

# New York Chapter ACP

Resident and Medical Student Forum

Saturday, November 14, 2015

SUNY Upstate Medical University  
Institute for Human Performance  
505 Irving Avenue  
Syracuse, NY 13202

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

**Medical Student  
Clinical Vignette**

## Medical Student Clinical Vignette

**Author: Rebecca Abi Nader**

Additional Authors: Adewale Olajubelo MD, Avery Davis( MS III), Omar Guitierrez MD  
Institution: Kingsbrook Jewish Medical Center

### **Title: Unexplained Acute Abdominal Pain- Appendicitis Epiploicae**

Acute abdominal pain lends itself to various etiologies depending on the location, character, and consistency of the pain.

Appendicitis epiploicae is an uncommon cause and can present with symptoms mimicking ovarian cyst, appendicitis, diverticulitis or ectopic pregnancy especially in a young woman of child bearing age.

A 26 year old female presents to the ED with complaints of acute abdominal pain for 4 days. Pain was located in the upper quadrant and radiates to the left lower quadrant and flank. The pain was sharp and 8/10 in severity. Pain is alleviated by lying down and exacerbated by movement. Associated symptoms include nausea, loss of appetite and constipation. Her past medical history was significant for mild asthma which is controlled with albuterol. She has no pertinent surgical history or family history. She takes no medications. She is allergic to penicillin and has anaphylactic reaction if used.

She is G0P0, not sexually active and has no history of STDs. Her LMP was on 06/14/15 with regular cycle lasting 3 to 4 days. Review of systems was significant for fatigue, and decrease frequency of urination. At the time of presentation, she was in mild distress but alert, awake, oriented to place, time and person. Vital signs were within normal limits. Physical exam shows hypoactive bowel sounds with positive rebound tenderness and guarding of the left upper quadrant. Laboratory studies reveals negative beta HCG, low: MCV (75.3), MCH (23.2) MCHC (30.8), and platelets (115). RDW was high at (15.1). These values were not specific enough to rule in any particular cause of these patient's symptoms. Urinalysis was significant for WBC >20 with positive cocci. A radiologic study was done to rule out other possible etiologies. CT of the abdomen without contrast reveals the following:

- An isodense soft tissue nodule anterior to the spleen measuring 1.6 cm typical for accessory spleen
- Bowel shows small foci of infiltration adjacent to distal descending colon with small amount of fluid in paracolic gutter. The right quadrant inferior to the cecum has a second pocket of infiltrative fluid. Appendix was unremarkable, terminal ileum is intact and no bowel dilatation.
- Fluid density structure and dilated fallopian tube typical for hydrosalpinx

Radiological studies confirmed a diagnosis of Appendicitis epiploicae. She was managed with normal saline, Ketorolac 30mg for pain and 4mg of zofran for nausea. She was discharged home with TMP/SMX, Percocet and Ibuprofen with instructions to follow up at the clinic in 1 week

Appendicitis epiploicae presentation is based on where the epiploic tissue is inflamed usually by the sigmoid colon. Initial management is often conservative through symptomatic management; therefore it should not be confused with other causes of acute abdomen thereby preventing unnecessary intervention.

**Author: Brandon Brown**

Additional Authors: Vadim Zarubin, MD; In-suk Seo, MD; Kevin Tsai, MD; Elizabeth Guevera, MD  
Institution: The Brooklyn Hospital Center

### **Title: Combined Subcutaneous, Intra-abdominal and Thoracic Splenosis: A Noninvasive Diagnosis in the Age of Nuclear Medicine**

Splenosis is a benign entity whereby splenic tissue autotransplants, generally in the abdominal or peritoneal cavity, following spleen rupture or splenectomy. Ectopic splenic implants in the thoracic cavity and subcutaneous tissue are comparatively rare. In the majority of cases, splenosis remains asymptomatic and is diagnosed incidentally. Previously reported cases often are limited to a single compartment, abdominal or thoracic. Moreover, a majority of those involving intra-thoracic or subcutaneous splenosis are histopathologic diagnoses after invasive biopsy. We describe a case of combined intra-abdominal, thoracic and subcutaneous splenosis in a 42-year-old male resultant to a gunshot wound requiring splenectomy 26 years prior diagnosed on nuclear imaging. This case uniquely features vast anatomic disbursement and is only the second such case to be reported. In this report and literature review, we emphasize the need for a high index of suspicion for splenosis in the setting of relevant imaging findings with history of splenic rupture or splenectomy. As the ectopic implants often resemble malignancy on imaging, patients may undergo undue testing including invasive procedures while splenosis may be easily diagnosed on radionuclide scanning

