP (617 pg/ml). Patient was admitted to CCU for further work-up and management of new onset heart failure. The echocardiogram revealed severely decreased LV Systolic function (EF <10%) with dilated LV, prominent LV trabeculations and deep intertrabecular recesses communicating with the LV cavity, consistent with LVNCC. A Cardiac MRI was also performed which confirmed the diagnosis. Patient underwent a right and left heart catheterization which showed elevated PCWP (25 mmHg), low cardiac output - 2.25 L/min along with normal coronaries. She was initiated on diuretics, a beta blocker and an ACE inhibitor. Given the history of CVA, she was also started on anti-coagulation with Coumadin. She was discharged in a stable condition to follow up with Cardiology for cardiac transplant evaluation and AICD implantation.

**Discussion -**

LVNCC was first reported in 1926 in association with other congenital cardiac abnormalities. The first description of an isolated case of LVNCC was by Engberding et al in 1984. It is a genetically heterogeneous disorder with a familial and a sporadic form and can be linked to mutations in mitochondrial, cytoskeletal, Z-line, and sarcomeric proteins.

The reported prevalence of LVNCC varies considerably and is estimated to be between 0.014 and 1.3%, however, the true prevalence is unknown as it is often unrecognized. Patients usually present with heart failure, ventricular arrhythmias or systemic embolic events. Echocardiography is the first line imaging modality and diagnostic criteria include end-systolic ratio of non-compacted layer/compacted layer > 2 and evidence of blood flow in intertrabecular recesses from the LV cavity on Color Doppler Imaging, in the absence of other cardiac abnormalities. The Cardiac MRI may be used for confirmation of the diagnosis. The treatment regimen consists of standard heart failure therapy, management of arrhythmias and consideration of oral anti-coagulation in high risk patients to prevent systemic embolic complications. Due to high familial recurrence, genetic testing of the affected individuals and screening of the first-degree family members is also recommended.

**Conclusion -**

Still considered a novel entity, LVNCC is getting increasingly recognized because of heightened awareness and improved cardiac imaging modalities and physicians should be aware of this rare cardiomyopathy.

## Resident / Fellow Clinical Vignette

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### **Title: Breathtaking: A Case of Platypnea-Orthodeoxia due to Patent Foramen Ovale**

Introduction -

Platypnea-orthodeoxia syndrome is a rare disorder characterized by dyspnea (platypnea) and arterial oxygen desaturation (orthodeoxia) accompanying a change to an upright position from a recumbent position.

Case Presentation -

An 87 year old male with past medical history of CAD, Ischemic cardiomyopathy (EF - 20%), lung carcinoma s/p right upper lobe wedge resection was admitted to our hospital with intracranial hemorrhage after a mechanical fall. During the hospitalization, patient had an episode of respiratory distress and had to be intubated. He was found to be in CHF secondary to an NSTEMI (Troponin I - 8.8 ng/ml). The decision was made to manage the patient conservatively and he was transferred to CCU for observation. Patient was diuresed and successfully extubated on day 2.

During his stay in the CCU, he was noted to be desaturating to low 80% while sitting upright with return to normal saturation on lying down. Platypnea-orthodeoxia syndrome was suspected secondary to history of partial pneumonectomy. The echocardiogram revealed a patent foramen ovale (PFO) with a remarkably positive bubble study in upright position as compared to supine position, confirming the diagnosis. The patient underwent successful transcatheter closure of the PFO with an AMPLATZER septal occluder device which resulted in resolution of his symptoms. Patient was subsequently discharged to a rehabilitation facility.

Discussion -

Platypnea-orthodeoxia syndrome was first described by Burchell et al in 1949. The word is derived from the Greek words *platus* (flat) and *pnoia* (breath). Two conditions must coexist to cause platypnea-orthodeoxia: an anatomical component in the form of an interatrial communication and a functional component that promotes abnormal (right to left) shunting when the patient rises from a recumbent to an upright position.

The former may be an atrial septal defect or a patent foramen ovale. The latter may be cardiac, such as pericardial effusion or constrictive pericarditis; pulmonary, such as emphysema, arteriovenous malformations, pneumonectomy, or amiodarone toxicity; abdominal, such as cirrhosis of the liver or ileus; or vascular, such as aortic aneurysm or elongation. Under normal conditions an interatrial communication allows blood to shunt from left to right due to a higher pressure in left atrium than right atrium. The functional components cause an anatomic distortion of the right atrium or the atrial septum resulting in redirection of the shunt flow which causes venous blood to go to the systemic circulation resulting in arterial desaturation. In patients with rare combination of partial pneumonectomy and PFO, the restriction of pulmonary vasculature due to lung resection may lead to generation of a pressure gradient across the interatrial defect.

Treatment of platypnea-orthodeoxia should be directed at the underlying source of vascular shunting. In patients with interatrial defect, percutaneous transcatheter closure is recommended.

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### **Title: Do Hispanics prefer to be Full Code at the End of Life? - The Impact of Palliative Care Consults in Clarifying Advanced Directive Preferences in Spanish-Speaking Patients.**

Background:

Hispanic patients are documented to face disparities in end-of-life care. In contrast to studies that suggest over 80% of Hispanic patients value the importance of comfort over extension of life, they are more likely to die full code, less likely to have discussions regarding prognosis and withdrawal of life support. As the Hispanic population accounts for over half the population growth in the past decade and comprises nearly 20% of the population under the age of 18, the urgency of addressing this disparity grows. Language, socioeconomic status, and medical literacy are identified barriers to patient-focused care. Palliative care teams utilize family meetings and professional translators to address these issues. We evaluated the impact of palliative care consults on the advanced directive preferences of seriously-ill Hispanic and Non-Hispanic White patients.

Methods:

This study is a prospective cohort study of all patients referred to the palliative care service at Santa Clara Valley Medical Center, a 574-bed, teaching county hospital, from 2006 to 2012. Patient who were not Non-Hispanic white or Hispanic were excluded. Their outcomes were followed until discharge or expiration. We evaluated ethnicity, patient language, code status at admission, code status after palliative care consult, and hospice discharge. Chi-squared tests were used to analyze characteristics between three groups: Non-Hispanic White, English-speaking Hispanic, and Spanish-speaking Hispanic patients.

Results:

Out of 925 patients, 511 (55%) were Non-Hispanic White, 208(23%) were English-speaking Hispanic and 206 (22%) were Spanish-speaking Hispanic patients. On admission, there was no statistically significant difference in code status between the three groups ( $p=0.5$ ). After palliative care consults, Spanish-speaking Hispanic patients were more likely to change their code status to DNR/DNI than Non-Hispanic White and English-speaking Hispanic patients (44% Vs 32% Vs 28%,  $p=0.05$ ). Of 243 (26%) hospice referrals that consultations generated, Spanish-speaking Hispanic patients were more likely discharged to hospice ( $p = 0.04$ ).

Conclusion:

Spanish-speaking Hispanic patients were more likely to change from full code to DNR/DNI compared to Non-Hispanic White and English-speaking Hispanic patients despite similar code-status profiles on admission. They were also more likely to be discharged to hospice. Palliative care consults may play an important role in helping patients to align their care with their personal values and may prevent unwanted aggressive interventions at the end of life.



## Resident / Fellow Clinical Vignette

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### **Title: A RARE CAUSE OF ISCHEMIC BOWEL**

**Introduction:** Acute mesenteric ischemia (AMI) is a rare vascular emergency associated with a high mortality rate of 60-80%, most commonly caused by arterial embolization from a cardiac thrombus. We present a unique case of AMI that highlights an uncommon complication of a rare disease in a medically complex patient.

**Case Presentation:** A 55 year-old male with hypertension and hyperlipidemia presented to an outside institution with acute onset of diffuse, crampy abdominal pain, non-bloody vomiting, and watery, non-bloody diarrhea after eating pizza. He denied fevers, sick contacts, recent travel or antibiotic use. CT of the abdomen showed findings consistent with acute enteritis. He was initially managed with aggressive hydration and antibiotic therapy for a presumed intra-abdominal infection. He subsequently developed new-onset atrial fibrillation and was transferred to our institution.

Three weeks prior, he experienced a syncopal event, and outpatient workup included a transthoracic echocardiogram which revealed a large, mobile 5.7 by 1.9 cm left ventricular (LV) mass. Cardiac catheterization was delayed due to his gastrointestinal symptoms.

During hospitalization, the patient's abdominal pain progressed and localized to his periumbilical region and left lower quadrant. He was afebrile, normotensive and tachycardic. Abdominal examination revealed hypoactive bowel sounds and significant left lower quadrant tenderness with rebound. Lab work was significant for leukocytosis (26.1 k/mcL) and an elevated blood urea nitrogen to creatinine ratio (35/1.22 mg/dL). CT angiography showed occlusion of the distal superior mesenteric artery (SMA) with ischemic necrosis, small bowel infarction, and mesenteric venous gas. The patient underwent urgent SMA embolectomy, small bowel resection, and abdominal washout. Cardiac MRI confirmed the presence of an LV mass, which was subsequently excised. Biopsy of the SMA thrombus revealed viable and necrotic malignant tumor cells that were also present in the LV mass.

Two different pathologic interpretations showed angiosarcoma with focal infiltration of the myocardium versus unclassified epithelioid and spindle cell sarcoma, high-grade. A prolonged hospitalization ensued with complications of respiratory failure, seizure disorder, renal failure, and sepsis. The sarcoma was aggressive, metastasizing to the brain, and resulted in intraparenchymal hemorrhage. The patient expired thirty-five days following presentation.

**Discussion:** AMI is rapidly progressive with an increasingly fatal prognosis when diagnosis is delayed. Timely diagnosis depends on high clinical suspicion and remains a challenge due to nonspecific symptoms, labs and imaging results. Primary cardiac tumors are exceedingly rare and are malignant in 25% of cases. Only two case reports have described embolization of a cardiac tumor to the small bowel. Our case is the first to demonstrate confirmation of the embolization with matching histology. Despite our patient's poor prognosis and outcome, this case highlights the need to incorporate clinical context with an attempt to uncover a unifying etiology when formulating a differential diagnosis.

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### **Title: Fever induced Brugada syndrome**

Brugada syndrome (BS) is a genetic disorder with a characteristic ECG finding of persistent or transient ST segment elevation in the right precordial leads with or without right bundle branch block. It is a major cause of life threatening ventricular arrhythmia and sudden death in young men with no evidence of structural heart disease. Here we present a patient with Brugada-type ECG induced by fever.

A 74-year-old female with history of hypertension and recently treated urinary tract infection (UTI) presented to our emergency department (ED) with complaints of fever and dysuria for 3 days. She had recently moved to United States from Peru. In the ED, she was noted to have a temperature of 103.8°F, tachycardia at 110 bpm with a blood pressure of 129/49 mm Hg; her exam was unremarkable otherwise. Laboratory work was notable for leukocytosis with bandemia and pyuria. She was being admitted for sepsis from a UTI. An ECG revealed sinus tachycardia and coved ST segment elevation in V1 and V2, more pronounced in V2, characteristic of Brugada pattern ECG. Echocardiogram showed normal left ventricular systolic function with an ejection fraction of 55-60% and no segmental wall motion abnormalities. The cardiac biomarkers were negative. After 48 hours on intravenous antibiotics, she showed clinical improvement and had no further fevers. The Brugada pattern on ECG disappeared with defervescence of fever. Two months later, the patient continues to remain well, with a normal ECG. Pedro Brugada and Josep Brugada described Brugada syndrome in 1992. An autosomal dominant pattern of transmission is reported in about 50% of familial cases. The ECG changes in Brugada syndrome are dynamic, often concealed and may reveal themselves in the presence of triggers like fever, intoxication, electrolyte imbalance and sodium channel blockers. In a study, the prevalence of type I Brugada pattern was found to be 20 times higher among patients presenting with fever than in afebrile patients (2% vs 0.1%, P = 0.0001). These patients are at risk of sudden death due to polymorphic ventricular tachycardia or ventricular fibrillation. The disorder manifests itself either spontaneously in the third or fourth decade of life or is triggered by medications or physiological stressors. Functional expression studies of a genetic mutation identified in patients with BS shows that loss of function of sodium channel current was accentuated at higher temperatures. BS should be included in the differential diagnosis of ST elevation in a patient with fever. A timely management of fever is crucial to avoid any malignant arrhythmia. Risk stratification of asymptomatic patients with a Brugada-type EKG induced by fever and a negative family history of syncope or sudden death remains controversial. These patients seem to have good prognosis and should be on regular follow up.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Rakeeba Din <b>Additional Authors:</b> Thyrotoxic periodic paralysis: Two cases in three months Shabnam Rehman MBBS, Adam Riordan DO, Paul Campagna DO, Nashat Rabadi MD, Henri Woodman MD <b>Institution:</b> University at buffalo Catholic health System</p> <p><b>Title: Thyrotoxic Periodic paralysis: two cases in a row!</b></p> <p>Introduction: Thyrotoxic Periodic Paralysis (TPP) is an uncommon disorder characterized by too much thyroid hormone, hypokalemia and muscle weakness or paralysis that alternate with periods of normal muscle function. Case presentation: 22 y-o- Caucasian and 28-y-o- AA male with no other significant past medical history, present to the emergency room at two different occasions with similar presentation of acute onset bilateral lower extremity weakness and inability to move. Both patients had similar symptoms in the past. Examination revealed severely diminished muscle strength in lower extremities and absent deep tendon reflexes. Initial work up showed potassium levels of 1.6 and 1.9 with magnesium of 1.4 and 1.5, respectively. TSH levels were found to be 0.05 and 0.01 with free T4 of 2.89 and 4.02, respectively leading to the diagnosis of thyrotoxic periodic paralysis. Appropriate thyroid work up was ordered to establish the cause of hyperthyroidism besides treating patients with IV potassium, magnesium, propranolol and methimazole leading to complete resolution of weakness within hours of presentation.</p> <p>Discussion: TPP is a channelopathy with an incidence of approximately 2% in patients with thyrotoxicosis of any cause. Hypokalemia results from an intracellular shift of potassium induced by thyroid hormone sensitization of Na<sup>+</sup>/K<sup>+</sup>-ATPase. It has been linked to genetic mutations in genes coding for certain ion channels- L-type calcium channel <math>\alpha_1</math>-subunit and potassium inward rectifier 2.6. Early recognition of TPP is vital to initiating appropriate treatment and avoiding the risk of rebound hyperkalemia that may occur if high-dose potassium replacement is given.</p>	<p><b>Author:</b> Neha Gupta MD <b>Additional Authors:</b> Shipra Gandhi MBBS, Austin Miller PhD, Laurie A. Ford, Elizabeth A. Griffiths MD, James E. Thompson MD, Meir Wetzler MD, Eunice S. Wang MD</p> <p><b>Institution:</b> University at Buffalo</p> <p><b>Title: Comparison Of Epigenetic Versus Intensive Chemotherapy For Newly Diagnosed Acute Myeloid Leukemia Patients =60 Years Old: The Roswell Park Cancer Institute Experience</b></p> <p>Purpose Epigenetic therapy (Epi) with the hypomethylating agents, azacitidine (Aza) and decitabine (Dec), is increasingly being utilized for induction treatment of older AML patients based on studies demonstrating both tolerability and prolonged survival. By contrast, standard "7+3" intensive chemotherapy (IC) effectively induces remission in many individuals but is associated with significant toxicity and higher mortality. We compared our institute's experience with Epi vs. IC for the upfront treatment of newly diagnosed AML patients =60 years old.</p> <p>Methods We performed a retrospective chart review of 164 patients = 60 yrs old with newly diagnosed AML who underwent initial therapy at our center between 3/2008-2/2013. Half (n=84; 51%) received IC with regimens containing cytarabine 100 mg/m<sup>2</sup> IV for 7 days and daunorubicin 60 mg/m<sup>2</sup> IV for 3 days. Half were treated with Epi (n=82; 49%) regimens containing either Dec (20 mg/m<sup>2</sup> IV daily for 5 or 10 days) or Aza (75 mg/m<sup>2</sup> sq daily for 7 days). Kaplan Meier method, log rank test, and univariate cox proportional hazard models were used to assess overall survival (OS) and correlation with covariates of interest.</p> <p>Results Baseline patient characteristics demonstrated a difference in median age of patients in each group (IC 67 vs. Epi 75 yrs; p &lt;0.01). All other factors were comparable. At our center, older AML patients receiving IC had superior complete response at any time point (CRatp)(43% vs. 21%; p&lt; 0.01). IC also resulted in a longer median OS compared to Epi (10.6 vs. 7.9 months; p=0.01). Thirty-day mortality and leukemia-free survival (LFS) were similar across the two groups (IC 10% vs. Epi 11%; p=0.8; 11.2 vs. 9.3 mos; p=0.47 respectively). Choice of Epi drug (Azacitidine vs. Decitabine) did not impact results. In multivariate Cox regression analysis, older age, higher ECOG score, increased peripheral blasts, and poor-risk cytogenetics were independently associated with inferior survival.</p> <p>Conclusions Our results suggest that IC and Epi represent clinically equivalent approaches for the upfront treatment of older AML patients. In spite of significantly higher CR and ORR in the IC group, our finding of improved OS following IC vs. Epi was not substantiated in multivariate analysis, suggesting that this difference may be explained by the comparatively younger age of patients in the IC group. Leukemia-free survival and 30-day mortality were the same for IC vs. Epi-treated patients, as were all response and survival outcomes in the poor-risk cytogenetics subgroup. These data highlight the growing need for prospective clinical trials to conclusively determine the respective roles of IC vs. Epi therapy in older AML patients.</p>
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## Resident / Fellow Clinical Vignette

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**Title: NON-ISCHEMIC CARDIOMYOPATHY IN A PATIENT WITH MULTIPLE MYELOMA WITHOUT AMYLOIDOSIS**

Background: Non-ischemic cardiomyopathy refers to a diverse array of myocardial disorders characterized by structural and functional remodeling of the left ventricle in the absence of significant coronary artery disease. Cardiac magnetic resonance imaging (MRI) provides an accurate and reliable assessment of left and right ventricular anatomy and function, and cardiac MRI with gadolinium contrast has emerged as the dominant imaging modality in diagnosis of non-ischemic cardiomyopathy. Myocardial fibrosis in non-ischemic cardiomyopathy is a substrate for arrhythmias and further cardiac remodeling, predisposing to future adverse cardiovascular events. Delayed gadolinium enhancement on cardiac MRI is an effective method to assess for myocardial scar or fibrosis. We report a rare case of a patient with non-ischemic cardiomyopathy and newly diagnosed multiple myeloma who had diffuse subendocardial delayed enhancement on cardiac MRI, fibrosis on cardiac biopsy without amyloidosis and a fatal course.

Case: A 69-year old male of Middle-Eastern descent presented to the emergency room with 2 days of worsening exertional dyspnea and intermittent, non-exertional, retrosternal chest pain. An electrocardiogram showed sinus rhythm with a previously known left bundle branch block. He had an elevated troponin I (0.25 ng/mL) and was taken for cardiac catheterization. He was found to have non-obstructive coronary artery disease with a severely reduced left ventricular ejection fraction of 30%. On workup for non-ischemic cardiomyopathy, patient was found to have an elevated free kappa light chain (641.2 mg/L) with an abnormal kappa/lambda light chain ratio of 58. Urine electrophoresis showed a monoclonal band with a concentration of 52%. Urine immunofixation confirmed the monoclonal band to be free kappa light chain. Bone marrow biopsy was consistent with multiple myeloma with approximately 10% of the total plasma cells clonal for kappa light chain. Cardiac MRI showed a diffuse increase in subendocardial T2 signal and delayed gadolinium enhancement. Endomyocardial biopsy showed interstitial fibrosis but no evidence of amyloid. The patient was discharged home with outpatient cardiology and oncology follow-up. He was subsequently started on lenalidomide and dexamethasone for treatment of multiple myeloma. He was re-hospitalized a month later with heart failure exacerbation requiring intra-aortic balloon pump and inotropic support and had a fatal course.

Conclusion: Non-ischemic cardiomyopathy in the absence of cardiac amyloidosis in multiple myeloma has been reported only twice in the literature. Myocardial fibrosis in non-ischemic cardiomyopathy is a substrate for arrhythmias and further cardiac remodeling, predisposing to future adverse cardiovascular events. Cardiac MRI can detect presence or absence of myocardial fibrosis by delayed enhancement and provide excellent prognostic information that can help guide risk stratification of patients with non-ischemic cardiomyopathy.

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**Title: Look for the CMV!!!**

Introduction: Splenic infarction is caused by a clot in the splenic artery presenting with left upper quadrant abdominal pain, fever, chills. Predisposing factors include inherited or acquired clotting diseases, such as sickle cell disease and thalassemia. Sickle cell trait however is a rare cause of infarction and has been described in patients exposed to high altitude or excess physical exercise. Other causes of splenic infarction include malignancies, emboligenic disorders and infections including babesiosis, malaria and CMV.

Thrombosis related to CMV infection has been described in medical literature in the immunocompromized host, but rarely in an immunocompetent individual.

Case Presentation: A 54 year old African American male with history of sickle cell trait, non insulin dependent diabetes mellitus, hypertension and dyslipidemia presented to ED with left upper quadrant abdominal pain, constant, none radiating with no modifying factors. The patient had complains of flu-like symptoms for past 9 days with low grade fevers, headache, fatigue, nausea, watery diarrhea in the first 3 days followed by left upper quadrant abdominal pain becoming progressively worse. The patient had also decreased appetite due to nausea and altered taste. There was no associated emesis, upper respiratory symptoms, chest pain, skin rashes or lymphadenopathy. The patient had been previously in good health, active without a history of similar complains. He denied history of blood transfusions, recent travel or recent altitude change. In the ER, he had a temp of 102F, and HR 107, with distended abdomen and tenderness in LUQ with negative FOBT. The balance of physical examination was unremarkable. Admission labs; WBC of 13.8, Hgb 12.7, Hct 37.4, LDH 463, ALT/AST 124/113, CRP 61. A stat CT of the abdomen revealed two wedge shaped splenic infarcts 5.7 and 2.1 cm. Additional laboratory diagnostics; showed a positive hemoglobin-S trait, low protein C and S activity, normal cardiolipin antibodies and no lupus anticoagulants. EBV and CMV IG were performed showing a non acute EBV and an elevated CMV IgM of 3.8, (CMV DNA PCR 85610 copies/ml), indicating an acute CMV infection. The Patient was treated supportively with resolution of his abdominal pain over 3 days.

Conclusion: CMV induced thrombosis is thought to be caused by a transient increase in anticardiolipin and antiphospholipid antibodies production levels which normalize following resolution of the infection. This is the most widely held view in the medical literature. However in this case our laboratory data showed normal levels of these antibodies thus placing into question the exact mechanism of splenic thrombosis. Alternatively does CMV infection with edema and inflammation create a hypoxic environment in the spleen leading into sickling, sequestration and subsequent thrombosis???

## Resident / Fellow Clinical Vignette

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**Institution:** Flushing Hospital Medical Center

### **Title: Kew Gardens Fever Returns**

#### Introduction:

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 papulovesicular 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 Northeastern metropolitan areas. The early 2000s witnessed another peak of incidences during the time of anthrax attacks, as the initial lesion of Rickettsialpox 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, myalgia, 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 rickettsialpox. Repeat serology for rickettsialpox and skin biopsy of eschar were sent for analysis. Subsequently, the rash spread, becoming papulovesicular without mucous membrane involvement or lymphadenopathy; patient's symptoms improved slowly. Results showed positive rickettsial antibodies in serology as well as a positive skin biopsy by immunohistochemical (IHC) stains for rickettsial organisms, confirmed by PCR, all performed at the CDC. Patient was discharged with course of doxycycline.

#### Discussion:

Rickettsialpox is caused by Rickettsia akari, transmitted by the mouse mite, residing in its host—the house mouse. During the initial outbreak investigation, the apartment incinerators were not operated daily, causing a buildup of waste, which attracted the mice. 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 rodent control is required for prevention under the domain of public health. As in this case, when initial serology is negative and clinical suspicion remains high, biopsy can be performed for IHC staining and PCR to confirm the diagnosis—mimicking Rickettsia rickettsii in Rocky Mountain spotted fever.

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### **Title: Brain Freeze**

#### Introduction:

The hypothermia protocol is believed to provide neuroprotection during ischemia via a reduction in energy usage as well as reducing reperfusion injury, i.e. damage caused by oxidative stress when the blood supply is restored to tissues after a period of ischemia. Inflammatory responses during reperfusion include increased intracranial pressure, which leads to cell injury and cell death. Hypothermia has been shown to help moderate intracranial pressure and minimize the harmful effects of a patient's immune responses during reperfusion. This case describes a unique presentation of bilateral globus pallidus infarction after a patient was on hypothermia protocol status post cardiac arrest secondary to respiratory arrest from complications of drug use.

#### Case presentation:

A 28-year-old male with history of polysubstance abuse was found unresponsive with respiratory arrest by EMS. During intubation, patient became pulseless, ACLS initiated with return of spontaneous circulation within 15 minutes. Patient arrived in ER, intubated, unresponsive to all stimuli, and hypotensive. Hypothermia protocol initiated with vecuronium and ativan drips; vasopressors also administered. Urine toxicology positive for cocaine, benzodiazepine, and methadone, alcohol screen was negative, troponins elevated at 0.814 with EKG showing sinus tachycardia at 120 beats per minute and no ST changes. Patient was admitted to ICU. Rewarming process began after 24 hours and patient hemodynamically stabilized over the next few days. Initially, patient could not be assessed neurologically due to sedation but improved after drips tapered off. Patient first exhibited spontaneous opening of eyes and then began to respond to simple verbal commands, undergoing successful CPAP trial and extubation. Patient subjectively felt weak from prolonged bedrest but on exam, displayed no cognitive impairment, motor deficits or involuntary movement. MRI head showed acute ischemic infarct in the globus pallidus bilaterally. Patient improved during hospital stay, transferred out of ICU, underwent physical therapy, ambulating with straight cane. Patient discharged with referrals for continued physical therapy and drug rehabilitation.

#### Discussion:

Classically, after cerebral infarction, the globus pallidus is selectively spared in contrast to the caudate and putamen. When the opposite occurs, i.e. in selective globus pallidus involvement, the known causes include carbon monoxide poisoning, MDMA, cocaine, opiates, and cyanide poisoning. The clinical presentation of globus pallidus infarct often involves acute cognitive impairment, personality changes, ataxia, coma, or even death. In the present case, the patient sustained cardiac arrest after developing respiratory arrest from the depressive effects of methadone. The combination of cocaine as a vasoconstrictor and methadone increasing the intracranial pressure led to patient having bilateral globus pallidus infarct. However, the neuroprotective effect of the hypothermia protocol allowed the patient to recover completely, with no residual deficits.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> OLANREWAJU ISHOLA MD Associate ACP <b>Additional Authors:</b> Kabir Olaniran MD, Vipul Shah MD, FACP. Department of Medicine, Lincoln Medical and Mental Health Center, Bronx, New York <b>Institution:</b> LINOLN MEDICAL AND MENTAL HEALTH CENTER</p> <p><b>Title: NEW ONSET ASCITES AND PERICARDIAL EFFUSION INDEPENDENT OF CONCURRENT CIRRHOSIS</b></p> <p><b>Introduction:</b> Although well documented in the literature, only 4% of cases of primary hypothyroidism are complicated by ascites. Because most cases of new onset ascites are related to liver dysfunction or malignancy, hypothyroidism is often overlooked. Thyroid-related ascites only occurs in severe hypothyroidism and may present without the classic features of myxedema, thus making an early and accurate diagnosis challenging.</p> <p><b>Case Report:</b> A 54-year-old male presented with progressive abdominal distension of 5 months duration associated with constipation. There was no jaundice, abdominal pain or vomiting and appetite was preserved. His medical history was notable for prior chronic alcohol abuse, benign prostatic hypertrophy and hypothyroidism due to radioactive iodine ablation for Graves' disease. The patient was not compliant with his levothyroxine but had an active lifestyle. On examination his vital signs were normal, mental status was excellent and reflexes were normal globally. The abdomen was distended with fluid thrill. No peripheral stigmata of chronic liver disease were found. Cardiopulmonary exam was normal. There was no limb edema. There were no clinical signs of heart failure or infection. Initial investigations showed a normal serum albumin of 4.2g/dl (3.7-5.1g/dl) and an INR of 1.26 (0.8-1.34). Transaminases were normal. Electrocardiogram was significant for low voltage. Abdominal CT scan showed liver cirrhosis with large ascites and no lymphadenopathy and an incidental finding of mild pericardial effusion. Paracentesis was performed revealing clear fluid with a serum albumin-ascites gradient of 1.0g/dl which was not suggestive of portal hypertension. Further ascitic fluid studies showed a protein level of 4.1g/dl, lymphocytosis, a negative Gram stain and culture and normal adenosine deaminase. Hepatitis serologies, Quantiferon and iron studies were also within normal range. Multiple tumor markers were also negative. Serum TSH, however, was 54.765 IU/ml (0.35-4.8 IU/ml). The free thyroxine level was 0.57ng/dl (0.9-1.9ng/dl). The patient had been scheduled for diagnostic laparoscopy but this was cancelled. He was discharged home on levothyroxine. Follow up TSH levels were 47.4 IU/ml and 23.095 IU/ml at 4 and 6 weeks post discharge respectively. By 6 weeks, the ascites had completely resolved. No other medications had been prescribed.</p> <p><b>Discussion:</b> Myxedema is a rare cause of ascites but even delayed diagnosis has favorable outcomes as treatment with levothyroxine typically leads to the complete regression of ascites. A high index of suspicion is required to diagnose these cases. More common etiologies must be ruled out even when concurrent, as in our patient. A systematic approach with proper interpretation of ascitic fluid studies may spare the patient unnecessary interventions such as diagnostic laparoscopy and biopsy.</p>	<p><b>Author:</b> Neville Jadeja MD <b>Additional Authors:</b> Shabbir, Syed;Kozicky, Mark; Opare-Addo, Paul; Jorge Cabrera, Valerie</p> <p><b>Institution:</b> Jacobi Medical Center , Albert Einstein College of Medicine</p> <p><b>Title: NON KETOTIC HYPERGLYCEMIC CHOREA BALLISM (NKHCB) : AN UNCOMMON PRESENTATION OF HYPERGLYCEMIC STATES</b></p> <p><b>Case Presentation:</b> We present the case of a 71 year old female presenting to the emergency room with an acute right upper extremity tremor. The patient was in her usual state of health, watching television at home when she noticed a tremor of her right upper extremity. The tremor worsened with intentional movement of the arm and the movements kept her awake all night. She described her arm as having a "mind of its own". She denied any numbness or weakness but had more difficulty holding things with her right hand. Except for worsening polyuria and polydipsia in the week prior to her presentation, the patient didn't have any other complaints. The patient had a past medical history significant for type II diabetes mellitus, hypertension and a prior history of stroke without residual deficits. She reported not taking any of the medications for diabetes during the past few weeks. On admission, the patient had a heart rate of 116 and a blood pressure of 151/80 mmHg. Neurologic examination was significant for a kinetic and postural tremor. She also appeared to have hemiballismus of the right arm at rest. Her initial serum glucose was 711 mg/dL. Serum and urine ketones were negative and a glycosylated hemoglobin (HgbA1C) was 11.5%. CT imaging of the head showed mild cerebral atrophy and chronic small vessel ischemic changes. She was diagnosed with non ketotic hyperosmolar state and the movements completely subsided hours later after fluid administration and glycemic control using long acting and short acting insulin.</p> <p><b>Discussion:</b> Non Ketotic Hyperglycemic Chorea Ballism (NKHCB) is a rare movement disorder characterized by unilateral hemichorea-hemiballism, contralateral abnormality on the putamen and caudal nucleus on imaging and rapid resolution of symptoms following glycemic control. Its pathophysiology is not clearly understood. SPECT and PET studies have shown hypofunction of the striatum, suggesting a role for regional metabolic dysfunction secondary to hyperglycemia and vascular insufficiency. We describe an atypical case of NKHCB limited to the right upper extremity characterized by both a kinetic and postural tremor of fluctuating severity. It is hoped that this case would increase the awareness of this disorder among providers and to the understanding of disease pathophysiology.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author</b> Faizul Suhail, MD <b>Additional Authors:</b> : Robert Siegel, MD. James J. Peters VA Medical Center, Mount Sinai School Of Medicine Mark mark.korsten@va.gov <b>Institution:</b> Mount Sinai School of Medicine <b>Title:</b> <b>PLEURAL EFFUSION IN CHRONIC LYMPHOCYTIC LEUKEMIA</b></p> <p>INTRODUCTION: Chronic Lymphocytic Leukemia (CLL) is characterized by clonal proliferation and accumulation of mature lymphocytes. Pulmonary involvement in CLL is uncommon and pleural effusion is a rare complication of CLL.</p> <p>CASE REPORT: 64 year old African American male smoker presented with shortness of breath and dry cough for the past few days. Physical examination revealed tachycardia and he had reduced breath sounds on the right hemithorax. He was afebrile and oxygenating normally on room air. Laboratory tests revealed new onset leukocytosis of 29,000 with normal hemoglobin and platelet counts. Chest film showed large right pleural effusion and compression atelectasis of right lung. CT chest with contrast confirmed right pleural effusion but also showed enlarged retrocrural lymphadenopathy, which was new since previous imaging in 2010. Right sided tube thoracostomy was performed and about 2.5 L of hemorrhagic pleural fluid was drained. Pleural fluid analysis showed an exudative effusion, which was predominantly lymphocytic. Pleural fluid cell count was: RBC 110,250/mm<sup>3</sup>, WBC 3,238 with 76% lymphocytes and only 6% neutrophils. Pleural fluid LDH was 305 and protein count was 3.9, while serum LDH was 241 and protein count 4.1. Cultures of pleural fluid were negative. Subsequent CT chest showed resolution of pleural effusion and right paratracheal, hilar and subcarinal along with mediastinal and retrocrural lymph adenopathy. Pleural fluid cytology was negative for malignant cells but showed atypical cells, lymphohistiocytic cells, eosinophils and scant reactive mesothelial cells. Flow Cytometry on pleural fluid was positive for CD 23, CD 20 and aberrant expression of CD 5, consistent with CLL. Further work-up for staging included bone marrow biopsy and CT Abdomen and Pelvis to rule out hepatosplenomegaly and intra-abdominal lymphadenopathy. Bone marrow Biopsy confirmed the diagnosis of CLL with CD 38 and ZAP-70 both negative.</p> <p>DISCUSSION: The differential diagnosis of pleural effusion in CLL includes infection, lymphatic obstruction from mediastinal lymph nodes, previous chemotherapy, radiotherapy or primary involvement by CLL. In primary pleural infiltration by CLL the lymphocytes are predominantly B cells. Whereas in reactive pleural effusion due to other causes the lymphocytes are predominantly T cell. In pleural infiltration by CLL, pleural fluid is usually hemorrhagic. The study of clonality by Flow Cytometry and Immuno-Histo Chemistry is required to provide a definitive diagnosis. Patients with pleural involvement by CLL, usually have a long standing history before pleural effusion develops. Rarely, CLL can present with pleural effusion as in our case. The pleural effusion responds well to systemic therapy. Flow Cytometric evaluation is an important diagnostic tool in patients with new onset hemorrhagic pleural effusion and clinical suspicion of CLL, which might be missed if cytology without flow cytometry is obtained.</p>	<p><b>Author:</b> Kristine Jang D. O. <b>Additional Authors:</b> Vanya Grover, Adebayo Shakir Adewale, Tahmeena Ahmed, and Nand K. Wadhwa <b>Institution:</b> Stony Brook University Hospital <b>Title:</b> <b>A CASE REPORT: CHALLENGES OF DIAGNOSING SARCOID-LYMPHOMA SYNDROME</b></p> <p>Background: Malignancy associated with Sarcoidosis was first suggested by Brincker and Wilbek in 1974. Subsequent case reports have documented that sarcoidosis and lymphoma can be detected simultaneously or one may precede the other. We report a case of patient requiring multiple hospitalizations until he developed hypercalcemia with acute kidney injury and the diagnoses of sarcoidosis and marginal zone lymphoma were made.</p> <p>Case History: An 81 year old man with hypertension, anemia, COPD, and coronary artery disease, presented with altered mental status, dragging of left foot, and worsening lethargy. Prior to this admission, the patient was admitted twice and treated for pneumonia within the 6- month period. Vital signs were unremarkable. Neurological exam revealed an awake, alert, and oriented only to person and place without focal neurological deficits. Chest exam showed bibasilar crackles. He had no palpable lymphadenopathy but had mild splenomegaly. Initial chest radiograph showed left pleural effusion without perihilar lymphadenopathy. Laboratory data showed WBC 6.2 k/mcL, hemoglobin 10.1 mg/dL, platelets 139 k/mcL, serum sodium 137 mEq/L, potassium 4.4 mEq/L, chloride 104 mEq/L, and bicarbonate 30 mEq/L, BUN 34 mg/dL, creatinine 2.4 mg/dL, calcium 13.3 mg/dL, angiotensin converting enzyme (ACE) 107 U/l and 1,25 dihydroxy vitamin D 103 ng/ml. Bone marrow clot section showed non-caseating granulomas and a small lymphocytic infiltrate composed of clonal B cells consistent with immunophenotype of marginal zone lymphoma by serum and bone marrow flow cytometry. He was treated with IV hydration and prednisone for sarcoidosis. Follow up laboratory showed normalization of serum calcium to 9.8 mg/dl and decreased creatinine of 1.67 mg/dl over a period of one week. He was discharged home on tapering doses of prednisone. Patient did not require treatment for marginal zone lymphoma since patient remained asymptomatic. Over a 2- year follow up period, serum calcium, ACE, and 1,25 dihydroxy vitamin D remained normal and patient remained clinically asymptomatic.</p> <p>Discussion: Coexistence of sarcoidosis and lymphoma was often refer to as sarcoid-lymphoma syndrome. In this case, the diagnosis of sarcoid-lymphoma syndrome was not made initially due to lack of sufficient classic findings associated with them. Hypercalcemia and acute kidney injury led to a series of laboratory testing and eventually bone marrow biopsy to make the diagnosis. This case was unique because unlike many of the previous reported cases of sarcoid-lymphoma syndrome, patient did not require treatments for lymphoma and was only treated for sarcoidosis. Understanding that bone marrow granulomas may be seen in patients with lymphoma but are nonspecific and do not usually cause elevated ACE and hypercalcemia led to an appropriate therapy. The exact relationship between sarcoidosis and lymphoma is not well understood; however, based on clinical manifestations unique to each disease entity, an appropriate selection of therapy can be made.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Henry Jean MD <b>Additional Authors:</b> Dr. Saka Kazeem, Sajid Mithaiwala MS-III, Jerry Jomi MS-III <b>Institution:</b> Kingsbrook Jewish Medical Center</p> <p><b>Title: Myxedema Coma with Subclinical Hypothyroidism: A Rare Finding</b></p> <p>Although myxedema coma has become a very rare complication of hypothyroidism because of much prompt diagnosis and treatment, it remains one of the most dreaded endocrine complications for any physician, including endocrinologists. Typically seen in patients with severe hypothyroidism, its development in patients with subclinical hypothyroidism is extremely unusual. Only few cases have been reported and the exact pathogenesis has remained unexplained. The following is a case of myxedema coma due to subclinical hypothyroidism in a patient with few co-morbidities who presented to our hospital.</p> <p>A 91-year-old female presented from home for confusion of one week duration. The patient was awake, but lethargic and oriented only to person. She presented with sluggish movements, slow and slurred speech and a temperature of 95&amp;deg;F. She had no prior hospitalizations and was not on any medication although she had a history of hypertension and leg ulcers. Upon arrival to the hospital the patient’s vital signs were as follows: BP: 151/82 and pulse rate of 100. Thyroid function tests showed subclinical hypothyroidism (TSH: 7.0, Free T4: 0.89, T3: 51). The patient’s biochemical profile demonstrated normoglycemia (Glu: 91), elevated liver enzymes (ALK-P: 96, ALT: 79, AST: 100), leukopenia (WBC: 3.1), EKG and CT of the head revealed no significant findings. CXR showed pulmonary congestion but no infiltrates or effusion. A workup for sepsis was initiated based on her initial findings and IV Rocephin and Zithromax were administered. Oral levothyroxine was initiated for the subclinical hypothyroidism. The patient’s status continued to deteriorate and she remained hypothermic. Blood cultures were negative for bacterial growth. ACTH stimulation tests ruled out adrenal insufficiency and serum FSH and LH were within the normal range. Antibiotics were stopped and IV Synthroid was then started. The patient’s cognition, speech and language improved significantly, as did her vital signs, including temperature. The patient is currently being managed as an outpatient and is doing well on PO Synthroid.</p> <p>This case illustrates an unusual presentation of myxedema coma, one with signs and symptoms more typical of other abnormalities, such as sepsis. We are reminded that myxedema coma should not be overlooked in elderly patients presenting with altered mental status. Physicians should consider myxedema coma in the setting of altered mental status and other related symptoms, with subclinical hypothyroidism as part of the differential diagnosis.</p>	<p><b>Author:</b> RAAGSUDHA JHAVAR <b>Additional Authors:</b> K Patel, K Thein, J Patel, R Mukherji</p> <p><b>Institution:</b> KINGSBROOK JEWISH MEDICAL CENTER</p> <p><b>Title: AN UNUSUAL CASE: LOW-DOSE METHOTREXATE INDUCED PANCYTOPENIA IN RHEUMATOID ARTHRITIS.</b></p> <p>Low-dose Methotrexate is well tolerated and efficacious when used in Rheumatoid Arthritis (RA). We report the unusual complication of near-fatal pancytopenia in a patient receiving low-dose Methotrexate.</p> <p>An 85-year-old African American female with a 6 year history of seropositive RA, chronic renal insufficiency, hypertension and diabetes mellitus was admitted with 3-days of oral ulcers, dark colored loose stools and myalgias. She had been in her usual health until 3 days prior to admission and had been on 7.5 mg of Methotrexate monthly, without significant side effects. Her hematologic laboratory values had been stable for 6 years. On admission, the patient appeared ill and had generalized pain. The temperature and other vital signs were within normal limits. She had oral mucosal ulcerations which had bled recently. The abdomen was soft, mildly tender without organomegaly. She had mild synovitis of her knees and hands with limitation of movement. A chest radiograph and electrocardiogram were normal. Blood, urine and respiratory cultures sent on admission, proved to be negative. Cat scan of abdomen showed possible colitis. Her initial blood work compared to her usual baseline, showed a drastic change with pancytopenia and acute on chronic renal failure.</p> <p>The leukocyte count was 2.7x10<sup>3</sup>/mm<sup>3</sup>, hemoglobin 7.1g/dl, hematocrit 23%, platelets 28x10<sup>3</sup>/mm<sup>3</sup>, MCV 101.7micrometer<sup>3</sup>, urea 25 mg/dl and creatinine 1mg/dl. Peripheral blood smear showed a low platelet count but no blasts, teardrop or immature cells. The patient was started on aggressive intravenous hydration and empiric intravenous antibiotics for pancytopenia and colitis. She was transfused packed red cells and platelets and was given granulocyte colony stimulating factor injections. (G-CSF). Despite these measures the patient did not improve clinically or biochemically. Next, a bone marrow biopsy was done and revealed no evidence of a lymphoproliferative disorder or any increase in marrow blasts. A tentative diagnosis of Methotrexate induced pancytopenia was entertained. Once the patient was started on intravenous steroids and folinic acid, she improved dramatically and was discharged home on the 7th day of hospitalization.</p> <p>Low-dose Methotrexate induced pancytopenia is very uncommon, and when it does occur, a frequent predisposing factor may be chronic renal insufficiency. The exact mechanism is not yet known. Our case illustrates the fact that severe pancytopenia from low- dose Methotrexate therapy can occur without any warning, even in patients who have tolerated the drug for many years without complications. It is important to be aware of this entity and patients presenting with a pattern similar to our case should be given a trial of systemic steroids and folinic acid after stopping Methotrexate. In addition, physicians prescribing low-dose Methotrexate to patients who are stable should not be complacent, since potentially fatal, severe pancytopenia can appear at any time without warning.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Arunpreet Kahlon Resident <b>Additional Authors:</b> Amrita K. Dhillon M.B,B.S, Ganesh Aswath M.B,B.S, Najam Ud Din MD, Jonathan Wright MD. <b>Institution:</b> SUNY UPSTATE MEDICAL UNIVERSITY</p> <p><b>Title: LACTATEMIA OF UNKNOWN SIGNIFICANCE IN A PATIENT WITH GLIOBLASTOMA</b></p> <p><b>Introduction:</b> Elevated lactate levels are usually seen in patients who have cellular hypoxia which can be either due to decreased blood flow to the cells, decreased oxygen in the blood reaching the cells or inability of the cells to utilize that oxygen. Since lactate is produced as lactic acid, it usually causes a positive anion gap acidosis when produced in significant quantity in the body. We are presenting a rare case of chronically elevated lactate levels in a patient with glioblastoma.</p> <p><b>Case:</b> A 37 year old patient with a past medical history of known glioblastoma multiforme status post ventriculopreitoneal shunt, glucocorticoid induced diabetes, admitted for treatment of newly found deep vein thrombosis. Except for bilateral leg swelling, patient was asymptomatic with vital signs within normal range. On lab workup it was found that patient had an anion gap of 20, but her serum bicarbonate levels were within normal range. Further workup revealed a plasma lactate level of 5.5 mmol/L and negative ketones. An arterial blood gas(ABG) revealed a ph- 7.40, pCO2-42mmHg, O2 sat-99%, Total CO2- 27mmol/L, Base excess-1 and PO2-11mmHg on 2 liters oxygen by nasal cannula. On retrospective review of patient's records it was found that her anion gap had been elevated for atleast 1 year. During the entire time her serum bicarbonate levels were within normal range. Serial lactate levels and ABG revealed similar results. An MRI of brain revealed a stable tumor unchanged when compared to prior imaging. The patient was not on any medication that could cause elevated lactate levels. She was worked up for bacterial infections and malaria, but no infection was found.</p> <p><b>Discussion:</b> Lactic acidosis has been reported in patients with highly malignant tumors. This has been described as type B lactic acidosis. It is mainly seen in hematologic malignancies where the cell proliferation is robust, but is rare in solid malignancies. The postulated hypothesis is ischemia of malignant cells causing anaerobic glycolysis. Another phenomenon known as aerobic glycolysis or Warburg effect describes lactate production in the tumor microenvironment as a selective mechanism used by tumor cells to suppress antioncogenes and promote tumor growth factors. However this is unlikely to cause such high lactate levels in the blood. In our patient, a thorough workup did not reveal any obvious cause of high lactate levels for such a long duration with normal bicarbonate levels. In the light of this we suspect that the patient's glioblastoma may be the cause, although there is little literature to support it. Further work needs to be done to study lactate metabolism in glioblastoma and its significance.</p>	<p><b>Author:</b> Victor Kairouz MD <b>Additional Authors:</b> Abdallah Kamouh, MD Susan Graham, MD</p> <p><b>Institution:</b> University at Buffalo (SUNY)</p> <p><b>Title: Type I-like Brugada ECG Pattern Induced by Severe Hyponatremia</b></p> <p><b>Background:</b> Brugada Syndrome is a genetic sodium channelopathy, which predisposes individuals to ventricular tachycardia and sudden death. The disease is characterized by classic EKG findings consisting of down-sloping ST-elevation and an inverted T wave in the right precordial leads. Brugada ECG pattern can be prompted by different medications, such as antiarrhythmic drugs, and many metabolic disorders, namely potassium abnormalities. Only three case studies of Brugada pattern induced by isolated hyponatremia were reported in the literature. Herein, we present a case of isolated hyponatremia secondary to adrenal insufficiency, which elicited a reversible type I-like Brugada ECG pattern.</p> <p><b>Case:</b> 56 year-old Caucasian male with hypertension, dyslipidemia, and Addison's disease presented with few days history of fever and cough. The patient denied any chest pain or tightness, palpitations or shortness of breath. The admitting diagnosis was pneumonia and dehydration. The laboratory data was significant for Na of 117mmol/L and K of 3.7mmol/L. The initial ECG showed type-I like Brugada pattern (Figure 1). After treatment with antibiotics, IVF, and steroids, ECG changes resolved when sodium level reached 135mmol/L. ST-segment returned to the isoelectric level (Figure 2).</p> <p>The patient was not on any medication that could potentially provoke ECG changes, particularly ST segment abnormalities. The remaining of the metabolic panel was within normal range.</p> <p><b>Discussion:</b> A possible explanation of this infrequent manifestation of severe hyponatremia is the reduction of the electrochemical gradient, which decreases the inward sodium current and prolongs the phase 0 of the Purkinje action potential. Consequently, hyponatremia should be included in the expanding list of drugs or conditions known to induce Brugada ECG pattern. It remains uncertain whether ECG Brugada pattern secondary to hyponatremia is associated with increased susceptibility of ventricular tachyarrhythmias.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Diviya Kaul MD <b>Additional Authors:</b> Verma, Sunita MD; Muttana, Rena MD; Janga KC; Greenberg, Sheldon MD. <b>Institution:</b> Maimonides Medical Center</p> <p><b>Title: Atypical Presentation of Proliferative Lupus Nephritis in A Geriatric Patient</b></p> <p>Lupus nephritis typically presents in the second through fifth decade of life. About 12-18 % of patients with lupus are diagnosed after age 50. Patients with late onset lupus typically present with a slow onset and a more insidious course. Acute lupus nephritis is unusual in this group and even less common in the geriatric population above age 80.</p> <p>We report an 82 years old female with a history of Hypertension and Non-Insulin Dependent Diabetes Mellitus, only on one oral hypoglycemic and one anti-hypertensive medication, who was previously in a healthy condition, when she acutely presented with a hypoglycemic episode. Her initial chemistries revealed a creatinine of 4.3 and blood urea nitrogen (BUN) of 83. Seven months earlier on a routine visit to her internist, her creatinine level was 1.0 and BUN level was 12. She had no symptoms suggestive of lupus, and no recent infections. Her urinalysis exhibited proteinuria and microscopic hematuria. A serologic evaluation revealed a significantly elevated ANA, DsDNA, and reduced complement levels (both C3 and C4). Labs also detected a mildly elevated Anti-myeloperoxidase Antibody. A percutaneous kidney biopsy was consistent with diffuse lupus nephritis with cellular crescent formations, specifically, sub endothelial and mesangial deposits. Immunofluorescence studies revealed a characteristic "full house" pattern with a predominance of IgA.</p> <p>There was a low chronicity index.</p> <p>Within a few days, her creatinine increased to 4.5. Her treatment plan included Intra-venous methyl-prednisolone 500 mg daily for three days, followed by oral prednisone and initially, with one dose of Intra-venous cytoxan 500 mg.</p> <p>Two weeks after her initial admission, the patient presented with pneumonia, urinary tract infection, anemia and thrombocytopenia. Her renal function deteriorated and she started on dialysis.</p> <p>An initial diagnosis of acute lupus nephritis in an 82 year old with no history of lupus and no other clinical manifestations of lupus is rare. We report this case to alert the medical community of this unusual presentation, and to create awareness that lupus nephritis is a differential to consider in older populations.</p>	<p><b>Author:</b> Sevak Keshishyan MD <b>Additional Authors:</b> Ravi Manglani , Shekar Murthy , Nehad Shabarek</p> <p><b>Institution:</b> Lincoln Medical and Mental health center</p> <p><b>Title: Pulmonary Cryptococcus presenting as a cavitory lesion as the only manifestation of Cryptococcal infection</b></p> <p>Introduction: Cryptococcal pneumonia is an important cause of pneumonia in immunocompromised patients. Isolated pulmonary involvement in this disease is uncommon (1). Cavitation is even rarer, constituting 10 – 15 % of all cases. We present a case of isolated pulmonary Cryptococcus presenting as a cavitory lesion.</p> <p>Case presentation: A 66 year old male with acquired immunodeficiency syndrome (AIDS) presented to the hospital with productive cough, fever, chills, and dyspnea for 4 days. The patient was tachycardic, tachypneic and febrile with crackles in the right axillary area. Chest X-ray showed infiltrates in right Middle lobe (RML). Chest computed tomography (CT) revealed a cavitory lesion in Right Middle Lobe.</p> <p>Blood cultures were sent and broad spectrum antibiotics were started. Serum cryptococcal antigen was strongly positive (1: 1024). Blood and fungal cultures grew Cryptococcus neoformans. Lumbar puncture was negative for meningitis. CSF cryptococcal antigen titres were also negative. CT guided biopsy of the lesion revealed fungal organisms with morphology consistent with Cryptococcus neoformans. Papanicolaou and grocott stains showed variable sized yeast. Liposomal Amphotericin B and Flucytosine were started. One month later, the patient has responded well to IV antifungal therapy.</p> <p>Discussion: The initial manifestation of the cryptococcal infection is often meningitis, while isolated pulmonary involvement with cavitation is rare (1). A study conducted by Cameron et.al showed no evidence of cavitation in AIDS patients (2). One study conducted by Hu Z. et.al discovered that solitary cavitory pulmonary nodules may be a common CT finding in AIDS associated pulmonary cryptococcosis but these were predominantly asymptomatic (3).</p> <p>Pulmonary cryptococcal disease presents with fever, cough, dyspnea, weight loss and interstitial infiltrates on chest X-ray, and is diagnosed with serum and CSF cryptococcal antigen titers. Cavitory pulmonary cryptococcosis is treated with 2 weeks of intravenous liposomal amphotericin B (5mg/kg/day) with flucytosine (100mg/kg/day) orally,(induction) then, fluconazole 400mg orally for 8 weeks(consolidation) followed by fluconazole 200mg a day orally for a minimum of 12 months antifungal therapy(maintenance).</p> <p>Conclusion: Isolated cavitory pulmonary cryptococcosis is a rare form of cryptococcal infection and should be on the differential in immunocompromised patients. All patients with proven cryptococcal disease should receive a Lumbar Puncture to look for meningeal involvement.</p> <p>References:</p> <p>(1) Chuck SL, Sande MA. Infections with Cryptococcus neoformans in the acquired immunodeficiency syndrome. N Engl J Med 1989; 321:794-99</p> <p>(2) Cameron ML, Bartlett JA, Gallis HA, Waskin HA. Manifestations of pulmonary cryptococcosis in patients with acquired immunodeficiency syndrome. Rev Infect Dis 1991; 13:64-7.</p> <p>(3) Hu, Z, et al. (2013). "Solitary cavitory pulmonary nodule may be a common CT finding in AIDS-Associated pulmonary cryptococcosis." Scand J Infect Dis 45(5): 378-389.</p>
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## Resident / Fellow Clinical Vignette