## Medical Student Clinical Vignette

**Author: Daniel Jipescu**

Additional Authors: Barie Salmon MD, Allison Hoyle DO, Tracy Salmon MD, Abdul A. Ameen MD, David Flores MD, Amer Syed MD FACP

Institution: Barnabas Health, Jersey City Medical Center

### **Title: THE UNIQUE CASE OF A LARGE DEBILITATING STROKE IN A YOUNG PATIENT WITH CORONARY ARTERY ECTASIA**

29 yo African American male with PMH of CVA, HTN, CHF, DVT, dilated cardiomyopathy, hypertensive nephropathy and obstructive sleep apnea was brought to emergency department with altered mental status for 4 hours. His co-workers called 911 after they noticed that the patient was not verbally responsive and was staring off into space for a long period of time. They reported that the patient was able to speak for a very short period of time prior to EMT arrival. The EMT personnel however found the patient to be non-verbal. In emergency department, the patient was noted to have memory loss and complained of mild generalized headache. He denied photophobia, blurry vision, dizziness, or seizure. He also denied chest pain or abdominal pain. Family history was significant for early-onset heart disease in his mother and two of her sisters. PE: V/S: R arm BP: 171/121, L arm B/P 161/121, HR: 116, O2sat: 96% RA. RR: 16-35, AAOx3 - with intermittent confusion; HEENT: NC/AT, PERRLA, EOMI. CV: Soft S1, S2, RRR, tachycardic, grade 2 apical and left lower sternal systolic murmur and a grade 2 systolic murmur in the second IC space on the right. Troponin I: 0.21 / 0.2 / 0.14. CT head showed mild diffuse atrophy slightly greater than expected for the patient's age. MRI of head showed acute infarction in the right middle cerebral artery distribution involving the majority of the right temporal lobe. 2D Echocardiogram revealed severe dilatation of all cardiac chambers and severe biventricular dysfunction with EF of 10%. Hospitalization records from a different hospital were obtained. A diagnosis of Coronary Artery Ectasia (CAE) was noted. The CCU team discussed the benefits of an S-ICD with the patient and his family. After the S-ICD placement and comprehensive medical treatment, the patient recovered well.

This is a unique case of a young patient with extensive cardiovascular pathology that presented with a large debilitating stroke. CAE is dilatation of a coronary artery segment to a diameter at least 1.5 times that of the adjacent normal coronary artery. It has an incidence of 1-5% in patients undergoing coronary angiography. More than half of CAE are due to coronary atherosclerosis, but occasionally they are related to other pathological entities. As the first report of coronary dilatation in a patient with syphilitic aortitis, CAE has been observed in association with connective tissue disorders such as scleroderma, Ehlers-Danlos syndrome and polyarteritis nodosa but also with bacterial infections and the Kawasaki disease. In a small percentage of patients CAE can be congenital in origin.

The importance of electronic medical records, interhospital medical records sharing and creating a portable medical record for the patient and family cannot be underestimated in the economics of these cases.

**Author: Daniel Jipescu**

Additional Authors: Bankim Shah MD, Karthik Donuur MD, Hassan Qureshi DO, Abdul A. Ameen MD, Amer Syed MD FACP