**Author:** Fausto Lisung

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**Institution:** North Shore LIJ - Lenox Hill Hospital

**Title: A Severe Case of Dengue in NYC, Yes Dengue!**

**Purpose:** To describe a rare and severe case of dengue in NYC and the wide variety of manifestations.

**Methods:** We conducted a detailed and chronological description of a clinical vignette.

**Case presentation:** 20 year-old autistic female, returning from Dominican Republic presented with a three-day history of abdominal pain, fever, nausea and vomiting. On admission, patient was tachycardic, in respiratory distress and had altered mental status. She was immediately intubated. Bedside echocardiogram showed a LVEF of 40% and pericardial effusion with signs of tamponade physiology. Pericardiocentesis was performed and a pericardial drained was placed. She had generalized myalgias and anasarca. Blood work was sent. Elevated AST (2674 U/L) and ALT (481 U/L). Creatine kinase was elevated: 153,390 U/L with large blood in urine and no RBCs. Serum creatinine rose to 2.18 mg/dL and patient had a decreased urine output, consistent with acute kidney injury, rhabdomyolysis and liver damage. Renal ultrasound, lower extremity dopplers and CT-PE protocol were negative. CT chest, abdomen and Pelvis showed bilateral pleural effusion, lower lobe infiltrates and diffuse anasarca. Broad spectrum antibiotics were empirically started due to leukocytosis and fevers

Hospital course was complicated by cardiogenic shock, demonstrated by a pulmonary artery catheter. Nor-epinephrine and fluid resuscitation was started. Troponin peaked at 34.7 ng/mL and her LVEF remained decreased, consistent with myocarditis. Patient became anemic and thrombocytopenic. DIC panel was negative. Infectious, nephrologic, rheumatologic and hematologic studies were sent. All cultures, flu antigens, rapid HIV, HSV, HAV, HBV, HCV, CMV, EBV, RSV, enterovirus, adenovirus, echovirus, parainfluenza, leptospira, Quantiferon-TB, RF, ANA, Anti dsDNA, Anti-Jo, Anti mitochondrial, factor VII essay, lupus anticoagulant and SPEP were negative. Dengue virus IgM was elevated (5.77) and IgG within normal range (0.73). One week later immunoglobulin sero-conversion was seen, IgG rose to 3.46, leading to the diagnosis of dengue. Patient was treated with supportive measures. Eventually, pressors were discontinued, extubated, transitioned to room air, off antibiotics and physical therapy for myositis. Patient gradually improved and was discharged home after 20 days of hospitalization.

**Discussion:** This case shows a picture of severe dengue (WHO classification), where shock and multi-organ dysfunction were present. Acute liver failure, pericardial effusion, and rhabdomyolysis with acute renal failure are atypical presentations and rarely reported clinical manifestations of dengue. Symptoms resolved with supportive treatment. With increased travel and new reported indigenous cases in the US, dengue should be a familiar disease entity. It is important to review the various clinical presentations of dengue in its great complexity and variety and include it in the differential diagnoses for travelers presenting with symptoms of unknown etiology. Early recognition allows for supportive treatment, proper and oriented care which is necessary to avoid fatal complications.

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**Title: A CASE OF HYPERAMMONEMIC ENCEPHALOPATHY SECONDARY TO VALPROATE TOXICITY**

**Introduction:**

Valproic acid (VPA) indirectly increases the amount of gamma-aminobutyric acid (GABA) available to the central nervous system (CNS). It also alters fatty-acid metabolism, impairs mitochondrial beta-oxidation, and disrupts urea cycle that leads to hyperammonemia. We present here our experience of managing a rare case of hyperammonemic encephalopathy from valproate overdose.

**Case Presentation:**

A 38-year-old male with bipolar disorder and currently going through marital problems was found on the floor of his father's basement unresponsive, with an empty bottle of VPA next to him. It was filled 1 week ago with 60 tablets. On admission, vitals were only notable for oral temperature of 36.1 degree Celsius. He was unconscious, minimally responsive to noxious stimuli and with pinpoint pupils. Cardio-pulmonary, abdominal and skin exams were normal. Labs were notable for mixed high anion gap metabolic acidosis and respiratory acidosis. Urine toxicology screen was positive for cocaine, ETOH level was 0.03, and acetaminophen and salicylate levels were negative. Valproate level on admission was 1463 mg/L (normal: 50-100 mg/L) and serum ammonia was 263 mcg/dL (normal: 28-80 mcg/dL).

Chest X-ray and Non-contrast CT head were normal. He was intubated in the intensive care unit (ICU) for airway protection, and received levocarnitine therapy and emergency hemodialysis the same day as recommended by Poison Control. Post-dialysis, VPA level came down to 250 mg/L and then 125 mg/L on day 2. Ammonia level also normalized. Patient was extubated on day 3 and discharged to inpatient psychiatry after 5 days of ICU stay.

**Discussion:**

Severe VPA poisoning may present with hypothermia, refractory hypotension, confusion, lethargy, hallucinations and coma, along with dose dependent respiratory depression that may require mechanical ventilation. Hyperammonemic encephalopathy is an unusual complication and results from inhibition of carbamoylphosphate synthetase-I that begins the urea cycle. Hyperammonemia leads to increased brain glutamine level that causes astrocyte swelling and cerebral edema. Management is mainly supportive. Decontamination and elimination may be required. Hemodialysis decreases VPA levels and should be initiated promptly when levels exceed 850-1000 mg/L. Long-term use of VPA is associated with serum carnitine depletion, which leads to hyperammonemia. Carnitine also plays a direct role in metabolism and elimination of VPA. Levocarnitine supplementation is believed to provide benefit in VPA toxicity, particularly with concomitant hyperammonemia, encephalopathy, or hepatotoxicity. It is best administered in consultation with a poison control center for dosing recommendations. More experience is needed before levocarnitine use for valproate toxicity becomes a standard of care. This case is unique; with remarkably high levels of acute VPA toxicity that responded very well to our therapy of intensive supportive care, emergent hemodialysis and levocarnitine therapy, and will add to the knowledge to other physicians facing similar presentation.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Saurav Luthra MD <b>Additional Authors:</b> Vasvi Singh MD; Disha Mehta MD; Carlos Palacio MD Unity Health System, affiliated to University of Rochester, Rochester, NY <b>Institution:</b> Unity Health System <b>Title: THE POISONOUS PUMP THAT FROZE A BRAIN, LITERALLY!</b></p> <p><b>Introduction:</b> Baclofen, a centrally acting skeletal muscle relaxant is primarily used to treat severe spastic conditions. It is available as oral and intrathecal formulations. Baclofen overdose commonly occurs with intentional ingestions or intrathecal-pump malfunctions. Latter has been reported only as case reports but its presentation mimicking sepsis is not documented.</p> <p><b>Case Presentation:</b> A 39 year-old-woman on intrathecal baclofen pump for advanced Multiple Sclerosis (MS) with an indwelling suprapubic catheter for neurogenic bladder, colostomy for severe constipation, presented with acute encephalopathy-confused and drowsy. Vital signs were stable except for an oral temperature of 34.8. She was lethargic with reactive and dilated pupils, normal cardio-pulmonary and abdominal exam. Colostomy and suprapubic catheter sites did not seem infected. Flaccid extremities at baseline. Labs including a complete blood count with differential, comprehensive metabolic panel, lactic acid, arterial blood gas and urine toxicology screen were normal. Urinalysis suggested an infection. Chest X-ray was normal and CT scan of head showed stable MS lesions. While still in Emergency Department, she became hypotensive despite adequate fluid resuscitation and got transferred to Intensive Care Unit with the impression of urosepsis. Broad spectrum antibiotics with high dose steroids (possible MS flare) were initiated along with vasopressors. Despite aggressive therapy for 24 hours, she remained hypothermic, hypotensive and subsequently bradycardic between 40-50 bpm. Additional history after spouse arrival disclosed a 20% increase in her baclofen pump dose 2 days prior to admission. This raised a suspicion of baclofen toxicity. Reviewing her case, an MS flare was unlikely given advanced disease, as was sepsis because she did not improve with adequate therapy. On day 3, heart rate (HR) dropped to 30bpm with brief pauses. Baclofen dose was decreased by 20% and within minutes HR was in 70s. With further 50% dose reduction, acute encephalopathy started to resolve. Her serum baclofen level was negative as is expected in patients with intrathecal-pumps. Antibiotics and steroids were stopped. Daily Creatine Kinase monitoring for baclofen withdrawal was done. Patient was discharged at her baseline.</p> <p><b>Discussion:</b> Severe baclofen toxicity may present with bradycardia, hypotension, respiratory failure, hypothermia, seizures, coma and death. Rarely, status epilepticus, rhabdomyolysis, and cardiac conduction abnormalities occur. Management is mainly supportive and symptomatic; activated charcoal in acute ingestions and emptying pump reservoirs following intrathecal overdose. Urine toxicology does not screen baclofen and serum concentrations are not readily available. Treatment is initiated based on clinical suspicion. This is a rare case of iatrogenic baclofen overdose presenting as acute encephalopathy with refractory hypotension, hypothermia and severe bradycardia. It reinforces the significance of educating patients/families regarding adverse effects after baclofen dose adjustments in order to prevent life-threatening complications. Also, physicians need to maintain a high index of suspicion of toxicity in patients having intrathecal baclofen pumps.</p>	<p><b>Author:</b> Ravi Manghani M.D. (PGY - 1) <b>Additional Authors:</b> Dr Adriana Mililo, Dr Nehad Shabarek</p> <p><b>Institution:</b> Lincoln Medical and Mental health center</p> <p><b>Title: A case of quadruple Multiple Primary Malignant Neoplasms</b></p> <p><b>Introduction</b> Multiple Primary Malignant Neoplasms (MPMN) in a single patient is rare, although the incidence of this is increasing in recent times. The median age of diagnosis of the index cancer is 53.7 years (+/- 12 years). We present the case of a patient whose index cancer was diagnosed at age 72 and subsequently developed 3 more cancers within 2 years.</p> <p><b>Case Report</b> A 72 year old male was diagnosed with a left lobe prostate nodule with elevated PSA. Biopsy revealed Prostate Adenocarcinoma with Gleason score of (4+3). CT scan of the abdomen done two years later revealed a lesion located peripherally in segment 5 of his liver. In addition patient was found to have worsening anemia and reported rectal bleed. Further workup revealed colon adenocarcinoma. Abdominal CT scan with liver protocol revealed two distinct liver lesions; one located peripherally, which was consistent with hepatocellular Carcinoma and another in the common bile duct. Biopsy of the latter was done and immunohistochemistry of this lesion favored cholangiocarcinoma.</p> <p>Thus, this unfortunate patient was diagnosed with four separate malignancies, at a late stage in his life. More-over, the patient was diagnosed with 3 gastro-intestinal malignancies at the same time.</p> <p><b>DISCUSSION</b> The three criteria to diagnose multiple primary malignancies are 1) each tumor should show specific malignant findings 2) the tumors should defer in their site of origin 3) One tumor must not be a metastasis from another site. The tumors are called synchronous if they arise within six months of each other. Two separate reviews concluded that the common features in these cases are the presence of prostate cancer, colon carcinoma, the age of onset of most patients being in the fifth or sixth decade and most cancers are diagnosed at a locally advanced stage, without a tendency to metastasize.</p> <p>In a literature review of over 1 million cancer patients, the prevalence of MPMN was found to vary from 0.734% to 11.7%, and incidence increases with age. This occurs much more frequently that can be explained by sheer chance. Patients who develop one cancer are at an increased risk of developing new primaries by about 10% above the general population. In addition, patients developing more than 3 primaries have a strong family history of cancers in first degree relatives. Thus, patients with 2 or more cancers must be followed for development of subsequent cancers. Family screening is also warranted, as there is usually a strong family history of MPMN.</p> <p><b>Conclusions</b> Incidence of one cancer predisposes the patient to other cancers. Family members of these patients may be screened and referral to genetic counselling may be beneficial.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Kaushal Parikh <b>Additional Authors:</b> Tanush Gupta, Nivas Balasubramaniam, Neha Paul, Pallak Agarwal Sachin Sule <b>Institution:</b> Westchester Medical Center</p> <p><b>Title: A CASE OF LEFT INTERNAL MAMMARY ARTERY AVULSION FOLLOWING CARDIOPULMONARY RESUSCITATION CAUSING HEMOTHORAX AND CARDIAC TAMPONADE</b></p> <p>Background: Effective chest compressions remain the cornerstone of successful cardiopulmonary resuscitation (CPR). We describe a case of left internal mammary artery avulsion after standard manual CPR causing cardiac tamponade and hemothorax, which was successfully managed with a thoracotomy, pericardial window and arterial ligation. Case: A 74-year-old male presented to an outside hospital with acute onset of shortness of breath. An electrocardiogram showed sinus tachycardia at a rate of 136 beats per minute and an age indeterminate inferior and anteroseptal infarct. He had elevated troponin I (0.93 ng/mL) and was diagnosed with a non-ST elevation myocardial infarction. His past medical history included coronary artery disease, chronic obstructive lung disease, and ischemic stroke with residual left sided hemiparesis. He was taken for a left heart catheterization that showed a 80% lesion in the right posterior left ventricular artery, which was thought to be the culprit lesion and was successfully stented with a bare metal stent. Subsequently, in the recovery room, he went into cardiac arrest and was found to have pulseless electrical activity (PEA). He was successfully resuscitated out of the PEA arrest. A bedside two-dimensional (2-D) echocardiogram showed a large pericardial effusion causing cardiac tamponade and mediastinal widening; and the patient was then transferred to our hospital for an emergent pericardial window placement. A left anterior mini-thoracotomy was performed; approximately 500 cc of bloody fluid was aspirated from the pericardial space and a pericardial window was created. Also, he was found to have left hemothorax that was drained and a chest tube was inserted. No obvious source of bleeding could be identified at this time. Subsequently, in the cardiothoracic intensive care unit (CT-ICU), he continued to be hypotensive requiring multiple vasopressors. A 2-D echocardiogram showed an echolucent mass anterior to the right ventricle causing diastolic collapse. He was taken back to the operating room and underwent an emergent median sternotomy for exploration of the mediastinal and pericardial cavities. He was found to have an avulsion of the distal left internal mammary artery with an adjacent costochondral fracture. The artery was mobilized and successfully ligated. There was no further bleeding identified and the patient was transferred back to the CT-ICU. He made good postoperative recovery and was eventually discharged to a skilled nursing facility. Conclusion: There have been reports of avulsion and rupture of internal mammary artery after minimally invasive direct coronary artery bypass (MIDCAB) procedures, spontaneous ruptures in patients with vasculitis or genetic syndromes like Marfan's syndrome; however, traumatic internal mammary artery avulsion after CPR has not been previously reported. Injuries to the thoracic wall, pulmonary and cardiovascular systems in the setting of manual chest compressions may cause significant morbidity and mortality and represent potentially reversible causes of resuscitation failure.</p>	<p><b>Author:</b> Tarak Rambhatla MD <b>Additional Authors:</b> Eric Berkowitz, MD; Tarak Rambhatla MD; Jason George, Andrew Miller MD, Georgia Panagopoulos, PhD, MD, Bushra Mina, MD</p> <p><b>Institution:</b> Lenox Hill Hospital, Department of Medicine <b>Title: Triage of patients that present to the emergency department with severe sepsis: do they all have to go to the ICU?</b></p> <p>INTRODUCTION Severe sepsis affects millions of individuals each year, with a high mortality rate and financial burden. Severe sepsis represents 10% of all intensive care unit (ICU) admissions and has become a major issue within healthcare as the incidence continues to increase. There is limited data however, regarding the triage of patients admitted from the emergency department (ED) with severe sepsis.</p> <p>OBJECTIVES Our aim was to compare outcomes of individuals presenting through the ED with severe sepsis triaged to different levels of care. There appears to be a subset of individuals presenting with severe sepsis that respond well to early resuscitative measures. If we can identify specific factors that contribute to this response, it could aid in the easy and safe triage of individuals with severe sepsis.</p> <p>METHODS A retrospective chart review of patients presenting to the ED with severe sepsis and triaged by an intensivist was conducted covering an 18-month period. Inclusion criteria included an initial systolic blood pressure less than 90 mm Hg or elevated lactate (&gt;2 mg/dl) and exclusion criteria included vasopressor/inotropic or invasive ventilatory support. Data collected was initial lactate level, repeat lactate, blood pressure response to initial resuscitation, triage location, age, length of stay, mortality, DNR status, site of infection, urine analysis, chest xray, urine culture, blood culture and if applicable sputum culture results.</p> <p>RESULTS Patients were divided into two groups: appropriate triage to the ICU versus appropriate triage to a non-ICU setting, based on no escalation or de-escalation of care within 24 hours of triage. Statistical analysis was performed with chi-squared and/or t-test on each variable to determine what factors were significant between the ICU and non-ICU cohorts. A total of 167 subjects met our initial triage criteria, and 139 subjects were correctly triaged not requiring a change in level of care within 24 hours; 27 subjects were in the ICU group and 112 subjects were in the non-ICU group. The ICU group when compared to non-ICU-group, had statistically significant associations with the presence of initial hypotension upon presentation to the ED (48% vs 27%, p=0.023), presence of clear CXR (25% vs 50%, p = 0.04), presence of unilateral infiltrate on CXR (35 vs 27%, p =0.038), presence of bilateral infiltrate on CXR (39% vs 22%, p = 0.04), and presence of having a negative (non infected) urine analysis (74% vs 52%, p=0.03).</p> <p>CONCLUSIONS Patients presenting to our ED with severe sepsis who were correctly triaged to the ICU were more likely to present with hypotension or to have pneumonia as the source of infection. Patients presenting with severe sepsis who were correctly triaged to a non-ICU setting were more likely to present normotensive or to have UTI as the source of their sepsis; this group was also less likely to have pneumonia.</p>
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## Resident / Fellow Clinical Vignette

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**Institution:** Flushing Hospital Medical Center

### **Title: Lyme Transverse Myelitis; Not as rare as you would think.**

#### Introduction

Lyme Disease is a systemic illness caused by *Borrelia burgdorferi*; transmitted via bite from an infected Ixodes Tick. Neuroborreliosis occurs when disease becomes disseminated resulting in meningeal seeding. Neurological manifestations occur in 15% of cases, include meningitis, facial nerve palsy, radiculopathy, diffuse polyneuropathy and rarely transverse myelitis. We present a case of a 30 year old female with Lyme associated transverse myelitis.

#### Case History

30 year old female presented to ER for bilateral upper extremities and abdominal numbness. Symptoms started two days ago when she felt numbness and weakness in her right hand. Two days later she woke up from sleep due to pain in her left arm, which then became numb and weak. Patient denied lower extremity numbness, however, her left leg felt weak. She denied visual changes, dysarthria, dysphagia, bladder or bowel incontinence, recent flu like illness, vaccination, recent travel or tick bite. Examination shows moderate lower extremity spasticity, ? power in distal upper extremities with decreased reflexes. No weakness in lower extremities noted. Sensory exam to light touch, pinprick and vibration was normal. Deep tendon reflexes were 2+ diffusely with Left ankle clonus. Plantars were down going. CT head and MRI brain failed to reveal demyelinating plaques. MRI cervical spine showed patchy abnormal increased T2 signal within anterior aspect of cervical cord; C4 through C7 level, favoring demyelinating and/or inflammatory etiology. CSF showed normal glucose, protein, cell count, negative oligoclonal bands and no elevated Ig G index. Patient was diagnosed with acute transverse myelitis and showed improvement with high dose intravenous solumedrol. Further workup revealed elevated serum Lyme antibodies and positive western blot IgM. Patient was treated with intravenous Ceftriaxone. Two months later, patient reported minimal residual numbness in arms with normal muscle strength.

#### Discussion

Transverse myelitis is an acute-sub acute acquired inflammatory disease of the spinal medulla presenting with weakness, sensory changes and bowel or bladder dysfunction. It may occur independently or associated with certain infections, vaccinations, connective tissue or demyelinating disorders. Evaluation of transverse myelitis includes gadolinium enhanced MRI of the spine and CSF analysis. Measurement of serum NMO-IgG antibodies B12, methylmalonic acid, HIV and Syphilis serology, ANA, Ro/SSA, La/SSB antibodies, and thyroid stimulating hormone are an integral part of the work up. Transverse myelitis may occur in 4 to 5 percent of all cases of neuroborreliosis. Disease can be diagnosed by detecting antibodies to *B. burgdorferi* in serum and CSF. Negative CSF antibodies results do not exclude Lyme disease. Recent studies have shown a promising role for CSF CXCL13 in diagnosis of neuroborreliosis. Lyme's Disease should be considered as part of the differential for the etiology of transverse myelitis even if the patient declined history of rash or tick bites.

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### **Title: AN UNWELCOMED NAP: PRAMIPEXOLE INDUCED NARCOLEPSY**

#### Introduction:

Sleep disorders have been reported as side effects of common medications. Excessive daytime sleepiness and insomnia are the most common of these adverse effects. It is of significant clinical importance to recognize and treat these secondary sleep disorders in a timely fashion. Increased daytime somnolence has long been recognized as a side effect of dopaminergic drugs which are used in the treatment of Parkinson's disease, although there are no descriptions of sudden irresistible sleep attacks in clinical trials like we noticed in our patient.

#### Case Presentation:

A 66 year old Caucasian male with past medical history significant for Parkinson's disease, coronary artery disease and ischemic cardiomyopathy presented with several weeks history of excessive daytime sleepiness, sudden episodes of falling asleep two to three times a day finally culminating in an alarming episode at a restaurant where he suddenly became unresponsive while having his meal. Patient's wife was unable to wake him up with verbal stimuli. He was woken up in ambulance by paramedics and was described as being completely lucid.

Patient was brought to the hospital and extensive work up was done in the emergency room that was inconclusive. Upon admission, his list of medications was reviewed. It included pramipexole which is known to induce sleep related disorders including narcolepsy. The medication was discontinued and patient was referred for a sleep study.

#### Discussion

Pramipexole is a non-ergot dopamine agonist used to treat Parkinson's disease. Patients on dopamine agonists are known to have reported relatively continuous drowsiness that leads to falling asleep without acute warning during periods of inactivity, defined as "sudden irresistible sleep attacks". In a study evaluating pramipexole with regards to driving safety concerns, 17 out of 29 patients reported that the sleep events happened during driving, leading to road crashes in 10. Sleep latency normalized after discontinuation of pramipexole. Somnolence usually resolves with pramipexole dose reduction or discontinuation. Some case reports demonstrated remission after an add therapy with modafinil and amantadine.

Patients who are experiencing generalized drowsiness and falling asleep during periods of inactivity be instructed not to drive because these patients do fall asleep without acute warning.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Ambreen Raza MBBS <b>Additional Authors:</b> SRIKRISHNA VARUN MALAYALA, MBBS, MPH; MUNIR AHMED KHAN, MBBS; MOHAMMAD UMER BUTT, MBBS <b>Institution:</b> University at Buffalo/Catholic Health System-Sisters of Charity</p> <p><b>Title: NOT ALL TROPONIN ELEVATION IS A MYOCARDIAL INFARCTION</b></p> <p>Introduction: The 2012 Joint European society of Cardiology/American College of Cardiology/American Heart Association/World Health Federation Task Force emphasized the importance of “BOTH” elevated cardiac biomarkers and clinical evidence of myocardial necrosis in a clinical setting consistent with myocardial ischemia for the diagnosis of Myocardial Infarction. However, in today’s clinical practice, diagnosis of MI has increasingly become dependent upon evaluation of cardiac biomarkers, particularly cardiac troponins. We present two cases where elevation in troponin rather than history and physical examination guided the care of patients presenting with chest pain. This inevitably led to excessive work up, re admissions and higher cost of care for both patients.</p> <p>Case 1: A 54 year-old-female with presented with 6-8/10 intensity sub sternal, pressure like chest pain radiating to her right shoulder accompanied by diaphoresis, shortness of breath, lightheadedness, headache, nausea and vomiting. Leaning forward relieved it. Significant elevation in cardiac biomarkers prompted treatment for NSTEMI. Subsequently she underwent an angiogram, which failed to demonstrate any coronary disease. She was discharged home only to return two days later with similar picture. Notably, after a detailed and careful history taking, patient had history of flu like illness a week earlier. A diagnosis of peri-myocarditis was then made, ibuprofen and colchicine were started and the symptoms resolved. Any further unnecessary work up was avoided.</p> <p>Case 2: A 31-year-old morbidly obese white male presented with 8/10 intensity, stabbing sub-sternal non-radiating chest pain for 5 days without any associated symptoms. Patient reported some relief of pain with IV NSAIDs in the emergency room. However, given that the patient had significant elevation in cardiac biomarkers, treatment for NSTEMI with full anticoagulation was initiated. Patient underwent an angiogram, which demonstrated normal coronary arteries. Incidentally, patient returned a couple of days later with similar presentation. He was then started on high dose ibuprofen, which relieved the pain. This was consistent with his earlier presentation to ER where his pain was relieved with NSAIDs and not with low dose aspirin or sublingual nitroglycerin.</p> <p>Discussion: Apical ballooning syndrome, massive pulmonary embolism, peripartum cardiomyopathy, renal failure, severe acute neurological diseases, stroke, infiltrative diseases, extreme exertion, sepsis, acute respiratory failure, frequent defibrillator shocks, severe burns, rhabdomyolysis, aortic dissection, tachyarrhythmias, severe bradyarrhythmias are few of the many causes that could present with chest pain and elevated troponin level. Among patients with a high-test probability of acute thrombotic coronary heart disease, the value of troponin for diagnosis is clear. However in patients with a low-test probability of MI, troponin elevation is not specific and may divert attention from the true underlying clinical problem and lead to unnecessary invasive interventions.</p>	<p><b>Author:</b> Sowmya Reddy PGY 1 <b>Additional Authors:</b></p> <p><b>Institution:</b> Montefiore Medical Center</p> <p><b>Title: HYPOTENSION IS OF VARIED ETIOLOGY WARRANTING A DIFFERENTIAL DIAGNOSIS: CASE OF CARDIAC TAMPONADE PRESENTING AS HYPOTENSION IN SETTING OF SEPSIS</b></p> <p>Introduction: Hypotension is of multifactorial etiologies and is common in hospitalized patients. While causes such as volume depletion, bleeding, sepsis and cardiac cause are always entertained, in a given case, the diagnosis may be different. This case, where the etiology of hypotension was cardiac tamponade, but not entertained at the outset, illustrates the importance of a wide differential diagnosis.</p> <p>Case: 65 year old nursing home female with history of hypertension, transient ischemic attack and chronic obstructive pulmonary disease presented with hypotension of 95/66 mm Hg, pulse rate of 120/m, and 102.9 F. Heart sounds were muffled; the jugular venous pressure (JVP) was elevated. EKG revealed pulsus alternans. Echocardiogram confirmed massive pericardial effusion with collapse of the right ventricle. A CT scan performed for the abdominal abscess suggested a large pericardial effusion. She also had an abdominal wall abscess at the site of a dislodged PEG tube, requiring incision and drainage of the abscess. She underwent closed pericardiocentesis and 600 ml of clear pericardial fluid was drained. Fluid cultures and cytological analysis were negative. She was monitored for hemodynamic derangements with serial echocardiograms for re-accumulation of pericardial effusion. She symptomatically improved. The hypotension resolved and she was discharged on usual medications.</p> <p>Discussion The causes of pericardial effusion are varied and include infectious, radiation, post myocardial infarction, drug induced, metabolic (uremia), and malignancy, besides being idiopathic. Hypotension can be multifactorial in origin with rare causes including Addison’s disease and cardiac tamponade. A thorough history and physical exam are essential to discern the likely cause. Our patient had clinical features and electrocardiographic evidence and met the components of the Beck’s triad including hypotension, muffled heart sounds and elevated JVP. In addition the CT scan performed for an abdominal abscess indirectly suggested the diagnosis of pericardial effusion. In patients admitted to the hospital, physicians tend to always consider the common causes (volume loss, bleeding, myocardial infarction and sepsis) but seldom think of rare causes such as cardiac tamponade. The diagnosis was not entertained until it was suggested by the radiologist from an abdominal CT scan.</p> <p>Key points • Hypotension is a medical emergency and requires a quick assessment of the etiology to offer prompt corrective treatment. • While cardiac tamponade is rare, correct diagnosis invariably leads to fluid drainage with huge potential for a favorable outcome, as in this case. • The cause of pericardial effusion may not be readily apparent.</p> <p>References Cardiac tamponade in hypothyroidism, BMJ case report 2014; doi: 10.1136/bcr-2014-204076</p>
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## Resident / Fellow Clinical Vignette

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**Institution:** Maimonides Medical Center

**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.readmissioncore.org/heart\\_failure.php](http://www.readmissioncore.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 (&#177; 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 (&#177; 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.

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**Title: Unusual presentation of primary hyperparathyroidism as acute hypercalcemia**

**Introduction:**

Hypercalcemia can be a manifestation of a serious illness such as malignancy or can be detected coincidentally in a patient with no obvious illness[1]. Mild hypercalcemia(11-11.5mg/dL) is usually asymptomatic and recognized only on routine calcium measurements. More severe hypercalcemia(>12-13mg/dL), particularly if it develops acutely, may result in lethargy, stupor, or coma[2]. Most patients with hypercalcemia have primary hyperparathyroidism, in which hypercalcemia is typically chronic and mild. Acute, severe hypercalcemia is unusual[3]. Rarely, hyperparathyroidism develops or worsens abruptly and causes severe complications such as marked dehydration and coma, so-called hypercalcemic parathyroid crisis[4]. Here we describe a case of a hyperacute symptomatic hypercalcemia due to primary hyperparathyroidism.

**Case description:**

An 82 year old lady with medical history significant for Hypertension, CVA, prophylactic bilateral mastectomy, was brought to ED because of weakness and altered mental status for 4-5days. Home medications include: Norvasc, lisinopril, Aspirin, Folic acid, Thiamine, loproressor. Physical examination was unremarkable except for decreased motor strength of extremities symmetrically. Laboratory findings were unremarkable except for Calcium 12.3, Magnesium 1.3. EKG and chest X ray were unremarkable. In Brain CT there was no acute event and unchanged compared to previous MRI.

The patient was admitted for acute hypercalcemia and was started on IVF. Calcium improved over several days to 9.3. Further evaluation revealed: PTH42.3, normal Vit D, SPEP and Free T4 level, elevated TSH and negative PTHrp.

Because of elevated calcium and high-normal PTH level, primary hyperparathyroidism was considered. On review of the record from the previous admissions including last admission on two months earlier, patient's baseline calcium level was 9.2. We then concluded that this episode of hypercalcemia is hyperacute. Given that it is unusual for primary hyperparathyroidism to present as acute hypercalcemia (usual presentation is chronic hypercalcemia), further diagnostic tests were done to rule out malignancy. CT scans of the chest, abdomen and pelvis were unremarkable. On third day, repeat PTH was elevated to 90.2 and same time calcium was 10.4, so diagnosis of primary hyperparathyroidism was confirmed.

**Discussion:**

Most patients with hypercalcemia have primary hyperparathyroidism, in which hypercalcemia is typically chronic and mild. Acute, severe hypercalcemia is unusual[3]. Rarely, hyperparathyroidism develops abruptly and causes hypercalcemic parathyroid crisis[4]. As in the case presented above, primary hyperparathyroidism may rarely present with hyperacute hypercalcemia. In such a patient, other causes must be excluded and treatment to lower calcium must be rapidly administered.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Dipankar Roy PGY-2  <b>Additional Authors:</b> M. Choudhury MD  M. Kanagala MD  T. S. Dharmarajan MD, MACP  <b>Institution:</b> Montefiore Medical Center, Wakefield Division  <b>Title: Planning for Surgery and Assessment of Risk in Severe Hypothyroidism</b>  <b>Purpose:</b>  Severe hypothyroidism is associated with significant perioperative morbidity and mortality, warranting planning before surgery. Presented is such a case.  <b>Case:</b>  63 year old community female with multiple comorbidities (diabetes mellitus, hypertension, chronic kidney disease Stage-V) was hospitalized for generalized fatigue and itching. She complained of dry skin, constipation, cold intolerance and muscle cramps. She had not seen a physician for a year and was on no medications. Examination confirmed a female looking older than stipulated age, lethargic, pale, flat affect, puffy face and dry excoriated skin. Thyroid was barely palpable; tendon reflexes confirmed delayed relaxation phase. Labs: BUN 83 mg/dl, creatinine 8.5mg/dl, TSH-298 uU/ml (N: .40-4.6), FT4 0.1ng/dl (N 0.8-1.7), anti TPO Negative, LDL cholesterol 339mg/dl, vitamin D &lt;4.0ng/ml (N: 30-100). The diagnosis of ESRD needed consideration for renal replacement therapy access creation. Severe hypothyroidism required optimization to lower perioperative risk.  <b>Discussion:</b>  Thyroid hormones have pleiotropic actions involving most every organ systems. Hypothyroidism impairs myocardial function, hemostasis, pulmonary ventilation and metabolism of drugs including anesthesia. A euthyroid state with adequate thyroid hormones is necessary for optimum outcomes following surgery. Inadequate cardiovascular responses to acute increase in intrathoracic pressures related to hypothyroidism-induced baroreceptor defect and depressed adrenergic tone lead to hypotension and predispose to myocardial infarction or cerebrovascular accidents in the perioperative period. In severe hypothyroidism, both hypoxic and hypercapnic ventilatory drives are depressed, with no increase in minute ventilation even at low alveolar oxygen tension. Muscle weakness and obesity may result in atelectasis, reduced lung volumes and reduced exercise capacity. Hypothyroid patients have slowing of drug metabolism and elimination; additionally when renal function is impaired (as in our case) there results accumulation of sedatives and anesthetics, with resultant respiratory function and risk for respiratory failure. Worsening renal function is intensified by hypothyroidism. Though data suggests that mild to moderate hypothyroidism is not associated with significant differences in outcome of surgery, severe hypothyroidism warrants optimization of thyroid function before proceeding with surgery.  <b>Lessons Learnt:</b>  -Though hypothyroidism is common, the diagnosis is often missed; a thorough history and physical examination usually provide clues.  -Severe hypothyroidism affects most organ or system functions, especially cardiac, pulmonary and renal  -Optimization of thyroid function is vital in preparing a patient for surgery to ensure the best possible outcomes.  <b>References:</b>  -Weinberg A et al. Outcome of anesthesia and surgery in hypothyroid patients. Arch Intern Med 1983;143:893-897  -Stathatos N et al. Perioperative management of patients with hypothyroidism. Endocrin Metab Clin N Am 2003; 32:503-518</p>	<p><b>Author:</b> Dipankar Roy PGY-2  <b>Additional Authors:</b> N. Thota MD(Associate)  T. S. Dharmarajan MD, MACP  <b>Institution:</b> Montefiore Medical Center, Wakefield Division  <b>Title: Unusual Presentation of Myocardial Infarction in the Elderly: An Established Fact, Often Forgotten</b>  <b>Purpose:</b>  Unusual presentation of myocardial infarction (MI) in older adults is well recognized and deserves consideration before hemodynamic instability sets in while patients are for concomitant illness.  <b>Case:</b>  76 year old Nursing Home female resident, with multiple comorbidities including systemic lupus erythematosus on chronic steroids, diabetes mellitus and peripheral vascular diseases was hospitalized with lethargy and altered mentation. Prior to hospitalization, she was treated with antibiotics for possible sepsis. Initial vitals confirmed systolic blood pressure of 95-100 mm. On day 2, she remained confused and could not verbalize; she was obtunded, her BP was 70/45 mmHg, heart rate 130-140/min and remained unstable despite adequate fluid resuscitation, broad spectrum antibiotics and stress dose of steroid. Repeat EKG confirmed ST depression and T changes in anterior chest leads that progressed to Q waves in v1 and v2 on repeat EKG.  Labs revealed troponin 0.10 and 0.24 ng/ml. Urgent bed side echo confirmed new antero-septal, apical and antero lateral akinesis with estimated EF of ~25%. The EF and LV wall motion were normal a month earlier. The patient was transferred to intensive care unit immediately to manage cardiogenic shock.  <b>Discussion:</b>  Coronary artery disease is most common in the old, but classic signs and symptoms of MI may be subtle or absent. Presenting symptoms are likely atypical and differ from the classical substernal pressure or pain with exertion. In the Worcester Heart Attack Study, chest pain was reported in 63% of the overall population, but noted in less than 50% women &gt;75 years. Symptoms are primarily dyspnea, syncope, shoulder/ back pain, weakness, fatigue, acute confusion and epicardial discomfort (precipitant: concurrent illnesses). Atypical presentations in the old divert diagnostic suspicion from the acute ischemic event. A diagnosis of "other" was often recorded at admission in older adults (5% of those &lt;65 versus 24% of =85 years). As presentation is often vague, a diagnosis of MI is not entertained or diagnosed late contributing to higher mortality. In the GUSTO-I trial, the 30-day mortality rate increased 10-fold (3.0% &lt;65 years versus 30.3% =85 years).  <b>Lessons:</b>  It is critical to consider a diagnosis of acute MI in the elderly when hemodynamic status is compromised.  As presentation may be atypical, serial ECGs and cardiac markers are vital aids to enable early diagnosis and prompt management.</p>
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## Resident / Fellow Clinical Vignette

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**Institution:** ST.JOHN'S EPISCOPAL HOSPITAL SOUTH SHORE

### **Title: Massive Upper Gastrointestinal Bleed in Patient with Gastrostomy Tube**

#### Introduction.

Upper gastrointestinal bleeding due to ulceration of the gastric wall owing to mechanical irritation of the opposite wall by a long standing gastrostomy tubes is uncommon. It was identified to be 1.6% of massive UGIB in 264 adult with gastrostomy tubes.

#### Case Presentation

A 58 yo male, SNF resident with a history of a gastrostomy tube, TBI, DM type II, CKD stage 2, seizure disorder presented with a few episodes of coffee ground emesis associated with melena. The patient was on 81mg of ASA.&nbsp;Last Hb was 11.2 g/dL 6 days prior to admission.

Physical exam was remarkable for stable vital signs, pale conjunctiva and mucous membranes. There was a gastrostomy tube in situ. Rectal exam revealed black stool on a glove.

Laboratory values: WBC 16.5, RBC 1.45, Hb 4.6 g/dL, Hct 13.3%, MCV 92fL, RDW 13.7%, Plt 206K/mL with positive FOBT, Na 130mEq/L, K 4.7mEq/L, Cl 98mEq/L, CO2 28mEq/L, BUN 139 mg/dL, creatinin 2.92mg/dL.

#### Hospital Course.

The pt was managed medically. The gastrostomy tube aspirate showed normal gastric content. Vigorous hydration with NS,and 4 units of PRBC,and intravenous PPI were&nbsp;administered. The upper endoscopy revealed that&nbsp;the tip of the gastrostomy tube was rubbing against the base of the ulcer on the opposite wall with the stigmata of recent hemorrhage. The gastric biopsy was negative for H. pylori. The gastrostomy tube was substituted for the PEG.

The patient&nbsp;improved, AKI resolved, Hb/Hct were stable upon discharge.

#### Discussion.

There are&nbsp;many reports regarding the early complications&nbsp;pertaining to tube feeding via a PEG or gastrostomy tube. However, the complication of a long standing gastrostomy tube such as massive upper gastrointestinal bleeding owing to contralateral mucosal abrasion by a gastrostomy tube was reported sporadically in literature. A&nbsp;study showed that 9 of the 92 patients developed the posterior wall gastric ulcers attributed the tip of the gastrostomy tube that came to the contact with the opposite wall of the gastric mucosa. Among the nine patients with the gastrostomy tubes related ulcers three had signs of gastrointestinal hemorrhage. It is believed that constant pressure by the protruding tip of the gastrostomy tube&nbsp;on the opposite wall resulted in abrasion and ulceration that may lead to gastric hemorrhage. A chronic use of low dose ASA in our case may aggravate the ulcer formation and lead to gastric hemorrhage.

#### Conclusion.

The design of the gastrostomy tube with the protruding tip may predispose the patient to upper gastrointestinal bleeding due to ulceration. In such case, the gastrostomy tube should be changed to the PEG.

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**Title: POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME(PRES):A TREATABLE CONDITION**  
POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME(PRES):A TREATABLE CONDITION

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**INTRODUCTION:** PRES is neurotoxicity caused by a predisposing clinical condition with pathognomonic posterior cerebral edema visualized on neuroimaging .It is a relatively newly recognized syndrome,published for the first time in medical literature in 1996(1).It is a completely reversible pathology,as evidenced by our case below.

**CASE:** 29 yo F with PMH of SLE,uncontrolled HTN,CKD 4,biopsy-proven membranous sclerosing glomerulonephritis and anemia of chronic disease presented to the ER with severe left-sided headache for six days,blurring of vision,photophobia,nausea ,vomiting and seizure.Initial BP was 239/144. The patient was treated with Ativan IV and labetalol infusion in ICU.CT head was unremarkable.MRI of brain showed extensive white matter signal abnormality involving parietal,occipital and cerebellar lobes in a subcortical,bilateral and paramedian distribution.PRES was diagnosed.All presenting signs and symptoms resolved by next day as optimal BP control was achieved.

**DISCUSSION:** PRES results from inability of posterior circulation to autoregulate in response to acute changes in blood pressure,causing an injury to blood-brain barrier with resultant hyperperfusion and vasogenic cerebral edema. It has been reported in patients aged 4 to 96 years,but most cases occur in young to middle-aged females. Early recognition of its signs,symptoms and etiology is paramount as it is typically reversible if the precipitating cause is removed.Signs and symptoms seen in a case series of 120 cases were seizures,hypertension,encephalopathy,headache,visual disturbances and status epilepticus(2) .