Institution: Barnabas Health, Jersey City Medical Center

### **Title: ANOMALOUS LEFT CORONARY ARTERY - DID THE PATIENT TAKE AN ANOMALOUS DECISION?**

68 YO female with PMH significant for hypertension, IDDM, TIA, gastritis, dyslipidemia, paroxysmal A-Fib, and vitiligo was sent to ED by her primary physician because of an abnormal finding on abdominal CT. This showed a possible thrombus in the left ventricle. Patient had been complaining of epigastric pain and exertional dyspnea for a month. V/S: T 97.8, HR 63, BP 158/59, RR 18, O2 Sat 100% RA. Allergies: percocet and codeine. Family history: stroke and diabetes mellitus in her mother. ROS: noncontributory, except epigastric pain of variable intensity, that occasionally was increased by walking. PE: HEENT: NC/AT, PERRLA, EOMI; Neck: supple, no JVD; CV: S1, S2, RRR, no M/R/G; Abd: soft, NT/ND, obese. Troponin I: 0.02/0.03/0.02. EKG showed inferior and anterior myocardial infarct. Nuclear stress test showed fixed perfusion defect throughout the apex and hypoperfusion of the distal anterior wall region, compatible with prior myocardial infarction; a fixed perfusion of the mid to distal inferolateral segment and distal lateral wall, compatible with prior infarction. 2D Echocardiography presented: Apical wall motion abnormality, EF of 72%, and intraluminal thrombus. Cardiac catheterization showed anomalous left coronary artery origin. A single right coronary artery originated from the right coronary cusp; the left circumflex (LCX) originated from the same cusp but a separate ostium; what appeared to be the left anterior descending artery (LAD) originated from the proximal right coronary artery (RCA). The posterior descending artery was noted to have a subtotal occlusion. What was considered to be the LAD was noted to have 90% stenosis. Obstructive disease was noted in the proximal sections of multiple medium size coronary arteries. After thorough consultation with the cardiothoracic team, the patient was recommended for coronary artery bypass graft surgery. The patient discussed with her family and requested comprehensive meetings with the medical and surgical team. In the end the patient decided to choose only medical management of her condition. According to the patient's wishes, she was managed medically and discharged in a well and stable condition.

The incidence of coronary anomalies in patients undergoing coronary angiography varies from 0.64% to 1.3%. The most common coronary anomaly is the separated origin of the LAD and LCX from the left sinus of Valsalva. The second most common anomaly is the origination of the LCX artery from the RCA or right sinus of Valsalva. Anomalous right coronary artery deriving from the left coronary sinus of Valsalva is rare; its prevalence is about 0.17%

We are presenting this case not only for this unique pathology but also to demonstrate, recommend and emphasize on the importance of the outstanding patient-physician interaction.

## Medical Student Clinical Vignette

**Author: Jaimal Johal**

Institution: Touro College of Osteopathic Medicine

### **Title: Aortoiliac occlusive disease initially presenting with arthritic symptoms**

Aortoiliac occlusion (Leriche syndrome) is described as arterial occlusion at the bifurcation of the aorta into the common iliac arteries. It is a condition characterized by the triad of bilateral hip, thigh, and buttock claudication, symmetric atrophy of the bilateral lower extremities due to chronic ischemia, and impotence in men.

A 77 year old Hispanic female with a PMHx of HTN, CVA, DM type II, seizures, and Alzheimer's, initially presented to the ED department with weeks of moderate bilateral knee pain. She was diagnosed with osteoarthritis of both knees and discharged. She presented again to the ED one month later with bilateral knee pain of two months duration accompanied with severe bilateral thigh and leg pain for the past four days, with pain severity of 10/10.

Her temperature at second presentation to ED was 36.8C (98.2F), blood pressure of 181/82 mmHg, pulse of 83/min and respirations of 19/min.

Examination revealed a patient in severe distress, with mottled skin on both feet and legs, cool to touch. Dorsalis pedis and posterior tibial pulses were unappreciated bilaterally. She, however, did not present with buttock or hip claudication bilaterally. Laboratory values were unremarkable except for a creatine kinase level of 1969 U/L.

Arterial Doppler of the lower extremities was performed and found an Ankle Brachial Index of zero, bilaterally. Segmental blood pressure measurements and pulse volume recordings showed flattened waveforms from the ankle to thigh bilaterally, indicating severe flow reduction.

Arterial duplex imaging of the lower extremities found that the right external iliac artery had evidence of complete occlusion. The left superficial femoral artery from mid to distal thigh and left popliteal artery were occluded.

CT with contrast of the pelvis and lower extremities found occlusion of the infrarenal abdominal aorta. There was absent contrast extending below this level into the pelvis and lower extremities. The distal right femoral artery was occluded along with the right popliteal artery. There was absent flow below the level of the right knee. There was complete occlusion of the proximal segment of the left femoral artery. There was absent flow below the level of the proximal left thigh.