Some of the common predisposing conditions are HTN,autoimmune disease,Toxemia of Pregnancy,solid organ or bone marrow transplantation,immunosuppressive treatment or conditions,cancer chemotherapy,biotherapy,sepsis,shock,renal disease and medications. Most common regions of brain involved (in a case study of 76 patients with PRES)(3) were parietooccipital,frontal,temporal,cerebellum,thalamus and brain stem. Diagnosis is established by a combination of suggestive clinical manifestations and the presence of characteristic cerebral edema on neuroimaging in a symmetric,bilateral,subcortical and watershed distribution.In doubtful cases,rapid clinical and radiologic improvement following appropriate treatment confirms the diagnosis.Multiple studies have shown that appropriate treatment of underlying cause is sufficient to completely resolve all clinical and radiologic manifestations of PRES in 1-2 weeks(4).However,clinicians must make timely diagnosis as it will obviate need for unnecessary procedures.Also,failure to treat or remove precipitating causes promptly can result in permanent neurologic impairment,coma or even death.

**KEY POINTS:** (1)PRES should be considered in any patient presenting with hypertension,altered mental status,vision abnormalities and seizure and MRI should be performed looking for diagnostic vasogenic edema.  
2)Prompt diagnosis and treatment of underlying cause will resolve clinicroadiologic manifestations of PRES;obviate need for unnecessary procedures and prevent neurologic impairment.

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## Resident / Fellow Clinical Vignette

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**Title: A RARE CASE OF ACUTE MYOCARDIAL INFARCTION SECONDARY TO CORONARY EMBOLISM IN A PATIENT WITH NON-ISCHEMIC CARDIOMYOPATHY**

**Introduction:**

Coronary embolism is a rare cause of acute myocardial infarction (MI). We are reporting a case of MI due to coronary embolism in a patient with Non-Ischemic Cardiomyopathy (NICM).

**Case Description:**

The patient is a 75 year old female with a medical history significant for NICM with an Ejection Fraction (EF) of 10-15% first diagnosed in 2008. At that time an angiogram revealed normal coronaries and Echocardiogram showed normal heart valves. Patient was admitted in 2014 for decompensation of her systolic congestive heart failure (CHF). While being treated for CHF, she suddenly developed new onset throat pain, followed by chest pain radiating to her left arm. Vitals at that time were notable for HR 92, BP 110/72, RR 18, Temp 98.4 F and SpO2 100% on room air. EKG revealed left bundle branch block (LBBB) and was similar to her baseline EKG except for nonspecific T wave inversions in Lead I and V6 and mild ST segment elevations of the inferior leads. Troponin-I were 0.11, 2.78 and peaked at 152. Emergent coronary angiogram revealed 100 % occlusion of Left Obtuse Marginal branch (OM1). Per cutaneous transluminal coronary angioplasty followed by embolectomy resulted in complete recanalization of the OM1. No atherosclerotic plaques were seen in any coronary arteries. Transthoracic Echocardiogram did not demonstrate any intra cardiac thrombus. Considering the fact that the patient had normal sinus rhythm, no valvular heart disease nor intracardiac thrombi, it was concluded that this embolus arose from the left ventricle given the severely depressed EF. Patient was anticoagulated with warfarin before discharge to home.

**Case Discussion:**

It is a well-established fact that myocardial infarction can occur due to coronary embolism especially in patients with known history of mechanical valve replacement with inadequate anticoagulation, bio-prosthetic mitral valve, hypertrophic cardiomyopathy, atrial fibrillation with or without valvular heart disease, and intramural thrombi. Coronary embolism in non-ischemic cardiomyopathy is extremely rare. An extensive search of literature revealed no case reports of MI secondary to coronary embolism in patients with NICM, normal valves, normal sinus rhythm and without intracardiac thrombi. Current guidelines recommend anticoagulation for at least 3 months in patients with LV dysfunction and intracardiac thrombi. However, there are no current guidelines on patients with embolic events and no intracardiac thrombi.

**Conclusion:**

This case provides a rare direct demonstration of a coronary artery embolus and subsequent MI that arose in the setting of a non-ischemic cardiomyopathy with no intracardiac thrombi, rhythm abnormalities or valvular heart disease. Regardless of having a "normal coronary angiogram", any new chest pain in patients with NICM should be investigated further to rule out new thromboembolic events. Duration of anticoagulation in this situation is not addressed by current guidelines and may require lifelong anticoagulation.

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**Title: Community-acquired Cavitory Lung Disease Caused by Group F Streptococcus**

A 48 year old man presented with fever, chills, night sweats, SOB, and productive cough for three weeks. He said his symptoms began while caring for his ex-wife's dogs. He also developed nausea, vomiting, weight loss, and malaise. A nonsmoker, he had no history of asthma, pet allergy, or recent travel. He was febrile, sweating, with mild respiratory distress. Pulse 110/min, BP 132/78mmHg, and respiratory rate, 25/min. Findings included nasal polyps, and lung crepitations. Laboratory studies included the following: WBC 22,600/uL, neutrophils 83.6%, anemia, and thrombocytosis, ESR 91mm/hr, CRP 32mg/dL. CXR revealed left basal opacity and LUL opacity.

He was treated empirically for CAP with ceftriaxone and azithromycin. He was isolated for possible TB. Blood cultures were negative. Three separate sputum cultures grew beta hemolytic Group F streptococcus. AFB, PPD, HIV, legionella antigen were negative. CT chest showed mass-like LUL opacities, left lower lobe consolidation, patchy right basilar opacities, and hilar and mediastinal lymphadenopathy. CT-guided biopsy showed acute fibrinous and organizing pneumonia. Antibiotic treatment was changed to ampicillin/sulbactam. After clinical improvement he was discharged on oral amoxicillin/clavulanate. CXR in two months showed resolving pneumonia; repeat CT chest in six months showed resolution of pneumonia with stable hilar, mediastinal lymphadenopathy.

The presentation was consistent with CAP. CT showed dense consolidation and abscess formation. Group F streptococcus, also named Streptococcus milleri group, may exist as part of normal flora. Although group F streptococcus is usually a harmless commensal, it is a pathogen in dental abscess, liver abscess, brain abscess, meningitis, empyema, peritonitis, and wound infections, especially after appendectomy.

While S. pneumoniae is the most common etiology of CAP worldwide, Strep milleri accounts for <3% of the cases of CAP. Our patient had acute fibrinous organizing pneumonia caused by Group F streptococcus. Exposure to dogs or cats has been described as a risk for Group F streptococcus infection; it is isolated from dog or cat bites.

Nearly all pathogenic isolates of S. intermedius and most of S. constellatus, but only 19% of S. anginosus were associated with abscess. They are second only to Klebsiella pneumoniae among pathogens isolated in lung abscesses (25% vs 16%). Their virulence is thought to be from cell surface properties that allow adhesion, fibronectin, platelet, and fibrin binding, and inhibition of phagocytosis and chemotaxis. The organisms also produce exotoxins: intermedilysin and hyaluronidase enzymes that are cytolytic and hydrolytic, facilitating tissue spread and pus liquefaction. Though group F Streptococci organisms are susceptible to penicillins, their pathogenic potential has been under-appreciated. Future ex vivo studies are needed to elucidate the factors which cause its virulence.

## Resident / Fellow Clinical Vignette

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### **Title: ACYLCOVIR IN RECURRENT ERYTHEMA MULTIFORME**

Erythema multiforme (EM) is a skin condition considered to be a hypersensitivity reaction to infections or drugs. It consists of a polymorphous eruption of macules, papules, and characteristic target lesions that are symmetrically distributed, with a propensity for the distal extremities. We present a case of recurrent EM.

A 55 year old lady presented to the dermatology clinic at the hospital with skin lesions. The lesions were symmetrically distributed 1-2cm dusky maculo-papules located on the thighs, arms and on the inner lips. A clinical diagnosis of EM was made and she was treated symptomatically. The lesions healed in a week. 5 months later she presented with a similar eruption. Serum chemistry, CBC and liver function tests were unremarkable. ESR was 17. HIV serology, mycoplasma and ANA were negative. HSV I and II IgG antibody titers were positive. A skin biopsy was consistent with EM. She was treated with oral acyclovir for 10 days with complete resolution of the lesions. 7 months later she had another recurrence. Further work up including hepatitis C viral serology and a search for underlying malignancy turned out negative. A skin biopsy again was reported as EM. Acyclovir was given and the lesions cleared in 7 days. She was offered long term acyclovir prophylaxis, which she declined.

Recurrent EM often is secondary to HSV-1 and 2 reactivation. In patients with herpes-associated EM (HAEM), HSV-1 is associated in 66.7% cases, HSV-2 in 27.8%, and HSV-1 and -2 coinfection in 5.6%. Patients with HAEM may have clinically apparent HSV reactivation without an episode of EM or EM without clinically apparent HSV infection. The pathogenesis of HAEM is consistent with a delayed-type hypersensitivity reaction. The disease begins with the transport of viral DNA fragments to distant skin sites by peripheral blood mononuclear cells. HSV genes within DNA fragments are expressed on keratinocytes, leading to the recruitment of HSV-specific CD4+ TH1 cells. The CD4+ cells respond to viral antigens with production of interferon- $\gamma$ , initiating an inflammatory cascade. Treatment is generally symptomatic for EM with local wound care, analgesics and antihistamines. HAEM is effectively managed with oral acyclovir. Prophylaxis for recurrent HAEM should be considered in patients with more than 5 attacks per year. Low-dose acyclovir (200 mg qd to 400 mg bid) for 6-12 months can be effective. Patients who have no response to acyclovir may have a response to valacyclovir or famciclovir, which have greater oral bioavailability and more convenient dosing. Alternative treatments for recurrent EM include dapsone, azathioprine, mycophenolate, thalidomide, cimetidine, and cyclosporine. Prophylactic antibiotics are not recommended because of the increased likelihood of selecting out resistant strains. Empiric treatment with acyclovir may be successful in cases of recurrent EM.

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### **Title: EOSINOPHILS AND PLASMA CELLS IN MULTIPLE MYELOMA**

Multiple myeloma (MM) is the second most prevalent blood cancer after non-Hodgkin's lymphoma. It represents approximately 1% of all cancers in white US residents and 2% of cancers in black residents. We present an unusual case of MM with eosinophilia, and eosinophilic pleural effusion.

A 69 year old female was evaluated for progressive shortness of breath and weight gain for 6 months. She had a history of diabetes and hypertension. She was on enalapril, losartan, insulin and simvastatin. No allergies were reported. She was obese with marked pedal edema. Vitals were unremarkable. Diffuse rhonchi with decreased breath sounds were noted at the lung bases. Routine blood investigations showed: Hb (8.7gm/dl), WBC 54,800/mm<sup>3</sup> with eosinophils 69%, neutrophils 25.8% and 3.5% lymphocytes. ESR was 109 mm, platelets 291,000/mm<sup>3</sup>, BUN (19mg/dl), creatinine (0.67mg/dl), LDH 240 IU/L, and calcium (8.0mg/dl). CXR showed bibasilar infiltrates with pleural effusion. CT scan of abdomen and pelvis showed hepatic vascular congestion with moderate splenomegaly with abnormal lytic process in the right iliac bone. Bone marrow aspiration showed mildly hyper cellular marrow with eosinophilia.

Immunohistochemical staining revealed CD138+ plasma cells, with >10% of marrow cellularity. Flow cytometry analysis showed markedly increased CD10-/CD16 eosinophils. Platelet derived growth factor receptor assay was done, and eosinophilic leukemia ruled out. Serum protein electrophoresis revealed: total protein 9.20g/dl, albumin 2.46g/dl, beta globulin 0.75g/dl, gamma globulin 5.12g/dl and M spike 4.09g/dl. IgG was markedly elevated: 6758g/dl and IgE 199g/dl. Pleural fluid analysis showed an exudate with 32% eosinophils. She declined any therapeutic intervention and passed away.

The association of plasma cell myeloma and eosinophilia is rare. Stromal cells induce MM cell proliferation predominantly through the secretion of IL-6. Neoplastic cells secrete IL-3 and IL-5, which may play a role in recruiting eosinophils. Eosinophils may then stimulate growth of malignant cells via an IL-6-independent mechanism. Pulmonary manifestations in MM are usually pulmonary infiltrates related to infections. Eosinophil predominant pleural effusions are very rare. Eosinophilia in MM must be differentiated from allergies, drug induced eosinophilia and eosinophilic leukemia. Whereas lenalidomide may be used in the treatment of MM, this drug may also cause eosinophilia. Our case highlights the complex relation between eosinophils and plasma cell proliferation.

## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Mohin Shah MD <b>Additional Authors:</b> Kaartik Soota MD, Nayef El-Daher MD <b>Institution:</b> Unity Health System</p> <p><b>Title: A RARE CASE OF SPONTANEOUS GAS GANGRENE CAUSED BY CLOSTRIDIUM SEPTICUM</b></p> <p><b>Introduction:</b> Spontaneous clostridium septicum associated infections are rare and commonly associated with colorectal malignancies. They carry a very high mortality rate making early diagnosis and appropriate treatment imperative. We present a case of spontaneous gas gangrene caused by clostridium septicum in a patient with adenocarcinoma of ascending colon.</p> <p><b>Case Presentation:</b> A 71 year old female with recent diagnosis of adenocarcinoma of ascending colon presented to the hospital with complaints of pain and rapid development of swelling in right foot that started 3 hours prior to arrival. She noticed a small lesion on her right heel which expanded to a large blister about tennis ball in size within 3 hours without any trauma to her foot. Physical examination was significant for fever of 38.7C, heart rate of 125 per minute along with edema and extreme tenderness in the right foot. At the right heel, there was a large blister about 10 cm x 8 cm in size. Laboratory studies revealed elevated leukocyte count of 25,000 with left shift and blood culture grew clostridium septicum. MRI of right foot showed gas gangrene of the right heel for which she underwent incision and drainage of the blister along with local debridement of necrotic tissue. All this while she was on broad spectrum intravenous antibiotics which were later changed to organism targeted oral ampicillin and metronidazole.</p> <p><b>Discussion:</b> Spontaneous clostridium septicum infections are rare and estimated to be the cause of only 1.3% of all clostridia infections. Being a rare infection, it is not well studied. However, It has been observed that almost half of the patients diagnosed with clostridium septicum infection have primary malignancy, most common being colon carcinoma (75%) of which 40% patients had cecal cancer. Current literature suggests that mucosal ulceration of the tumor surface and hematogenous invasion allow a portal of entry for the bacteria. It usually presents as spontaneous gas gangrene of either upper or lower extremity. Early initiation of antibiotics along with aggressive surgical debridement is the mainstay of treatment. Penicillin group of medications are the drug of choice for clostridium septicum infection, however duration of the antibiotics is not well studied. Generally 10-14 days of antibiotics have shown successful response.</p> <p><b>Conclusion:</b> Clostridium septicum is a rare cause of spontaneous gas gangrene most commonly associated with colon cancer. Hematogenous invasion from the gut is the most common portal of entry. Early diagnosis and treatment is very essential as delay in the diagnosis can lead to adverse outcomes with mortality rate up to 70%.</p>	<p><b>Author:</b> Bikram Sharma MD <b>Additional Authors:</b> Dr Dinesh Subedi <b>Institution:</b> SUNY Upstate Medical University</p> <p><b>Title: MELOXICAM INDUCED ASEPTIC MENINGITIS</b></p> <p><b>INTRODUCTION:</b> We are presenting an interesting case of meningitis from Meloxicam in a young otherwise healthy patient who was recently started on Meloxicam. Although there have been various case reports of ibuprofen causing aseptic meningitis, there has been no reported case from Meloxicam to the best of our knowledge.</p> <p><b>CASE PRESENTATION:</b> 43 year old woman presented with 2 days history of occipital headache 8/10, low grade fever, nausea and photophobia. Her past medical history included depression and viral meningitis 3 years ago. She did not have known drug allergies. She injured her right hand 4 days ago and was taking frequent doses of Meloxicam 7.5mg 3-4 times a day. Her other medication was Sertraline which she was taking for past 3 years. Examination revealed neck rigidity and positive Kernig's sign. She did not have any rash, oral ulcers or joint effusions. CT head and MRI brain were normal. CSF fluid analysis revealed clear fluid, WBC 260 with 76% monocytes and 24% polys, glucose 60 (concurrent blood glucose 101), protein 102, RBC 3. This picture was suggestive of aseptic meningitis. Basic metabolic panel and complete blood count were normal. She was initially started on vancomycin, ceftriaxone and acyclovir. CSF fluid PCR was negative for Herpes simplex and eventually all the antibiotics were discontinued. She improved in 48 hours and was discharged on stable condition</p> <p><b>DISCUSSION</b> Drug-induced aseptic meningitis is a rare but serious complication of drug therapy. As per the FDA report, most common drug reported to cause aseptic meningitis is Infliximab. Aspirin and Ibuprofen are the most common (NSAID) non-steroidal anti-inflammatory drugs. Meloxicam is one of the non selective COX inhibitor which is used for its analgesic, antipyretic and anti-inflammatory properties. The clinical signs and symptoms of drug-induced meningitis are similar to those of infectious meningitis and include fever, headache, photophobia, and stiff neck. The laboratory findings are also similar, including cerebrospinal fluid (CSF) pleocytosis mainly neutrophils, elevated protein, normal or low glucose levels and negative cultures. Drug induced meningitis has an excellent prognosis. Diagnosing it early is imperative to prevent ongoing harm and also potential harm from subsequent antibiotics.</p> <p><b>CONCLUSION</b> NSAIDs are very popular medications for various clinical entities. Even though the risk for serious side effects like aseptic meningitis are low, clinicians should consider NSAIDs like Meloxicam as potential causes of aseptic meningitis, especially in patients with proper clinical context and no obvious infectious cause.</p> <p><b>REFERENCES</b> Drug-induced aseptic meningitis March 2005, Vol. 4, No. 2 , Pages 285-297 (doi:10.1517/14740338.4.2.285) Susan Hopkins , Stephen Jolles Nonsteroidal anti-inflammatory drugs exposure and the central nervous system. Auriel E, Regev K, Korczyn AD.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Niraj Shenoy M.D <b>Additional Authors:</b> Tesfaye, Melaku M.D., Meholfi, Mimoza M.D., Mable, Peter M.D. <b>Institution:</b> Jacobi Medical Center, Albert Einstein College of Medicine</p> <p><b>Title: A CASE OF STEROID RESISTANT BULBAR NEUROSARCOIDOSIS RESPONSIVE TO INTRAVENOUS IMMUNOGLOBULIN</b></p> <p>We report a rare case of bulbar neurosarcoidosis presenting with dysphagia and dysphonia that was steroid resistant, making both diagnosis and management challenging. A 37 yr old man presented with dysphagia to solids and liquids, dysphonia, fatigue, generalized pruritus and 50 lb weight loss over 2 months. Positive examination findings were his dysphonia, mild bilateral palatal weakness with depressed gag reflex but preserved sensation in the posterior third of the tongue, mild neck flexor and R deltoid weakness (4+/5), multiple diffuse 0.5cm-1cm palpable hyperpigmented skin lesions. Initial laboratory tests revealed an elevated ACE level.</p> <p>Chest X ray and CT chest revealed prominent hilar lymphadenopathy. Barium swallow revealed severe oropharyngeal dysphagia consistent with a neurogenic cause. A transbronchial biopsy of one of the enlarged lymph nodes revealed non-caseating granulomas with negative AFB and fungal stains, confirming sarcoidosis. MRI of the brain was unremarkable. CSF analysis revealed a mildly elevated protein level. EMG of the sternocleidomastoid and genioglossus muscles showed denervation changes.</p> <p>When the pathology report confirmed sarcoidosis, he was started on steroids, initially methylprednisolone IV and later prednisone 60mg/day. Given no symptomatic response to steroids, the possibility of co-existent progressive bulbar atrophy was considered, which is nearly always fatal. He was then treated him with IVIG 2 g/kg over 2 days, as he was reluctant to have any stronger cytotoxic immunotherapy, which has been better studied in neurosarcoidosis.</p> <p>He was discharged home on low dose prednisone- which he tapered off against our advice- completely dependent on PEG feeds. Within one week of completing IVIG treatment, he reported significant improvement, and was able to meet 50% of his required nutritional intake by mouth. Over the next 3-4 weeks his dysphonia almost completely resolved and at 12-14 weeks after IVIG treatment, his swallowing was back to normal.</p> <p><b>Discussion:</b></p> <ul style="list-style-type: none"><li>- This case is a classic example of how neurosarcoidosis can mimic the clinical and electrophysiological features of neurological disorders such as progressive bulbar palsy. It should therefore be considered as a treatable mimic in the differential diagnosis of such disorders.</li><li>- Patients with neurosarcoidosis may benefit from a trial of IVIG, which is a safer alternative to cytotoxic immunotherapy. Although literature search reveals a few reports of the efficacy of IVIG in peripheral neurosarcoidosis, this is the first report of its efficacy in steroid resistant bulbar neurosarcoidosis.</li></ul>	<p><b>Author:</b> Yash Shravah MD <b>Additional Authors:</b> Roxana Elena Lazarescu, MD</p> <p><b>Institution:</b> New York Hospital Medical Center of Queens</p> <p><b>Title: Smoking - What is it 'good' for?</b></p> <p>Smoking what is it 'good' for? Yash Shravah, MD (Member), Roxana Lazarescu, MD (Member) Introduction: Tobacco is an extremely well studied substance with many detrimental side effects, however its beneficial effects are less well understood. There are many harmful products within tobacco plant itself, most notably the addictive alkaloid nicotine. However, there are other compounds that are less well studied such as anatabine. Anatabine is a minor alkaloid found in the same tobacco plant that has recently started to be studied has a beneficial effect in patients with Hashimoto's thyroiditis.</p> <p>Case Presentation: This is a 58-year-old male with a past medical history of hypertension, hyperlipidemia, and sudden cardiac arrest 4 months ago, who presented with an inability to chew. Jaw weakness was associated with generalized weakness, fatigue, muscle tightness, severe tiredness, and increased sleepiness for the past 4 months. Patient also complained of dyspnea on exertion and occasional chest pain. He stopped smoking 4 months ago following sudden cardiac death in Russia (as per patient with extensive work-up and unclear etiology). He also says he has a 10-12 pound weight gain since smoking cessation. He denies hematuria, dysuria, constipation, unusual hair loss, or fever. He also denied illicit drug use (no cocaine, heroin, marijuana, amphetamines), and had no alcohol use. Vital signs were unremarkable as well as was physical exam including thyroid examination. Labwork was significant for elevated creatinine kinase, high thyroid stimulating hormone, and strongly positive anti-thyroid peroxidase antibodies.</p> <p>Discussion: Smoking seems to induce changes in thyroid function tests, like a decrease in TSH and increase in thyroid hormones. In Hashimoto's disease, a lower prevalence of thyroglobulin antibodies, thyroperoxidase antibodies and hypothyroidism were found in smokers. Carlsson et al assessed in a recent study the association between smoking habits (smoking cessation in particular) and development of autoimmune hypothyroidism. Incidence of hypothyroidism was very common in people who had recently stopped smoking. Results were consistent in both sexes irrespective of age. Within two years after smoking cessation, the percentage of hypothyroid cases attributable to cessation of smoking was 85%. In conclusion, the risk of having overt autoimmune hypothyroidism diagnosed was more than 6-fold increased the first 2 years after cessation of smoking. However the component that is responsible for such effects has only recently been suggested to be the minor alkaloid anatabine. There are several studies that have shown a benefit to the use of anatabine in mice. Currently there are ongoing studies called the ASAP human thyroid study which is looking at the effect of taking supplemental anatabine in thyroid disease. While this was a randomized control trial showing beneficial effect of anatabine, it was performed with low power and further studies will be required.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Aditya Shreenivas <b>Additional Authors:</b> Aditya Shreenivas, MD; Arlene Tieng, MD; Giovanni Franchin, MD, PhD; Sridhar Chilimuri, MD <b>Institution:</b> Bronx Lebanon Hospital Center <b>Title: EOSINOPHILIC VARIANT OF GRANULOMATOSIS WITH POLYANGIITIS PRESENTING AS HENOCHE-SCHONLEIN PURPURA: A CASE REPORT</b></p> <p>Background: Granulomatosis with polyangiitis (GPA) rarely masquerades as Henoch-Schönlein purpura at initial presentation(1). Tissue biopsies help to direct organ-saving therapy, but can also further complicate a diagnosis. The eosinophilic variant of GPA seems to coincide with the revised Chapel Hill Consensus Conference definition of eosinophilic GPA (2). <b>Objective:</b> To report a case of granulomatosis with polyangiitis with an atypical initial presentation and tissue eosinophilia. <b>Case report:</b> In December 2013, an 18-year-old Hispanic man presented to our emergency department with a rash and dark urine as well as a one-week history of hand swelling with limited range of motion. Three weeks before admission, he experienced vomiting, abdominal pain, and bloody diarrhea which resolved spontaneously. He then developed nasal congestion and cough. He had no history of asthma. Family history was significant for rheumatoid arthritis in his mother and psoriasis in his father. A physical examination revealed a temperature of 100.5°F, macular purpura on his legs, knee tenderness, and ankle swelling. The findings of neurological examination were normal. Initial work-up demonstrated normocytic anemia and an eosinophil level of 1012/L (8.8%). Urine examination revealed hematuria and spot protein-creatinine ratio of 0.3. Henoch-Schönlein purpura was considered as a differential diagnosis. Chest X-ray demonstrated ground-glass attenuation. Despite empiric treatment with azithromycin and ceftriaxone, he remained febrile and his respiratory status worsened. Skin biopsy showed eosinophils and leukocytoclastic vasculitis. Direct immunofluorescent studies were negative for immunoglobulin (Ig) A. ANCA was positive for proteinase 3 (PR3) antibody. Chest computed tomography demonstrated bilateral ground-glass opacities. Bronchoscopy revealed diffuse alveolar hemorrhage. Bronchoalveolar lavage revealed eosinophilia of 52%. A transbronchial lung biopsy showed capillaritis, abundant eosinophils, and macrophages with hemosiderin, without granuloma. The patient was treated with pulse intravenous methylprednisolone 1g/day for 3 days followed by oral prednisone at 60mg/day, resulting in an improvement in the chest X-ray. Renal biopsy revealed pauci-immune necrotizing and crescentic glomerulonephritis, eosinophils, and granulomatous inflammation. Immunofluorescent microscopy revealed an absence of Ig and complement. Induction therapy with rituximab (375mg/m<sup>2</sup>) for four weeks resulted in remission. The PR3-ANCA became undetectable and the eosinophil count normalized. Maintenance therapy included prednisone and methotrexate. <b>Discussion:</b> Our patient initially fulfilled the clinical classification criteria for Henoch-Schönlein purpura, but the positive PR3ANCA, absence of IgA, and eosinophilic infiltration in the skin, lung, and kidney led to the diagnosis of an ANCA-associated vasculitis. Tissue biopsy with immunofluorescent studies is critical in the management of an ANCA-associated pulmonary-renal syndrome. <b>References</b> 1. Bui T, Chandrakasan S, Poulik J, Fathalla BM. Granulomatosis with polyangiitis presenting as Henoch-Schönlein purpura in children. <i>J Clin Rheumatol.</i> 2013; 19:199-202. 2. Jennette JC, Falk RJ, Bacon PA, Basu N, Cid MC, Ferrario F, et al. 2012 revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. <i>Arthritis Rheum.</i> 2013; 65:1-11.</p>	<p><b>Author:</b> Anup Shrestha <b>Additional Authors:</b> Purnima Sharma MD Amit Kachalia MD Sethu Muralidharan MD</p> <p><b>Institution:</b> Icahn School of Medicine at Mount Sinai, Queens Hospital Center</p> <p><b>Title: GBS Endocarditis and Multifocal pneumonia presenting as a postpartum fever</b></p> <p><b>Introduction:</b> Group B streptococcus (GBS) is an important organism causing fever in postpartum patient. Fever due to Tricuspid Valve endocarditis (TVE) is a rare condition. We present an interesting case of TVE and Multifocal pneumonia causing postpartum fever.</p> <p><b>Case Presentation</b> A 37-year-old female with no prior medical condition, had cesarean section 18 days prior to admission, comes in with non-productive cough, fever, shortness of breath of 4 days without orthopnea or paroxysmal nocturnal dyspnea. Physical examination was remarkable for fever, bilateral diffuse crackles, pansystolic murmur in the apex. Labs showed elevated white count 16.4k/mcl, positive D Dimer, ESR of 125. Chest x-ray revealed bilateral fluffy infiltrates. CT Chest showed small bilateral pleural effusion, patchy consolidation and ground glass opacities all over the lungs consistent with multifocal pneumonia with no pulmonary embolism. Echocardiography showed multiple large mobile masses attached to tricuspid valve with largest measuring 2.5 cm in length suggestive of vegetation. Ceftriaxone was started initially for pneumonia, which was switched to high dose penicillin G based on two-blood culture positive for GBS growth. Patient has negative GBS screening test during this pregnancy. Fever persisted intermittently for a 10 days due to large bacterial burden from septic pulmonary emboli. Follow up echocardiography showed significant reduction in the size of Tricuspid valve mass. Repeat blood cultures were negative for any growth. Her symptoms improved with 6 weeks of intravenous antibiotics.</p> <p><b>Discussion:</b> Isolated native valve endocarditis is very rare valvular condition and accounts from 5-10% of infective endocarditis 1. IV drug abuse is the most common cause of TVE 2. Staphylococcus aureus is the most common cause. Streptococcus species and gram-negative bacilli are other common causes 1. Pulmonary symptoms of pneumonia and dislodged vegetative material are commonly seen than cardiac manifestations in TVE 1, 2. Literature review has shown 31 cases of GBS endocarditis associated with pregnancy of which 6 of them colonized tricuspid valve. Fever and chills in postpartum woman should always raise suspicion of endometritis. However same symptoms with signs of multifocal pneumonia should raise suspicion of IE with septic emboli 3.</p> <p><b>Conclusion:</b> TVE is a rarely caused by GBS. It manifests mostly as a pulmonary symptoms due to septic emboli. We suggest considering TVE and multifocal pneumonia with GBS endocarditis as a cause of postpartum fever even if the patient had a negative GBS screening test.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Anup Shrestha <b>Additional Authors:</b> Michael Babigumira, MD Vincent Rizzo, MD <b>Institution:</b> Icahn School of Medicine at Mount Sinai, Queens Hospital Center</p> <p><b>Title:</b> <b>AGEP leading to Acute Kidney Injury and Stroke secondary to Nifedipine</b></p> <p><b>Introduction</b> Acute Generalized Exanthematous Pustulosis is one of the severe cutaneous adverse reactions. We present an interesting case of AGEP due to Nifedipine. No previous reports of AGEP with Nifedipine have ever been reported to date.</p> <p><b>Case Presentation</b> A 52-year-old female with PMH of HTN, Psoriasis and Diabetes Mellitus type 2, recently treated for Hypertensive urgency, comes in generalized development of small pustules all over her body over the past 3 days. Last discharge medications included Nifedipine ER, Labetalol, and Metformin. Negative history of fever, headaches, or recent sick contacts. Physical examination was significant for innumerable, small fine pustules covering her entire body. Labs were significant for leukocytosis with bandemia. Intravenous fluids and Vancomycin were initiated. Impression of AGEP vs Pustular Psoriasis was made. Skin biopsy was done, which turned out positive for AGEP. Four sets of blood culture were negative for any growth. On day 6 of admission the patient developed Acute Kidney Injury. AKI workup was consistent with ATN with a negative urinary eosinophil count. On hospital day 9, the patient developed new right Cerebellar and left frontal stroke with change in her speech pattern. She was started on aspirin, statin. Her BP meds were continued. After 18 inpatient days, the patient recovered with complete new skin. She was discharged home with outpatient appointments in medical and dermatology clinic.</p> <p><b>Discussion</b> AGEP is a very rare but severe cutaneous adverse reaction that is usually caused by drugs 1. Antibiotics have been implicated as the causative agents in more than 90% of the cases<sup>3, 4</sup>. According to the EuroSCAR study, the agents conferring the highest risk are pristinamycin, aminopenicillins, antibacterial sulphonamides, and diltiazem<sup>5</sup>. CD4 and CD8 cells are involved in the pathogenesis of the disease. The treatment of AGEP is supportive care.<sup>2</sup> The entire self-limiting episode may last up to 15 days<sup>3</sup>. AGEP has a favorable prognosis. In previous studies, liver and kidney injury were reported<sup>3</sup>. 32% of patients with AGEP were found to have disturbed renal function in a retrospective analysis.<sup>3</sup> Our search in PubMed with AGEP leading to AKI revealed only one article and no articles with AGEP leading to stroke.</p> <p><b>Conclusion:</b> This is a very rare and interesting case of AGEP along with AKI and stroke secondary to Nifedipine, the only calcium channel blocker used in the patient.</p>	<p><b>Author:</b> Varda Singhal MD <b>Additional Authors:</b> Vasvi Singh* MD, Gokul Samudrala* MD, Wajid Choudhry* MD *Unity Health System, Rochester, NY</p> <p><b>Institution:</b> Unity Health System</p> <p><b>Title:</b> <b>KEEPING BLOOD THIN BROKE THE SKIN: NON UREMIC CALCIPHYLAXIS</b></p> <p><b>Introduction:</b> Calciphylaxis is a metastatic calcification of blood vessels causing microvascular occlusion and target organ hypoperfusion. It is most commonly associated with end stage renal disease and hyperparathyroidism along with a high serum calcium phosphorus (CaPO<sub>4</sub>) product. We present a case of biopsy proven calciphylaxis with warfarin use and normal CaPO<sub>4</sub> product.</p> <p><b>Case description:</b> A 60-year-old white male presented to the office with rapidly progressive painful leg ulcers that started 4 weeks ago. He has diabetes mellitus controlled with oral hypoglycemic agent, atrial fibrillation and a history of stroke. On physical examination, he had necrotic skin ulcerations bilaterally below the knees, on medial and lateral aspects of his legs, covered by eschars in some areas. Initial differential diagnosis were diabetic ulcers and peripheral vascular disease. He was treated with topical antibiotics without any improvement. Three months later he underwent a skin biopsy which revealed subcutaneous vascular calcification, fibrosis, reactive angioendotheliomatosis with no vasculitis and a diagnosis of calciphylaxis was made. Laboratory findings: serum creatinine- 0.92mg/dl, serum albumin 3.9 gm/dl, serum calcium- 9.9 mg/dl, serum phosphorus- 3.7 mg/dl and CaPO<sub>4</sub> product 36.6. Vitamin D level, LFTs, hypercoagulability work up, ESR, CRP and intact parathyroid hormone level were normal. One year after his initial presentation, he was started on intravenous pamidronate and treated for 6 months with no improvement. On retrospective analysis, it was found that his lesions were first noticed at the time of warfarin initiation. The warfarin was discontinued and treatment with intravenous sodium thiosulfate was initiated. He responded well and his lesions started improving within a month.</p> <p><b>Case discussion:</b> Calciphylaxis is an uncommon condition with a mortality rate of 60-80%, mainly secondary to infected skin lesions. The gold standard for diagnosis is incisional cutaneous biopsy. Very few cases of calciphylaxis have been reported in patients with normal renal function. Review of literature suggests that it has been associated with hypoalbuminemia, warfarin use, steroid use, liver disease, hypercoagulable states, subcutaneous insulin and iron dextran injections. Our case is one of the rare cases of calciphylaxis associated with warfarin use, normal renal function and normal CaPO<sub>4</sub> product. The postulated mechanism is that warfarin inhibits the synthesis of matrix G1A protein which is Vitamin K dependent and is responsible for inhibiting the mineralization of tissues. As a result microcalcification promotes occlusion leading to tissue ischemia and ulceration.</p> <p><b>Conclusion:</b> Calciphylaxis should be considered as a differential diagnosis for painful skin ulcers even in patients with normal renal function so that treatment can be initiated early during the course. Sodium thiosulfate has shown promising results in a few case reports.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Chamundeswari Subramanian MD, PGY-2 medical resident <b>Additional Authors:</b> Nima Hani MD, Binaya Dahal MD, Prasanta Basak FACP, Stephen Jesmajian FACP. <b>Institution:</b> Montefiore New Rochelle</p> <p><b>Title: DUODENAL ADENOCARCINOMA - A RARITY</b></p> <p>Introduction: Cancer of the small bowel is rare, accounting for 1 % of gastro-intestinal malignancies. Adenocarcinomas are the most common of small bowel malignancies, followed by carcinoid tumors, lymphomas, and leiomyosarcomas. Adenocarcinoma of the duodenum is uncommon, accounting for less than 0.4% of all gastro-intestinal tract tumors. About 45% of these tumors arise from the third and fourth parts of the duodenum. We present a case of duodenal adenocarcinoma arising from the second part of the duodenum.</p> <p>Case: A 70 year old male presented with progressive loss of appetite, 30 pound weight loss and dysphagia for 2 months. 10 days prior to admission he developed burning retrosternal pain. CT abdomen showed a stomach distended with food and an edematous stomach wall. Upper GI endoscopy revealed copious food material in the esophagus and stomach; suspicious for gastric outlet obstruction. An explorative laparotomy with simultaneous upper GI endoscopy was performed. A mass was felt in the 2nd part of duodenum. Upper GI endoscopy showed an open pylorus with stricture and abnormal mucosa at the junction of the duodenal bulb and the 2nd part. Frozen section biopsy revealed adenocarcinoma. The tumor involved the duodenal and gastric wall, ampulla of Vater, pancreas and peri pancreatic soft tissues. He underwent pancreaticoduodenectomy with jejunostomy. Pathology confirmed moderately differentiated duodenal adenocarcinoma stage 3 (T4N1).</p> <p>Discussion: The incidence of small bowel GI adenocarcinoma is highest in the duodenum. Its vague symptoms often lead clinicians to suspect other more benign differential diagnoses. Recommended treatment for localized disease of 1 st and 2nd portion of duodenum is pancreaticoduodenectomy, and for 3rd and 4th part with segmental resection. Definitive surgery is the only means of potential cure, with the prognosis being significantly better for nodenegative patients. Lymph node positive disease requires postoperative chemotherapy. Locally advanced unresectable or metastatic disease is managed with systemic chemotherapy. Resectability and presence of distant metastatic disease are the strongest determinants of outcome for patients with duodenal adenocarcinoma. Nodal status offer little prognostic information and nodal positivity should not preclude resection. As patients have symptoms similar to those of pancreatic adenocarcinoma but have an outlook more comparable to gastric adenocarcinoma, a vigorous approach to resection is justified. The 5-year survival rate for resected adenocarcinomas of the duodenum is about 50 to 60%. Adenocarcinoma of the duodenum remains a rare disease, though the prevalence appears to be rising possibly due to improved diagnostic techniques. Clinicians need to be aware of this rare disorder.</p>	<p><b>Author:</b> Tarun Tandon <b>Additional Authors:</b> Lalit Wadhvani* M.D, Varda Singhal* M.D *Unity health System, Rochester NY</p> <p><b>Institution:</b> Unity Health System</p> <p><b>Title: A RARE CASE OF GIANT CELL MYOCARDITIS IN AN ELDERLY FEMALE</b></p> <p>INTRODUCTION: Giant Cell Myocarditis(GCM) is an infrequently encountered fatal form of myocarditis usually seen in young individuals. It generally presents as acute congestive heart failure (CHF) or heart rhythm abnormalities including heart block and ventricular tachycardia. We are presenting a rare case of fatal GCM in an elderly female.</p> <p>CASE DESCRIPTION: A 67-year-old female with hypertension and hyperlipidemia presented to the cardiology outpatient clinic for exertional dyspnea and dizziness for a period of 4 months. An electrocardiogram was done which revealed 1st degree AV block with no ischemic changes. She had normal troponins. An exercise treadmill test revealed a rate related 2:1 Mobitz type 2 AV block. Subsequent stress echocardiogram and nuclear myocardial perfusion scan were negative for ischemia and showed an EF of 55%. To evaluate the underlying AV block, she underwent an EP study which revealed an infra-hisian block and she had a dual chamber pacemaker placed. She was doing well initially, but three months after the pacemaker was implanted, she presented to the office complaining of palpitations. A subsequent pacemaker interrogation showed non-sustained ventricular tachycardia which was treated with atenolol. Despite the medical therapy, her palpitations and dyspnea persisted and she developed chest pain leading to hospital admission. A troponin of 2.69 lead to an angiogram which showed non-obstructive lesions in two vessels and a severely reduced EF of 25% with global hypokinesis compared to the prior EF of 55%. Two months later, an echocardiogram done showed an EF of 5%-10%. To evaluate her progressively worsening EF, a cardiac biopsy was performed which surprisingly showed GCM. Further therapy included IV Steroids and a Bi-Ventricular assist device, but unfortunately the patient died 1.5 months after biopsy, 8 months after initial presentation.</p> <p>CASE DISCUSSION: GCM, is poorly understood and may be an autoimmune process mediated by T lymphocytes. It has a very high mortality rate. The disease is predominantly seen in women compared to men, with greater incidence in young or middle aged individuals. These patients generally present with an AV nodal blockade or heart failure. Conventional treatment with medications for these symptoms have no effect. Immunosuppressants and steroids slow disease progression. Cardiac transplant may be required for mortality benefit.</p> <p>CONCLUSION: The presence of the triad including AV block, heart failure and ventricular tachycardia, without evidence of ischemia, should prompt consideration of GCM since early treatment with AICD, left ventricular assist device, or cardiac transplant is shown to have survival benefit.</p>
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## Resident / Fellow Clinical Vignette

<p><b>Author:</b> Mohamad Tarazi MD <b>Additional Authors:</b> Kartik Ramikrishna, MD Roy Trumbo, MD <b>Institution:</b> Unity Health System</p> <p><b>Title: Krokodil Bit His Legs</b></p> <p>Introduction: With the crack down on drugs like heroin, new drugs are emerging, such as Krokodil (Desmormorphine), a home-made opioid which is notorious for its necrotizing effects on the skin. This flesh eating drug is a serious concern to both the medical community and drug-enforcement agencies.</p> <p>Case Presentation: 47 year old male with history of cocaine and heroin abuse, presented with worsening ulcers over both legs. He admitted to injecting a new, cheaper form of Heroin into his legs for the past 3 months, and began to develop ulcers soon after. He denied fever, chills, trauma or swelling. Physical exam was noticeable for wide spread excoriation and ulceration extending from ankles to knees. Purulence noted at the base of the ulcers and islands of eschar were present throughout. His feet were warm to touch and dorsalis pedis pulses were palpable bilaterally. Urine toxicology screen was positive for cocaine, opiates, benzodiazepines and cannabinoids. Arterial Doppler study showed adequate flow. Venous Doppler Ultrasound was negative for any thrombosis. He was treated with empiric antibiotics and local wound care. The following day he developed withdrawal symptoms and left against medical advice. Final blood and wound cultures were negative for any bacterial growth.</p> <p>Discussion: Krokodil (Desmormorphine) is an intravenous home-made opioid, prevalent in Russia and Ukraine. Krokodil, the Russian term for crocodile, likely got its name from desmormorphine's precursor chemical, a-chlorocodide, or from the skin manifestations it causes. It is easy and cheap to manufacture and is made from codeine, iodide, red phosphorus (from matchsticks), paint thinner and gasoline. These along with other impurities from the manufacturing process are responsible for skin, bone and muscle necrosis at the site of injection, which can be limb threatening. It is ten times more potent and faster in onset than Morphine. Compared to heroin it has a shorter half-life, although withdrawal symptoms have been reported to last longer. The drug has gained popularity in Russia and Eastern Europe, with an estimate of 100,000 to 500,000 Krokodil abusers. A few cases have been reported in the US and Canada, raising concern for its emergence in this region. Public awareness about this drug is required to identify its dangers and to limit its spread within the community. Unfortunately, there is no current specific test to identify Krokodil abuse.</p>	<p><b>Author:</b> Nishant Tripathi MD <b>Additional Authors:</b> Hasmukh Patel, Sidharth Jain, Pankaj Mathur</p> <p><b>Institution:</b> Brookdale University Hospital Medical Center</p> <p><b>Title: Spiked HELMET SIGN- AN EKG MARKER FOR GRAVE PROGNOSIS IN CRITICALLY ILL PATIENTS</b></p> <p>Introduction: It's difficult to identify ischemic myocardial events in critically ill patients. A different variety of EKG findings namely spiked helmet pattern has been recently reported in literature as a very poor prognostic marker of critically ill patients. Curiously this anomaly is mostly seen in terminally ill patients presented usually with non-cardiac complaints.</p> <p>Case Summary: A 90 year old man with PMH of HTN, DM-2, and Coronary artery disease, s/p coronary artery bypass graft in 2001 presented with new onset seizure and unresponsiveness. The physical exam revealed afebrile, stuporous elderly male with BP of 148/70 mm Hg, and pulse of 85/min. He had reactive pupils, supple neck and no focal neurological deficits. On labs patient was noted to have elevated WBC count (14,500/microliter), BUN of 54 mg/dl, Creatinine of 1.9 mg/dl, Troponin of 4.7 ng/ml, pro BNP of 28100, blood sugar of 427 mg/dl, serum osmolality of 332 and VBG ph of 7.39. CT head revealed diffuse cerebral atrophy, bilateral hygromas, no intracranial hemorrhage, mass or infarct. CXR revealed bilateral increased markings, EKG revealed NSR at 85 bpm, with LVH (left ventricular hypertrophy). The echocardiogram revealed Left ventricular ejection fraction of 35-45% with diffuse hypokinesis, severe tricuspid regurgitation, and pulmonary artery pressure of 60-65 mm Hg.</p> <p>The patient was managed for seizures, hyperosmolar state, NSTEMI, and acute renal failure. Patient continued to have recurrent seizures valproic acid and levetiracetam was started. Patient's troponin peaked up to 13 ng/ml. Patient was started on therapeutic anticoagulation with heparin drip for NSTEMI. On third day of hospitalization patient went into pulseless electrical activity, was resuscitated and started on vasopressor which was tapered off within 12 hours. On day 4 spiked helmet pattern in EKG appeared. The patient remained stable for couple of days but on day 6 of hospitalization patient became hypotensive, tachycardic, and 12 lead EKG revealed new onset atrial fibrillation. After failed electrical cardioversion, amiodarone drip was started. The family was explained about the poor prognosis and patient was subsequently made DNR. Later on Day 8 of hospitalization patient became hypotensive, bradycardic and patient expired.</p> <p>Discussion: Spiked helmet pattern in EKG is being reported more frequently among the cardiologists, but given its association with usually grave prognosis, primary care/ internists need to be aware about this curious finding. The exact mechanism of the spiked helmet ECG pattern and its association with critical illness is uncertain, but repeated diaphragmatic pulsations either directly by inferior wall of heart or stimulation of left phrenic nerve has been postulated as the causative factors. More research is needed to find out the exact mechanism of this anomaly and also if any intervention is warranted after this EKG finding.</p>
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## Resident / Fellow Clinical Vignette

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### **Title: CHRONIC LYMPHOCYTIC MYOCARDITIS, AN UNUSUAL CAUSE OF CARDIOGENIC SHOCK**

**DISEASE OVERVIEW:** Chronic lymphocytic myocarditis is a disease entity characterized by inflammation, necrosis and lymphocyte infiltration of cardiac myocytes leading to fulminant cardiac dysfunction and/or death. It is a rare disease occurring in 1 per 100,000 persons per year.

We present a 56 year old woman from El-Salvador, with a known history of hypertension, dyslipidemia and diabetes mellitus who was admitted with cardiogenic shock. She was dyspnoeic, hypotensive, tachycardic with elevated JVD, right basilar crackles and holo-systolic murmur maximal at the apex. She also had elevated parvovirus IgM, positive HBSag and positive ANA.

Serum chemistries, cardiac enzymes, thyroid function, lipid profile, CBC, Lyme titer, quantiferon TB gold, HIV serology, dsDNA as well as urine toxicology were unremarkable. ECG revealed low voltage QRS, long QT interval and non-specific ST wave pattern. CXR showed cardiomegaly with clear lungs. Cardiac MRI showed LVEF of 10%, dilated RV with moderately reduced RV function. Right heart catheterization revealed elevated right atrial pressure, while angiography showed normal coronary arteries. Cardiac biopsy revealed numerous foci of lymphocytic infiltrates associated with myocyte necrosis. Treatment with afterload reduction and inotropic agents was unsuccessful, following which she was implanted with a ventricular assist device (VAD) as a possible bridge to cardiac transplant. Postoperatively she was hospitalized for three weeks and discharged home with stage II heart failure.

#### **Discussion**

Chronic lymphocytic myocarditis usually manifests as acute decompensated heart failure or conduction abnormalities. Initial presentation may mimic or follow a viral illness, endocrinopathy or immunologic disorder thus posing diagnostic challenges. Physical findings include tachycardia, heart murmur, cardiomegaly, arrhythmia, leucocytosis, elevated ESR/CRP.

Diagnosis requires a high index of suspicion and the disease is confirmed by biopsy finding of lymphocytic infiltration of cardiac myocytes. Elevated blood levels of viral IgG/IgM and finding of a viral genome on cardiac biopsy is also utilized in identifying the specific cause.

Our patient demonstrated a rapidly deteriorating cardiac function, biopsy evidence of lymphocytic infiltration and necrosis of cardiac myocytes. Elevated blood levels of parvo virus IgM/IgG implicates parvo virus as cause of the myocarditis leading to cardiogenic shock, while normal coronary vessels made ischemia unlikely.

Treatment is mainly supportive; diuretics, digoxin, ace inhibitor and spironolactone may provide symptomatic relieve. In some patients, steroid may hasten recovery. For patients without reasonable response despite adequate medical management, a VAD may be employed. However, definitive therapy is cardiac transplant. Most described cases end in sudden cardiac death, but our patient survived because of early implantation of a VAD.

There is not enough literature to explain why lymphocytic myocarditis causes fulminant cardiac dysfunction or sudden cardiac death. Hence, we recommend more research on this subject.

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### **Title: Tumor Lysis Syndrome In Metastatic Breast Cancer After Treatment with Paclitaxel**

**Introduction:** Tumor lysis syndrome (TLS) is an oncologic emergency characterized by spillage of intracellular material into the blood caused by disruption of massive load of tumor cells. It is more commonly reported in hematological cancers and can have fatal consequences due to renal and multi-organ failure and arrhythmias due to electrolyte imbalance.

We describe an unusual case with metastatic breast cancer who presented with TLS after a single dose of paclitaxel. As more potent chemotherapy agents are developed, it is imperative to be aware of this potentially fatal phenomenon

**Presentation:** 52 year old female with locally recurrent estrogen and progesterone receptor positive, HER-2 negative left breast cancer status post bilateral mastectomy, was found to have liver and widespread bony metastases despite being on anastrozole treatment. She received single dose paclitaxel chemotherapy, a weekly dose of 80 mg/sq. m., for metastatic breast cancer. One week later, she presented with acute onset confusion and was found to have hypotension, bradypnea and extreme somnolence. Relevant laboratory reports on the day of admission were: potassium 5.7mEq/L, creatinine 3.4mg/dL (baseline <1.0mg/dL), phosphorus 7.1mg/dL, LDH 9390 U/L, Uric acid 11.3mg/dL, AST 12833 U/L, ALT 2864 U/L.

A diagnosis of tumor lysis syndrome was made, complicated with acute kidney and liver injury, pneumonia and metabolic encephalopathy. Patient was started on aggressive fluid resuscitation, antibiotics and rasburicase.

The next day patient's oxygen requirements continued to increase with a possible compromise of the airways, which necessitated intubation. Due to her worsening renal function and electrolyte imbalance, patient required hemodialysis. Her condition improved over the course of her ICU admission with supportive treatment and she was successfully extubated.

**Discussion:** Paclitaxel is one of the most commonly used chemotherapy drug for metastatic breast cancer. Our case is only the second reported case of metastatic breast cancer treated with paclitaxel to develop TLS(1). Tumor lysis syndrome is extremely rare in solid tumors in adults.

TLS is more commonly associated with a large tumor load, metastatic disease and peculiarly with involvement of liver.

TLS in solid tumors is practically always preventable but the mortality rate for mis-diagnosed TLS could be upto 50%. The risk for TLS is often under-estimated in clinical practice.

Appropriate preventive strategies involve ruling out pre-existent electrolyte/renal disorders prior to chemotherapy, close monitoring of high risk patients and adequate hydration.

Once TLS develops, treatment is mainly supportive with adequate fluid resuscitation and hypouricemic drugs.

#### **Keypoints:**

1. TLS should be suspected in patients with solid tumors undergoing chemotherapy who present with acute decompensation.
2. TLS is practically always preventable but difficult to manage
3. Proper and regular monitoring in patients with widespread metastatic disease can avoid chemo-induced TLS.

#### **References:**

1. Ustundag Y et al. Acute tumor lysis syndrome associated with paclitaxel. Ann Pharmacother.1997;31(12):1548-1549.

## Resident / Fellow Clinical Vignette

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**Title: Drunk in the Hospital. Intentional Ingestion of Hand Sanitizer.**

**Introduction:** Patients, families and health care workers are at risk of acquiring healthcare associated infections. One of the methods that have proved efficacious for hospitals are hand sanitizers (HS). Good hand hygiene via HS has been universally adopted due to their easy use, quick result, and eradication of most bacteria that spread infection. Unfortunately, they can also be harmful or even life threatening if they fall into the "wrong hands". We present a case of intentional consumption of HS by a hospitalized patient in order to satisfy alcohol withdrawal.

**Case report:** A 36 year old woman with a past medical history of alcohol abuse, depression, anxiety, and seizures, presented to the hospital for chemical detoxification. During the hospitalization, she was found on the medical floor unresponsive, with one and a half empty bags of HS (total 1500 ml). A nurse reported that the patient had entered the bathroom minutes before the incident. Subsequently she was intubated and transferred to MICU. The examination was normal except for respiratory distress, dilated pupils with a sluggish response and the patient being unresponsive to painful or verbal stimuli. Laboratory tests were notable for serum alcohol level of 545mg/dl. CXR and CT head were unremarkable.

**Discussion:** HS or commercially available alcohol products are generally made up of two different alcohols; ethanol and isopropanol. These products may also contain smaller amounts of hydrogen peroxide, denatonium benzoate, and other products. Life threatening complications may be seen in normal adults who consume as little as 360 ml of alcohol hand rub containing 80% ethanol. Complications of ingestion include: depression of central nervous system followed by aspiration and respiratory arrest. Therapy for severe overdose is mostly supportive (airway protection), with hemodialysis done in some situations for ethanol removal. Fomepizole (competitive inhibitor of alcohol dehydrogenase) is contraindicated in hand sanitizer ingestion since ethanol or propanolol will be prolonged without added benefits.

**Conclusion:** Consumption of HS has increased since it is free and easily accessible to everyone. Some patients even prefer HS over other regular alcoholic beverages, since it gets you drunk faster than other alcoholic drinks. This case illustrates that a good device can be abused. HS should have a safety device to prevent this nosocomial sentinel event. Anti-lock devices and foam base products can help accomplish this goal. High risk patients should also be identified, and restriction to access to HS should be instituted.

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**Title: DISSEMINATION OF INVASIVE ASPERGILLOSIS: DIAGNOSTIC AND MANAGEMENT DILEMMAS**

Aspergillus species are ubiquitous fungus in the external environment and inhalation of infectious conidia is a common event; however tissue invasion is uncommon and occurs most frequently in the setting of immunosuppression. The profile of patients considered immunocompromised continues to expand and despite advances in diagnosis and treatment, invasive aspergillosis remains a highly lethal opportunistic infection.

A 63-year-old female with past medical history of hypertension, diabetes mellitus type II, recently diagnosed autoimmune hepatitis on methylprednisolone for approximately one month and azathioprine for one week, presented with generalized weakness, malaise, confusion, and a productive cough for five days. On examination, the patient was afebrile, tachycardic, tachypnic and mildly hypoxemic. There were bilateral diffuse rales, a holosystolic murmur, grade 3/6, at the left lower sternal border, and the neurological exam was normal except for mild bradykinesia. Laboratory findings were significant for leukocytosis, anemia, thrombocytopenia, and elevated liver function tests. CT of the lung showed multifocal pulmonary opacities in all lobes, some with cavitation. CT of the head showed several hypodense parenchymal lesions. A transthoracic echocardiogram showed no definite evidence of vegetation. The patient was admitted to the intensive care unit (ICU) for severe sepsis secondary to healthcare associated pneumonia versus endocarditis with septic emboli phenomenon and started on broad-spectrum antibiotics. The patient's condition rapidly declined requiring intubation for hypoxic respiratory failure and pressor support for septic shock. Antibiotic coverage was adjusted for the possibility of a resistant bacterial infection, and empiric micafungin was added. A bronchoscopy with bronchial alveolar lavage (BAL) was unremarkable. Final BAL and blood cultures were negative. Despite maximum ICU care, the patient expired on hospital day three. Autopsy revealed invasive fungal hyphae in the lungs, brain, thyroid, heart, and right kidney. A post-mortem fungal culture from the lung grew *Aspergillus fumigatus*.

An immunosuppressive regimen for the treatment of autoimmune hepatitis most likely contributed to the tissue invasion of aspergillosis. Histopathological examination with microbiological confirmation remains the gold standard in diagnosis; however it is often difficult or not feasible to obtain a biopsy specimen. Blood cultures are rarely positive and sputum and BAL cultures are of limited value. Other modalities for diagnosis have been developed and are an area of continued research. Despite improvements in diagnosis, and the advent of newer formulations of amphotericin B and the use of voriconazole, mortality remains high. This case emphasizes the importance in maintaining a high index of suspicion to ensure that the proper workup, diagnosis, and treatment can be initiated earlier in the clinical presentation in an effort to decrease the extremely high mortality of these cases.

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**Title: BRUGADA SYNDROME AND EPILEPSY- TWO SIDES OF THE SAME COIN?**

**INTRODUCTION**

Brugada syndrome (BS) is characterized by specific changes in the electrocardiogram (EKG) and may manifest as syncope, arrhythmia or sudden cardiac death. Epilepsy is a convulsive disorder which has multiple etiologies including genetic abnormalities. We are describing a case in which the patient has features of both these conditions which could represent a new clinical entity.

**CASE DESCRIPTION**

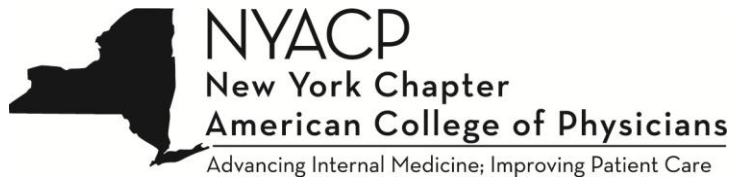
A 41 year old man with a history of epilepsy was brought to the emergency department (ED) after an episode of witnessed seizure and syncope. At the time of presentation, his vital signs were stable and he was alert but disoriented. There was no ongoing seizure activity and no focal neurological deficits were noted. The routine labs including complete blood count, serum electrolytes and troponin level were within normal range. EKG was suggestive of sinus rhythm with 1st degree atrioventricular block and type 1 Brugada pattern in leads V1 and V2. On review of records, we found that he had multiple visits to ED over the last 16 years for episodes of generalized tonic clonic and complex partial seizures. An interesting pattern was observed in his old EKGs, which showed Brugada pattern in leads V1 and V2 after each episode of seizure. Whereas the EKGs obtained on routine office visits showed no evidence of Brugada morphology. He was diagnosed with epilepsy at the age of 25 and underwent left temporal lobectomy a year later. The seizures continued despite the surgery and multiple anti-epileptic drugs. Multiple electroencephalograms were done after the surgery but no epileptiform discharges were ever noted. During this hospitalization, no significant events were noted on telemetry and a loop recorder was placed soon after discharge from the hospital.

**CASE DISCUSSION**

Type 1 (coved type) BS is characterized by ST segment elevation with an upward convexity and an inverted T wave in leads V1-V3. BS and epilepsy are both associated with sodium channelopathies. The cause for BS is SCN5A gene mutation whereas SCN1A gene mutation is associated with epilepsy. The appearance of brugada pattern on EKG only at the time of seizure activity could be explained in several ways. Seizures in the absence of an epileptogenic focus in our patient may be due to cerebral hypoperfusion secondary to an arrhythmia in the setting of BS. However, an implantable loop recorder has documented normal sinus rhythm prior to and during his episodes of seizure. Hence a more interesting and likely possibility is the presence of a unique sodium channelopathy affecting the myocardium and neurons simultaneously.

**CONCLUSION**

An EKG must be obtained in patients with seizure to rule out arrhythmogenic convulsive syncope. Further genetic research may isolate a common sodium channelopathy for BS and epilepsy.



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## Resident / Fellow Quality/Patient Safety and Outcomes Measurement

<p><b>Author:</b> Eric Karu MD  <b>Additional Authors:</b> Vahe Shahnazarian M.D. Parag Mehta M.D.  <b>Institution:</b> New York Methodist Hospital</p> <p><b>Title: DEVELOPMENT AND IMPLEMENTATION OF A NEW PHYSICIAN HANDOFF TOOL IN THE DEPARTMENT OF MEDICINE</b></p> <p><b>Purpose:</b>  To create and implement an electronic physician handoff system in order to improve patient safety as well as resident physician sign-out satisfaction.</p> <p><b>Methods:</b>  The information technology fellow and house staff team leaders developed and distributed a survey to the first year resident physicians while coordinating development efforts between the house staff and IT team. A physician handoff was developed based on the results of that survey, and it was implemented on February 11, 2013.</p> <p><b>Results:</b>  The newly implemented handoff was a success, based on the initial survey responses of 24 Internal Medicine residents:</p> <ul style="list-style-type: none"> <li>- While 91.3% of residents were not using the old handoff system, 83.4% were using the new system.</li> <li>- A majority of residents were dissatisfied with the old handoff, while a majority of residents were satisfied with the new system.</li> <li>- Residents were especially satisfied with the "free text" and "comment" sections of the new handoff.</li> <li>- 83.33% of residents stated that the new system saved them time when compared to the old system.</li> </ul> <p><b>Conclusion:</b>  The residents were not satisfied with the old handoff tool, which caused non-compliance and had residents creating their own non-standardized templates. In order to increase compliance, and in turn, increase both the quality of physician handoffs as well as patient care, a new handoff tool was implemented based on the residents' needs. Since its implementation, numerous changes have been made to further increase resident satisfaction with the system. With the implementation of this standardized, mandatory handoff tool, resident satisfaction has increased with not only the format of the tool itself, but also with the quality of peer sign-outs, which is the most important benefit of all.</p>	<p><b>Author:</b> Eric Karu MD  <b>Additional Authors:</b> Parag Mehta M.D.  <b>Institution:</b> New York Methodist Hospital</p> <p><b>Title: Reducing Unnecessary Blood Glucose Testing at a Community Hospital to Save Costs While Preserving Patient Safety</b></p> <p><b>Purpose:</b>  To reduce unwarranted blood glucose testing in order to decrease costs and lessen the burden of wasted time and energy on the medical staff. There were approximately 500,000 blood sugar testing done per year at New York Methodist Hospital</p> <p><b>Methods:</b>  The pattern of ordering blood sugar was studied. A consensus and a guideline was developed for the appropriate testing of blood sugar, through appropriate multidisciplinary committees. Nursing, pharmacy, hospital administration and the information technology department were involved. Afterwards, information was disseminated via emails, meetings, and was reinforced by daily rounds. A rule and order set were created via the Cerner electronic medical record. It was decided that health care providers may only place chem strip orders only on patients with a definitive diagnosis of diabetes mellitus. The frequency of blood glucose monitoring was adjusted to twice a day, before breakfast and before dinner in a controlled diabetic patients. The medicine residents, RNs, MAs, and Unit Clerks were all educated through meetings and one to one encounters. The total number of chem strip use was electronically charted and separated by hospital location.</p> <p><b>Results:</b>  The results demonstrated that we had achieved our goal of reducing the number of unnecessary blood glucose testing. From March of 2014 to May of 2014 the number of finger sticks had decreased from 39,201 to 34,396 which equates to a difference of 4,805.</p> <table style="margin-left: 20px;"> <tr> <td>Mar-14</td> <td>39,201</td> </tr> <tr> <td>Apr-14</td> <td>37,506</td> </tr> <tr> <td>May-14</td> <td>34,396</td> </tr> </table> <p><b>Conclusion:</b>  There's absolutely no question that patients with a diagnosis of diabetes mellitus should have the blood sugar checked regularly to prevent complications. The New York Methodist Hospital medical staff was educated on the appropriate use of blood glucose testing on patients. By the simple creation of an order set, placing a simple rule via the EMR, and educating the medical staff, we had managed to curtail the unnecessary testing by a significant amount without compromising patient safety. This represents a reduction in costs of approximately \$30,000 a year in medical supplies (Chem Strip, Lancets, Alcohol Pads) and a reduction of 5,000 man hours (or two full time employees). In addition to reduced costs to the patient and hospital, there's a reduction of pain, reduction of inconvenience, while preserving patient satisfaction.</p>	Mar-14	39,201	Apr-14	37,506	May-14	34,396
Mar-14	39,201						
Apr-14	37,506						
May-14	34,396						

## Resident / Fellow Quality/Patient Safety and Outcomes Measurement

<p><b>Author:</b> Isabel Preeshagul MD  <b>Additional Authors:</b> Daniel Steinberg  <b>Institution:</b> Beth Israel</p> <p><b>Title: UNDERSTANDING TRIAGE CHOICES FOR PATIENTS WITH DIABETIC KETOACIDOSIS ADMITTED FROM EMERGENCY DEPARTMENTS IN NEW YORK STATE</b></p> <p><b>Purpose of study:</b>          No published guidelines exist directing where in the hospital to admit diabetic ketoacidosis (DKA) patients. Large variation exists across New York state (NYS) in how often patients are triaged to the wards versus the ICU. This variation may be explained by substantial differences in protocols for care and available resources across institutions.</p> <p><b>Methods:</b>          We created an eleven question multiple choice survey pertaining to the management and triage of DKA patients. The same investigator (IP) phoned each hospital three times and requested answers to the scripted survey from an ED physician director, charge nurse, ED physician or ED nurse. We targeted hospitals in NYS for which risk-adjusted rates of ICU utilization for patients with DKA are known.</p> <p><b>Results:</b>          We identified and attempted to survey 146 hospitals. For each site, 3 calls were made and we obtained a total of 23 responses (15.8%). Of the 23 hospitals providing data, nearly all (82%) had a protocol for DKA management in the ED. Most (82%) used continuous intravenous (IV) insulin infusions with a bolus for the mainstay of therapy, as opposed to exclusively bolus administration. All hospitals permitted insulin infusions in the ED and in the ICU. The majority (59%) of the institutions had step down units, but less than half of these institutions (41%) permitted insulin infusions in these units. Only 9% of hospitals permitted insulin infusions on a telemetry unit and 5% on general wards. The greater part of the hospitals (55%) had a protocol for triage of DKA patients. The majority of institutions (64%) held patients in the ED until closure of their anion gap.</p> <p><b>Conclusions:</b>          Our data provides useful insight into the triage and management of DKA patients statewide. Of the institutions surveyed, most had a protocol for DKA triage. The majority did not allow patients to be admitted to non-ICU locations prior to closure of the anion gap. Our data suggest that many EDs indiscriminately triage patients with DKA to the ICU despite prior data suggesting that care for patients with DKA outside of the ICU is safe. Future research examining DKA triage from the ER to the hospital is warranted to understand how to optimize triage protocols, improve resource allocation and most importantly enhance care for DKA patients on the general wards.</p>	<p><b>Author:</b> Tarak Rambhatla MD  <b>Additional Authors:</b> Andrew Miller MD; Bsuhra Mina, MD.</p> <p><b>Institution:</b> Lenox Hill Hospital, Department of Medicine</p> <p><b>Title: Central venous catheter removal: a procedure well performed?</b></p> <p><b>Background:</b>          Central venous catheterization (CVC) is performed more than 5 million times a year in the US. The complications of CVC insertion and removal are well known but formal training is typically provided only for CVC insertion and minimal emphasis is placed on CVC removal. Venous air embolism and expanding hematoma are well known complications of improper CVC removal and have been known to cause hypoxemia, airway compression, and even death.</p> <p><b>Methods:</b>          46 internal medicine residents at a large academic medical center were surveyed. Residents from all three PGY levels were included. A survey consisting of open-ended questions was used to assess the technique used by residents in performing CVC removal and their knowledge of potential complications of the procedure. Study authors coded descriptions to normalize the responses to the open ended questions (e.g. "patient placed in head down position" was coded as "Trendelenburg")</p> <p><b>Results:</b>          46 internal medicine residents were surveyed, 18 PGY1s, 14 PGY2s, and 14PGY3s. 18 PGY1s, 9 PGY2s, 10 PGY3s listed hematoma as a potential complication; 1 PGY1, 1 PGY2, 4 PGY3 reported they check coagulation parameters prior to CVC removal; 12 PGY1s, 12PGY2s, 12 PGY3s reported holding pressure after removal; 8 PGY1s, 8 PGY2s, 10 PGY3s reported checking the removal site within 30 minutes after removal. 2 Residents (both were PGY3) reported performing all three steps. 11PGY1s, 9 PGY2s, 8PGY3s listed venous air embolism as a potential complication; 11 PGY1s, 11PGY2s, 10PGY3s reported placing the patient in Trendelenburg or supine prior to CVC removal; 14 PGY1s, 10PGY2s, and 9 PGY3s listed patient breathing as an area of focus, and less than half of all PGYs reported asking the patient to perform the Valsalva maneuver; 11 PGY1s, 6 PGY2s, and 8 PGY3s reported using an air occlusive dressing to dress the site. 39% of PGY1s, 36% of PGY2s, and 29% of PGY3s reported performing all three maneuvers (Trendelenburg, focus on breathing, air occlusive dressing).</p> <p><b>Conclusions:</b>          The medicine residents surveyed do not perform CVC removal in a manner that optimizes patient safety. Nearly all residents are aware of hematoma and venous air embolism as major complications associated with CVC removal, yet many do not properly perform key steps in the procedure that would minimize those risks. The lack of awareness of these serious and often preventable complications of improper CVC removal needs to be addressed by training programs and training in this procedure should be included into the curriculum.</p>
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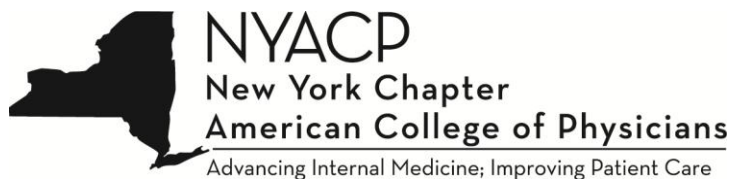
# Resident / Fellow Quality/Patient Safety and Outcomes Measurement