The disease had progressed to the point where her legs were unsalvageable because of the increased length of time of misdiagnosis and she underwent a bilateral above the knee amputation (AKA).

This case illustrates the need for a complete workup and accurate diagnosis of aortoiliac occlusive disease, initially disguised as arthritis of the knees. Because of the misdiagnosis on a prior admission, the patient's health deteriorated to the point that she required bilateral AKA that may have been prevented if an accurate diagnosis was initially made.

**Author: Sanjay Karatam MBBS**

Additional Authors: Ahmer Ishtiaq MD, Adewale Olajubelo MD, Jigar Patel MD

Institution: Kingsbrook Jewish Medical Center

### **Title: I Can't Move My Leg-An Interesting Case of Neuromyelitis Optica**

A middle aged Hispanic female who presents with acute onset of hemiparesis with no focal neurological deficit may point towards the diagnosis of multiple sclerosis however other etiologies including autoimmune diseases should not be overlooked.

A 50 year old Hispanic female presents to the ED with a complaint of left side weakness for the past 24 hours. According to patient's son, she had a similar episode a few years ago. Her past medical history is significant for Transverse Myelitis. She was awake, nonverbal, oriented to person and place. She had a slurred speech with difficulty breathing on admission. An ABG, CBC, Chest X-ray (AP), MRI of the brain and CT of the chest were ordered. Results of the chest x-ray revealed a left lung retrocardiac atelectasis with left chest volume loss. ABG revealed respiratory acidosis (pH: 7.12, PCO2: 135, HCO3: 43.9) with metabolic compensation hence the patient was put on BiPAP. She was stabilized and transferred to the CCU where she was intubated and placed on PRVC when progress was very minimal. To rule out other possible etiology for her left side weakness, an RPR and FTS-Ab studies including Hepatitis screen was done. The result came back positive indicating past history of Hepatitis A and Syphilis.

MRI of the spine and brain revealed cervical spine transverse myelitis in the area of C2-C6 which had progressed upwards into the inferior left side of the medulla. This could explain the respiratory suppression observed in this patient. She was started on Corticosteroids for the acute exacerbation of transverse myelitis and Gabapentin for the neuropathy. Patient was unsuccessfully weaned from the Bipap on day 3 of admission and was unable to perform vital capacity. Patient was transferred on day 4 to Columbia University Hospital for further management. She was found to have an anti-aquaporin 4 antibody. NMO-IgG was elevated which is highly specific to Neuromyelitis optica. The presence of myelitis in this patient is a major criteria for the diagnosis of NMO for which NMO-IgG is positive and confirmed with MRI. Treatment for this disease often includes immediate plasmapheresis after steroid management. Therefore it is important to identify the signs and symptoms of NMO because its often confused with Multiple Sclerosis. This case illustrates the possibility of an autoimmune disease in a middle aged female with neurological deficits. In addition, it is a good idea to rule out common etiology associated with patient presentation such as Multiple Sclerosis. However, the LP would not have revealed oligoclonal bodies nor the MRI would have shown demyelination of the white matter. These are important criteria to move onto the uncommon disorders such as Neuromyelitis Optica.

## Medical Student Clinical Vignette

<p><b>Author: John Kim</b> Additional Authors: Dr. Amit S. Dhamoon M.D., Ph.D Institution: SUNY Upstate Medical University</p> <p><b>Title: Efficiency in Lymphoma Diagnosis: FNA vs. Excisional Biopsy</b></p> <p>Background: Fine-Needle Aspiration (FNA) is a relatively safe, minimally-invasive technique used to biopsy lymph nodes. The efficacy of FNA to diagnose cancers is limited by the small amount of tissue that is aspirated and the loss of tissue architecture that is preserved by more invasive techniques, such as excisional biopsy. This case report discusses the diagnostic history of a patient with suspected lymphoma in which both FNA and excisional biopsies were performed.</p> <p>Case Presentation: A 56-year old male with diabetes mellitus presented to the emergency room with a 5 week history of fever, drenching sweats and 40 lb. weight loss. He also experienced fatigue, nausea, constipation and diffuse abdominal pain that was constantly present, but varying in severity. He has no family history of cancer. At the time of admission, he had smoked 2 packs of cigarettes a day for 41 years. On presentation, the patient was febrile and tachycardic. Physical exam was unremarkable, apart from the abdominal exam demonstrating diffuse tenderness to palpation, with hepatomegaly. No cervical, supraclavicular, or axillary lymphadenopathy was found on exam. A CT-scan of the abdomen and pelvis showed multiple hepatic and splenic lesions with bulky retroperitoneal lymphadenopathy and hepatosplenomegaly. Based on these imaging results, an IR-guided Core Needle biopsy of the patient's retroperitoneal lymph nodes was ordered. However, due to a communication error, an FNA of the retroperitoneal lymph nodes was performed instead. A definitive pathological diagnosis could not be made with the FNA sample. The Hematology-Oncology team was consulted to evaluate the patient and a firm cervical lymph node was found on exam. An excisional biopsy was done and a pathological diagnosis of Hodgkin's Lymphoma was made.</p> <p>Discussion: Although both FNA and excisional biopsy were utilized in this patient's care, only the excisional biopsy could delineate lymph node architecture as well as provide a sufficient tissue sample to make a definitive diagnosis. As a consequence of FNA utilization in this case, tissue diagnosis was delayed by one week, increasing length of stay, hospital costs and patient anxiety. Thus, this case is a strong example of why excisional biopsies are generally preferred over FNA in the diagnosis of lymphoma.</p>	<p><b>Author: Jonathan Komisar</b> Additional Authors: Lieter, A. *, Komisar, J. *, Chang, D. *(Co-first authors) Institution: Icahn School of Medicine at Mount Sinai, Department of Hospital Medicine</p> <p><b>Title: Staphylococcus Lugdunensis: The Bad and The Ugly Coagulase-Negative Staph</b></p> <p>Case presentation: A 31-year-old woman with a history of rheumatic heart disease and a murmur presented with two months of intermittent fevers, diffuse malaise and inability to walk. Physical exam revealed a systolic 2/6 murmur heard best at the apex, 3 out of 5 strength with right knee extension and flexion and 3 out of 5 strength with right shoulder abduction. Laboratory studies were significant for a WBC of 13, an ESR of 99 and a CRP of 108. Blood cultures grew <i>Staphylococcus lugdunensis</i> and transthoracic echocardiogram revealed a mobile thickening on the left anterior leaflet. Given her right sided weakness, CT spine was done revealing a right external iliac artery septic embolus, MRI brain showed septic emboli and evidence of cerebritis and meningitis. Due the MRI findings, CT angiogram of the head was done, showing a mycotic aneurysm. Her symptoms improved with nafcillin, but on hospital day 9, the patient's course was complicated by another septic embolic event causing transient dysarthria. The patient subsequently had urgent mitral valve repair surgery. The patient's post-operative course was uncomplicated. She was discharged home a week later on 6 weeks of IV nafcillin due to mycotic aneurysm and septic embolus to her right external iliac artery.</p> <p>Discussion: <i>Staphylococcus lugdunensis</i> is a coagulase-negative staphylococcus (CoNS) that was first described in 1988 and is most commonly known as a skin colonizer. It is unique among CoNS because the majority of cases are community acquired with an unknown portal of entry and a virulence similar to <i>Staph Aureus</i>. It causes an aggressive, often fatal native valve infective endocarditis (IE). In one case review of <i>S. lugdunensis</i> IE, researchers found a 42% mortality rate and more than 30% of patients had septic emboli. In comparison for other CoNS IE patients, mortality rates are approximately 20% and septic emboli rates are approximately 5%. Further differentiating <i>S. lugdunensis</i> from other CoNS IE, medical treatment with antibiotics is not sufficient and in one systematic review, medical treatment alone was an independent risk factor for mortality with an odds ratio of 4.79 (1.16-19.78). In most cases reviewed in this study, the only patients with good outcomes were those where early heart surgery was performed.</p> <p>Conclusions: We classically think of CoNS infections as being of low virulence, but <i>S. lugdunensis</i> is an aggressive cause of native valve IE with high rates of morbidity and mortality. Clinicians should be aggressive in diagnosing and treating patients with CoNS bacteremia because of the risk for virulent bacteria such as <i>S. lugdunensis</i>. They should be especially suspicious of <i>S. lugdunensis</i> when patients have septic emboli and early cardiac surgery should be considered in any patient with <i>S. lugdunensis</i> infective endocarditis as this may improve outcomes.</p>
--	--