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M.D.</p> <p><b>Institution:</b> Jacobi Medical Center/Albert Einstein College of Medicine</p> <p><b>Title: IMPROVING MEDICINE RESIDENTS' PRACTICES ON ADULT SCREENING AND IMMUNIZATION</b></p> <p><b>Purpose:</b>          We aimed to assess the effect of production of a health maintenance (HM) card for distribution to the medicine residents and posting within each adult primary clinic exam room on knowledge of and compliance to screening and immunization national recommendations.</p> <p><b>Methods:</b>          In December 2013, an anonymous online questionnaire was sent to medicine residents including 11 multiple choice questions; each focused on a certain screening or immunization task, stratified by PGY level. Pocket-size and office-size HM cards including up to date screening and immunization national recommendations were distributed to residents and also posted within each clinic exam room. A year after distribution of cards, the same questionnaire was sent to residents in January 2014. Scores derived from this questionnaire at 2 time points were compared and stratified by PGY level, to assess the improvement of residents' knowledge. In addition, 100 patient charts were reviewed from the year before and after implementation of HM cards. The number of documented HM items were compared to evaluate the improvement in residents' compliance.</p> <p><b>Results:</b>          Overall, both knowledge of and compliance to recommendations significantly increased a year after implementation of HM cards. There was an increase of 18% in questionnaire scores and an increase of 13% in documentation of HM items overall and for all PGY levels. Univariate analysis of residents' knowledge showed a significant increase in rate of hyperlipidemia screening (28%, p&lt;0.0001), diabetes mellitus screening (13%, p&lt;0.01), cervical cancer screening (51%, p&lt;0.0001), breast cancer screening (20%, p&lt;0.0001), abdominal aortic aneurism screening (28%, p&lt;0.0001), osteoporosis screening (16%, p&lt;0.01), influenza immunization (15%, p&lt;0.01) and Tdap immunization (11%, p&lt;0.01). Overall number of documented items was also increased; however, this was only statistically significant for colorectal cancer screening (19%, p&lt;0.0001).</p> <p><b>Conclusions:</b>          In this single-center study at Jacobi Medical Center, implementation of simplified and up to date HM cards had a positive effect on both knowledge and compliance of medicine residents of up to date health maintenance national recommendations. This favorable outcome was not attributable to the natural evolution of advanced residency knowledge and practice over time as significant improvement was seen regardless of PGY level. These measures can be nationally adapted to improve residents' knowledge and patient care in teaching hospitals. Larger multicenter studies are warranted to show the generalizability of our findings.</p>	<p><b>Author:</b> Vahe Shahnazarian MD  <b>Additional Authors:</b> Eric Karu MD (Non-Member), Parag Mehta MD (FACP)  <b>Institution:</b> New York Methodist Hospital</p> <p><b>Title: IMPLEMENTING AN INPATIENT &amp; AMBULATORY HEPATITIS C TESTING PROTOCOL</b></p> <p><b>Purpose:</b> To increase the quality of patient care by meeting hepatitis C testing requirements set forth by the CDC, USPSTF and NY State Law.</p> <p><b>Methods:</b> Approval of the New York Methodist Hospital (NYMH) Institutional Review Board was obtained to begin this study. In conjunction with the Information Technology Services Department at NYMH, 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. The two are essentially the same in that they automatically check the patient's birth year, and if the patient is a "Baby Boomer" (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. The main difference between the inpatient and ambulatory testing is in how often a reminder will pop up for the clinician. In the ambulatory setting, the reminder will occur much more frequently given the shorter nature of the visit. This was implemented on Tuesday, April 15, 2014.</p> <p><b>Results:</b> A total of 3987 patients were included in the study in a three-month period. In December 2013, one month prior to the enactment of the Hepatitis C testing law in NY State, 41.1% of our patients were tested. In the month after the law was implemented (January 2014), we still had a 41.4% rate for patient testing. However, in the month after our testing protocol was implemented, that percentage went up to 46.6% of patients either being tested or being offered testing. When comparing the December group to the Intervention group, the result was statistically significant with a p-value of 0.008; when comparing the January group to the Intervention group, that result was also statistically significant with a p-value of 0.0048.</p> <p><b>Conclusion:</b> Since the implementation of our testing protocol, we have seen an increase in the number of patients being offered Hepatitis C testing (very strong statistical significance with a p-value &lt; 0.01). The protocol has already undergone a number of Plan-Do-Study-Act (PDSA) cycles, whose outcomes we will be studying in the coming weeks. Our ultimate goal with the protocol is to attain 100% offering of testing to our patient population, thereby providing them with the best care possible in this regard. We believe this is possible, but will require further PDSA cycles to accomplish.</p>
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## Resident / Fellow Quality/Patient Safety and Outcomes Measurement

<p><b>Author:</b> Aisha Siraj M.D  <b>Additional Authors:</b> Sridhar S. Chilimuri, M.D</p> <p><b>Institution:</b> Bronx Lebanon hospital</p> <p><b>Title:</b> THE JULY EFFECT: A MYTH OR REALITY?</p> <p>In teaching hospitals the July Effect and cohort turnover are viewed by some experts as having adverse impact on patient outcomes in the month of July. There is a similar perception in the lay public that new residents entering teaching hospitals as being inexperienced leading to cancellation of many elective procedures. In the past few years teaching hospitals have implemented many changes to improve transition of new residents into teaching hospitals. We believe it is time to change public perception of teaching hospitals.</p> <p><b>Objective:</b>To observe trends in monthly mortality rates in a large safety net teaching hospital.</p> <p><b>Methods:</b>We calculated the monthly mortality rates of all admissions to our internal medicine teaching service from 2009 to 2013. During the 5 year study period, there were an average of 1100 admission per month. The average monthly mortality rate was 2.80% (30/1100) with no increase in death rate in July of each year which was 2.78%(32/1153) in 2009, 2.23%(24/1075) in 2010, 1.42%(15/1060) in 2011, 2.25%(25/1110) in 2012, 1.40%(16/1139) in 2013. We did not calculate risk adjusted mortality rates as the case mix intensity of all patients admitted to the service remained relatively constant throughout the year. There were no significant differences in the mortality rates in July compared to other months in a year. In fact we noticed a steady decline rate in July mortality rates in our hospital. In same study period there was a slight increase in case mix intensity for the teaching service.</p> <p><b>Discussion:</b> We found that there was no significant increase in mortality rates in July each of the years during study period. This could be the result of several factors including improved orientation, supervision, implementation of EMR systems with graded controls for new residents, improved patient hand-offs and implementation of patient centered clinical teams.</p> <p><b>Conclusion:</b> Teaching hospitals appear to have improved integration of new residents. The July effect and cohort turnover do not appear to have any adverse impact on patient outcomes. Hospitals should continue to monitor and improve processes to integrate new incoming residents.</p>	<p><b>Author:</b> Mohammad Vaziri MD  <b>Additional Authors:</b> Parag Mehta, MD; Lucille Bael; David Conner MD  <b>Institution:</b> New York Methodist Hospital</p> <p><b>Title:</b> Disparities in Healthcare Delivery: using Core Measures as a tool in reviewing patient data to compare healthcare delivery among high risk patient populations from 2011-2013 at New York Methodist Hospital, Brooklyn, NY</p> <p>A key function of hospital care is to deliver the same quality care to all patients with similar conditions regardless of race, sex or socioeconomic status. The Center for Medicaid and Medicare Services (CMS) and The Joint Commission are assessing quality derived from Core Measures which define a set of healthcare delivery goals in each of several common hospital conditions. Recent disparities report from the Agency of Healthcare Quality Review shows improved but continued disparate delivery of care among some high risk groups, namely Blacks (Blk) Hispanics (Hsp), and low socioeconomic populations.</p> <p>We used data reported to CMS by a trained and validated chart abstraction team for patients admitted from September 2011 to September 2013. Patients had to have either had pneumonia (PNA) exacerbation of congestive heart failure (CHF) or acute myocardial infarction (AMI) on admission. There were 5 performance measures for pneumonia, 6 for AMI and 3 for CHF. We gave those patients who received all the appropriate care as defined by CMS Core Measures guidelines (Perfect Care) as score of 1 and those who did not receive this care a score of 0. Patients were then sorted by race, sex, and socioeconomic status (SES) based on zip code, (Low \$ 0-45K/yr; Mid- 45-85K/yr; High 85+K, as per 2010 US Census Report). An average was then obtained for each group in each of the three core measures we studied.</p> <p>We report on 1,684 patients, 338 who had pneumonia, 670 who had AMI and 676 who had CHF. Percent Compliance with perfect care measures is shown in the below: % Perfect Care, (n):  PNA: White: 92% (171), Blk: 95% (111), Hsp: 90% (39), Other: 94% (17), Male: 93% (133), Female: 92% (205), Low SES: 93% (135), Mid SES: 93% (122), High SES: 88% (76)  AMI: White: 93% (321), Blk: 93% (231), Hsp: 93% (57), Other: 93% (61), Male: 91% (377), Female: 95% (293), Low SES: 94% (322), Mid SES: 92% (218), High SES: 93% (123)  CHF: White: 95% (242), Blk: 94% (318), Hsp: 93% (81), Other: 94% (35), Male: 93% (328), Female: 95% (348), Low SES: 95% (350), Mid SES: 94% (244), High SES: 88% (80)</p> <p>Results show nearly equal care among all races, sex or income status. Some populations like Hispanics (Hsp) in pneumonia arm and high SES in CHF arm had less sample size compared to other groups and this may explain their lower averages. Also this data has not undergone any statistical analysis, so we do not know the statistical power of these results. However, grossly they are optimistic and show how our implementation of standardized procedures and notes in electronic medical records can help eliminate healthcare disparities.</p>
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**Title: Does completing a MOLST prior to terminal admission reduce length of stay and lead to early institution of comfort measures?**

Medical Order for Life Sustaining Treatment (MOLST) is an important document to honor patient preferences for end-of-life care, especially for patients with a terminal diagnosis. We provide a significant amount of end-of-life care at our institution. Therefore, in this study we tried to determine how many of our terminally ill patients have MOLST orders prior to terminal admission, and whether having a MOLST form in the Electronic Medical Records leads to earlier institution of comfort measures and affects total length of hospital stay.

Electronic Medical Records of 112 deceased patients between October 2013 and February 2014 were reviewed. Only patients with a terminal diagnosis i.e. life expectancy of less than 1 year were included. Data regarding patients' age, sex, life limiting diagnosis, total length of stay (LOS), length of stay in Intensive Care Unit (ICU), and length of stay under comfort measures was collected. Comparison between the two groups (MOLST vs. no MOLST) for continuous variables (age, LOS, LOS ICU, % of LOS in comfort measures) was done using the non-parametric Wilcoxon Rank Sum test. The comparison for sex was done using 2x2 Chi-Square.

MOLST completion rate at our institution was 46%. Patients with severe COPD and metastatic disease had the highest and the lowest rates of MOLST completion (64% and 31%) respectively. Patients with a MOLST form were older than patients without MOLST (76.8 +/- 12.7 vs 71.1 +/- 12.9 p=0.03). Although there was an absolute difference in the median length of stay between the two groups (6 days for the MOLST group vs 7 days for non MOLST), statistical significance could not be achieved (p=0.15). The MOLST group spent a greater proportion of their hospital stay on comfort measures versus non MOLST group (23% vs 18%; p=0.20). Patients with a prior MOLST form were less likely to stay in the ICU than patients without a MOLST form (median of 2 vs 0 days; p=0.02). Hence, our study revealed a shorter ICU stay, and a trend towards earlier institution of comfort measures in patients with MOLST forms and possibly better care at the end of their lives.

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**Additional Authors:** Sandhya Manohar MD, Melissa Aleta MD, Linda Williams MD, Prasanta Basak MD, Stephen Jesmajian MD

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**Title: ELEVATED TROPONINS IN CRITICALLY ILL PATIENTS**

Cardiac troponin I (cTnI) is a myocardial contractile protein, the plasma levels of which are increased after myocardial damage. Troponin elevations are common in critically ill patients, and often are associated with an adverse prognosis. Whether this effect is related to the severity of the underlying disease process or to primary cardiac involvement per se, is unclear. We investigated whether cTnI elevations are independently associated with in-hospital mortality in patients admitted to the intensive care unit (ICU).

**METHODS**

We conducted a retrospective chart review for 1 year of all patients admitted to the ICU. We sought to observe the prevalence of mortality among patients with normal and elevated cTnI. APACHE II score, and use of any inotropes, sedation and ventilator support were also recorded. Inclusion criteria included patients admitted to the ICU with cTnI measurement and EKG recording within the first 24 hours of admission. Patients diagnosed with acute coronary syndrome, those who underwent major surgery within one month prior to admission or cardio pulmonary resuscitation prior to admission, were excluded. Continuous variables were presented as mean values. The student T-test was used to observe for any difference between groups.

**RESULTS**

A total of 145 patients were included in the study. There was no difference of APACHE II scores at baseline for both groups (p=0.48). The prevalence of elevated cTnI was 15%. Overall mortality rate was 28% and 48% for those with normal cTnI and elevated cTnI, respectively.

Four different group combinations were investigated and compared for mortality. Group 1 low APACHE II score and normal cTnI; Group 2 low APACHE II score and elevated cTnI; Group 3 high APACHE II score and normal cTnI; and Group 4 high APACHE II score and elevated cTnI. The mortality rate for Group 1, Group 2, Group 3 and Group 4 were 13%, 30%, 35%, and 58% respectively.

There was a trend towards statistical significance with inotrope support (29% in normal cTnI group vs 41% in elevated cTnI group, p=0.0752). There was no difference between groups for ventilator support (59% vs 68% p=0.1862) and sedation (25% vs 27% p=0.7471).

**CONCLUSION**

Our findings demonstrate that in critically ill patients, troponin elevation is associated with a reduction in survival, even after adjustment for severity of illness. Troponin measurements in critically ill patients can be a useful prognostic marker. Elevated cTnI in patients on pressor support in the absence of overt myocardial dysfunction could portend a bad prognosis. Further studies are necessary to explore other specific subsets of patients and the relation to cTnI elevation. Routine measurement of cTnI in critically ill patients could allow for development of new strategies for evaluation and treatment.

## Resident / Fellow Research

<p><b>Author:</b> Kunal Brahmhatt MD  <b>Additional Authors:</b> Sonya Gadhvi, MBBS, Zack Williams, MD, Juan Gaztanaga MD, Shahidul Islam MPH, Kevin Marzo MD, and Beevash Ray, MD  <b>Institution:</b> Winthrop University Hospital</p> <p><b>Title: CAN CORONARY COMPUTED TOMOGRAPHY ANGIOGRAPHY (CCTA) BE UTILIZED TO DECREASE LENGTH OF STAY FOR ACUTE CHEST PAIN PATIENTS?</b></p> <p><b>PURPOSE:</b> Early CCTA for patients presenting with acute chest pain with low to intermediate likelihood of Acute Coronary Syndrome (ACS) has been shown to be safe and efficient in clinical decision making compared to stress testing. Patients with low to intermediate suspicion of ACS evaluated by CCTA for acute chest pain will have reduced length of hospital stay (LOS), and thereby lower cost, compared to patients undergoing Nuclear Stress Testing (NST).</p> <p><b>METHODS:</b> In an observational, retrospective study, we collected data for 48 consecutive patients who underwent CCTA evaluation for ACS and 67 consecutive patients who had undergone NST, but had met criteria for CCTA. Demographic data, cardiac risk scores and cardiac risk factors were collected on all patients. The primary clinical outcome was LOS between subjects undergoing CCTA versus NST (Non-CCTA). Secondary outcomes include incidence of invasive coronary angiography (ICA), percutaneous coronary intervention (PCI), or coronary artery bypass grafting (CABG) in each group. Wilcoxon rank-sum test and Fisher's exact test were used for univariate analysis. Multiple linear regression was used to perform an adjusted multivariable analysis.</p> <p><b>RESULTS:</b> Baseline demographics and cardiac RF were comparable between 2 groups. Non-CCTA group had longer LOS compared to CCTA group (70±177;60 hours vs 15±177;16 hours, p&lt; 0.0001). Unadjusted analysis of LOS, showed group, age, history of hypertension, history of diabetes mellitus, ICA and CABG were significant factors. A multiple linear regression model was developed (Adjusted R-square=0.56) to detect independent predictors of LOS. Independent factors for longer LOS include group, Non-CCTA versus CCTA (coefficient= -1.6, p&lt;0.0001) and history of diabetes mellitus (coefficient 0.53, p=0.0063). CCTA reduced LOS by 80% compared to the non-CCTA group. Age did not contribute towards LOS in adjusted model. In comparison, the incidence of ICA was lower in the CCTA group (4% vs 15%, p=0.073) while the incidence of PCI and CABG was comparable between 2 groups.</p> <p><b>CONCLUSION:</b> Patients at low to intermediate risk of ACS can be evaluated by CCTA to reduce LOS. A limitation to our study was the small number of CCTA patients in our single center registry, hence a larger cohort is needed to strengthen the validity of our results.</p>	<p><b>Author:</b> Abhishek Sharma MD  <b>Additional Authors:</b> Ajay Vallakati, MD; Andrew J. Einstein, M.D., Ph.D, FACC; Carl J. Lavie, M.D., FACC, FACP, FCCP Armin Arbab-Zadeh MD, Ph.D, FACC; Francisco Lopez-Jimenez, MD, MSc, FAHA, FACC; Debabrata Mukherjee, MD, MS, FACC; Edgar Lichstein, MD, FACC</p> <p><b>Institution:</b> Maimonides Medical Center</p> <p><b>Title: Relationship of Body Mass Index with Total Mortality, Cardiovascular Mortality and Myocardial Infarction after Coronary Revascularization: Evidence from a Meta-analysis of 257,333 patients from 36 studies</b></p> <p><b>Background:</b> Clinical studies have shown conflicting results linking obesity with worse clinical outcomes after coronary revascularization procedures (coronary artery bypass grafting [CABG] and percutaneous coronary intervention [PCI]). We aim to investigate the relationship of body mass index (BMI) with total mortality, cardiovascular (CV) mortality and myocardial infarction (MI) after coronary revascularization procedures.</p> <p><b>Methods:</b> A systematic search of studies published between 1966 to May 2013 was conducted using Pub Med, CINAHL, Cochrane CENTRAL and the Web of Science databases. We identified studies reporting rate of myocardial infarction, cardiovascular mortality and total mortality among CAD patients postcoronary revascularization procedures in various BMI categories [&lt;20 (underweight); 20-24.9 (normal reference); 25-29.9 (overweight); 30-34.9 (obese); ≥35 (severe obese)]. Event rates were compared using a random effects model assuming inter-study heterogeneity.</p> <p><b>Results:</b> Two study authors (AS, AV) independently reviewed the 324 articles obtained in the search and identified 36 (12 CABG; 26 PCI) studies with 257,333 patients for final analyses. After a coronary revascularization procedure, the risk of total mortality, CV mortality and MI was highest among patients with low BMI (RR 2.59 [95% CI 2.09 - 3.21]; 2.67 [95% CI 1.63 - 4.39], 1.79 [95% CI 1.28 - 2.50] respectively) at the end of mean follow up period of 1.7 years. Risk of CV mortality was lowest among overweight patients (RR 0.81 [95% CI 0.68 - 0.95]). Increasing degree of adiposity as assessed by BMI had a neutral effect on risk MI (RR (0.92 [95% CI 0.84- 1.01]), (0.99 [95% CI 0.85 - 1.15]), and (0.93 [95% CI 0.78 - 1.11]) for overweight, obese and severe obese, respectively).</p> <p><b>Conclusion:</b> After a coronary revascularization procedure, the risk of total mortality, CV mortality and MI was highest among underweight patients as defined by low BMI, and CV mortality was lowest among overweight patients. Further, prospective studies are needed to investigate this association and explore potential underlying mechanisms for this obesity paradox.</p>
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## Resident / Fellow Research

<p><b>Author:</b> Vasvi Singh  <b>Additional Authors:</b> Saurav Luthra MD; Ruth Kouides MD; Nathan Ritter MD, Ryan Hoefen MD  <b>Institution:</b> Unity Health System</p> <p><b>Title: PERSONALIZED PATIENT EDUCATION HANDOUTS IMPROVE UNDERSTANDING OF HEALTH STATUS AND MOTIVATION</b></p> <p><b>Purpose of Study:</b>          Nearly 40% of mortality in the United States is related to social and behavioral factors such as smoking, diet and sedentary lifestyle. Research demonstrates increased motivation when individuals perceive greater autonomy, competence and relatedness to a goal. We hypothesized that giving patients a personalized handout with educational information tailored to their health data would result in a more accurate sense of their health status and opportunities for improvement, thereby increasing their motivation to engage in healthier lifestyle habits.</p> <p><b>Methods:</b>          After IRB approval, consecutive presenting to a cardiology office were asked to participate and those who agreed were given a pre-visit survey to gauge perceptions of their health status and motivation to make behavior changes. Alternate subjects were assigned to the Experimental and Control groups. Experimental Subjects were given a personalized health summary with graphics indicating current risk factor status and short text summaries with advice on health improvement. These summaries were generated based on basic patient characteristics (age, height, weight, blood pressure, smoking status, lipid levels, fasting plasma glucose, kidney function, diet and activity level) and were reviewed with the cardiologist during the visit. The Control subjects had a routine office visit and were given standard patient education handouts at the physician's discretion. At the end of their visit, all subjects received a post-visit survey to assess changes in health perceptions and motivation.</p> <p><b>Results:</b>          77/79 patients agreed to participate and completed pre-visit and post-visit surveys. The Experimental Group (N=39) and Control Group (N=38) had mean ages (+/-SD) of 66.6 (+/-15.2) years and 64.2 (+/-12.8) years (p=0.46), and female percentage of 35.9% and 71.0% (p&lt;0.01), respectively. Subjects who received personalized handouts changed their responses to more questions regarding their current health status than those with routine office visits (28.4% vs. 12.3%, p&lt;0.01) suggesting they gained a more accurate insight into their health. On questions regarding "perceptions of health", 31.6% of Experimental vs. 14.9% of Control group (p&lt;0.01) changed responses. On questions regarding "motivation to improve health", Experimental Subjects were also more likely to indicate increased motivation following their office visit (30.5% vs. 12.8%, p&lt;0.01), as well as improved knowledge on how to improve their health (26.8% vs. 11.1%, p&lt;0.01).</p> <p><b>Conclusions:</b>          Use of a personalized patient education handout resulted in more accurate health perception, increased motivation to make healthy changes, and a better knowledge of how to go about making those changes. Further studies will be needed to determine whether the intervention results in behavior change and improved health outcomes.</p>	<p><b>Author:</b> JAYANTHI VIJAYAKUMAR MBBS  <b>Additional Authors:</b> Wei Tan, MA*; Nikhil I .Khushalani, MD*          *Roswell Park Cancer Institute, Buffalo, New York</p> <p><b>Institution:</b> UNIVERSITY AT BUFFALO, Department of medicine</p> <p><b>Title: METFORMIN USE AND PROGNOSIS IN MELANOMA</b></p> <p><b>PURPOSE FOR STUDY:</b> In pre-clinical studies metformin has been shown to inhibit proliferation of various cancer cell lines including melanoma. The anti-melanoma effects of metformin are mediated through p21 and adenosine monophosphate (AMP) independent cell cycle arrest, apoptosis, autophagy, mitochondrial damage and oxidative stress. The clinical effects of metformin in melanoma are not known. We sought to examine the prognostic effect of metformin use in diabetic subjects who developed melanoma with our hypothesis being that metformin users would have superior outcomes to those not on metformin.</p> <p><b>METHODS:</b> This is a retrospective review of melanoma patients in the Clinical Data Network at Roswell Park Cancer Institute from 2000-2012. Organ transplants recipients or those with other malignancies were excluded. Of 3605 total patients, 171 unique patients with diabetes mellitus and melanoma were identified. Standard demographics, including AJCC (American Joint Committee on Cancer) pathologic staging were recorded. Survival analysis was calculated using Kaplan-Meier method. The difference in survival between metformin users and non-users were done using log rank tests.</p> <p><b>RESULTS:</b> Median age of patients was 66.5 years (range, 29, 95). Median overall survival was 73.6 months (95% CI, 52.9, 107.3) in metformin users versus 70.5 months (28.2, 79.5) in non-users (p=0.14). Recurrence-free survival (RFS) was similar between the two groups (p=0.15). When analysed by pathologic stage, metformin use was associated with better survival among patients with stages I and III melanoma. Median survival was superior in stage I melanoma patients on metformin compared to patients without metformin, [median not reached, NR (95% CI, 86.0, NR) versus 116.6 months (76.8, 116.6) respectively; p=0.03]. Similarly, stage III melanoma patients on metformin had a better median survival compared to patients not on metformin [53.9 months (16.2, 73.6) versus 26.7 months (7.5, 50.4); p=0.03]. A similar effect was not seen in stage II patients (p=0.42). There was a trend towards improved RFS in stage III patients on metformin (median RFS 37.6 months versus 15.4 months, p=0.07). Tumor BRAF status did not impact survival in this analysis.</p> <p><b>CONCLUSION:</b> In this study, we show that metformin use is associated with superior survival in stage I and III melanoma. Prospective trials should examine the role of metformin as adjuvant treatment in stage III melanoma who are at high risk for relapse.</p>
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