## Medical Student Clinical Vignette

<p><b>Author: Ian Kratzke</b> Institution: SUNY Upstate Medical University</p> <p><b>Title: A Stiff Drink</b></p> <p>A 51 y.o. male was brought to the ED by his sister after a night of profuse vomiting. His affect was blunted and he said that he regretted it. What he wasn't ready to say. He was hypertensive but not in acute distress. Physical exam showed no pertinent positives. IV's were placed, fluids were given and blood was taken. He was found to have an anion gap of 35 and an arterial pH of 7.34. The pneumonic "MUDPILES" for High Anion Gap Metabolic Acidosis (HAGMA) likely went through the resident's head. His serum osmolality was found to be 307, which was 14 more than the calculated osmolality of 293. This gap indicated that something else unmeasured was in his blood. These findings led the medical team to a most likely scenario, that this man drank ethylene glycol.</p> <p>The fomepizol was prepared, which would competitively bind the alcohol dehydrogenase that was currently metabolizing the less toxic ethylene glycol down the pathway to the very toxic glycolate and oxalic acid. As the team began to explain to the patient their treatment plan, he finally revealed his story: he had been suffering from progressive insomnia and depression due to the stress of caring for his mother with dementia. As his mental health deteriorated, so did his social life and his partner ended their relationship. He decided to end his life. He found the anti-freeze and tried to drink the 32 ounces he had in his garage. However, he was scared and wanted to calm his nerves first, so he drank half a bottle of whiskey, which unwittingly, acted much like the fomepizol and bound up the metabolizing enzymes, keeping most of the ethylene glycol from being converted to its toxic form. He went to bed hoping to never wake up, but early the next morning after repeated episodes of uncontrollable vomiting, he decided to reach out to his sister for help.</p> <p>During his hospital stay, he was given over 10 rounds of hemodialysis to clear the toxin and temporarily replace his kidneys, as his serum creatinine rose to over 12 with an estimated GFR of 5. He remained anuric for 2 weeks. Over time, the AKI resolved and his physical state improved, as did his mental health and will to live.</p> <p>Ethylene glycol is widely available, affordable and apparently tolerable to the taste buds. Its textbook presentation and effects are readily applicable to real-life clinical scenarios. While fomepizole should be the standard treatment, ethanol may serve as a replacement if necessary. Importantly, neither are an antidote, but rather buy time until hemodialysis can be performed.</p>	<p><b>Author: Karim Lashin</b> Additional Authors: Daniel Jipescu OMS-IV Institution: Nassau University Medical Center</p> <p><b>Title: Right subclavian vein occlusion with venous collateralization in a young adult with Acromegaly</b></p> <p>39 YO Nigerian male with PMH of hypertension and questionable Marfan's Syndrome presented to the ED complaining of generalized body aches for 2 days, burning chest pain radiating down his left arm and headache. Patient was become increasingly short of breath and noticed a left foot drop after walking 3 blocks. On physical examination it was noted: large facial features and hands, fair tone, strength in extremities, and bowing of legs, Ht: 220 cm Wt.: 136.9 kg. Significant laboratory: Trop I 0.072/0.072/0.072, IFG-I: 1085, Total testosterone: &lt;20, GH: 20, ALKP: 159, CK 1035, CK-MB 4.0/3.9/3.3. TTE showed grossly normal systolic function limited by poor endocardial definition and a dilated aortic root, measuring 5.3 cm. CT was done for better evaluation. This showed occlusion of the right subclavian vein/thrombosis with venous collateralization. CT of head without contrast, showed large heterogenous and slightly hyperintense soft tissue pituitary mass (3.9 cm x 4.4 cm x 3.4 cm). The mass extended into the right cavernous sinus and appeared to encase the internal carotid artery, resembling a large pituitary macroadenoma. Smooth corticated bony outgrowths in the left frontal bone were noted.. Patient refused to stay in the hospital, despite encouragement and thorough explanation of risks. Patient was discharged in medically stable condition with Endocrinology follow up.</p> <p>Elevated levels of Growth Hormone (GH) in adults evidence acromegaly, most commonly due to a benign GH-secreting adenoma of the pituitary gland. The leading cause of death in patients with acromegaly is cardiovascular (38-62% of deaths). Increased fibrinogen, tissue plasminogen, plasminogen activator inhibitor and decreased protein S in acromegaly patients may represent a potential hypercoagulable state.</p> <p>We are presenting a unique case of subclavian vein thrombosis with collateralization. Our conclusion and recommendation is: close monitoring and increase education of the patients with acromegaly could save lives.</p>
---	---

## Medical Student Clinical Vignette

**Author: Mallory LeSieur**

Additional Authors: Shalin Kothari, Kan Liu  
Institution: SUNY Upstate Medical University

### **Title: RECURRENT ACUTE HYPERTENSIVE PULMONARY EDEMA (AHPE) IN A PATIENT WITH SEVERE ISCHEMIC CARDIOMYOPATHY**

**INTRODUCTION:** Acute Hypertensive Pulmonary Edema (AHPE) is considered a separate entity from acute pulmonary edema due to cardiogenic shock, acute valvular insufficiency, or acute respiratory distress syndrome. We describe a case of acute pulmonary edema with comparable pathophysiology to AHPE, in the setting of severe ischemic cardiomyopathy.

**CASE PRESENTATION:** A 60 year old female, with a history of severe ischemic cardiomyopathy, presented to the emergency room with acute hypoxic respiratory failure, requiring emergent intubation. Her blood pressure (BP) was 150/90 mmHg (her baseline BP is  $\leq 90/60$ , in the range of 90-100/60-70). Physical exam showed no evidence of peripheral edema, jugular venous distention, or murmurs. A chest radiograph showed diffuse bilateral patchy opacifications consistent with acute pulmonary edema. An echocardiogram showed no acute changes in systolic function or new segment wall motion abnormalities. Cardiac catheterization ruled out new culprit coronary artery stenosis. Left ventricular end-diastolic pressure (LVEDP) was elevated (35mmHg). Treatment with intravenous furosemide and afterload reducers led to effective BP control, and clinical improvement, which allowed for extubation on the second day of hospitalization. Of note, she had similar episodes with acute pulmonary edema, relative hypertension, and negative cardiac catheterizations three times in the past year.

**DISCUSSION:** Although acute heart failure secondary to new myocardial infarction and pump failure was an initial consideration in our patient given her history and presentation, the negative cardiac catheterization and recurrent nature of these events raised a suspicion for non-cardiac causes of pulmonary edema. AHPE was entertained due to the relative difference between her baseline BP and BP during these presentations. For AHPE, it is important to carefully compare BP's during the event with the patient's baseline values, rather than just consider absolute values (1). AHPE has a strikingly sudden onset leading to "flash pulmonary edema" and rapid reversal with appropriate treatment (1), as in our case. AHPE might result from severely increased sympathetic tone stimulated by increasing pulmonary vascular pressures, and that positive feedback occurs because elevated sympathetic tone transfers blood from peripheral veins to pulmonary vessels, further increasing sympathetic stimulation and causing alveolar fluid accumulation. In addition, AHPE is likely to develop in patients with decreased systolic and diastolic capacity to adapt to acute changes in loading (2).

(1) Ford L.E. (2010). Acute hypertensive pulmonary edema: a new paradigm. *Canadian Journal Of Physiology And Pharmacology*, 88 (1), pp. 9-13.

(2) Margulescu, A.D., Rimbasi, R.C., Florescu, M., Dulgheru, R.E., Cinteza, M., Vinereanu, D. (2012). Systemic hypertension: Cardiac Adaptation in Acute Hypertensive Pulmonary Edema. *The American Journal of Cardiology*. 109(10):1472-148

**Author: Paridhi Malik**

Additional Authors: Anupriya Razdan, MBBS  
Prateek Mathur, MS4  
Institution: Kings brook Jewish Medical Centre

### **Title: The Psychiatry of a Pituitary Adenoma**

Pituitary Adenomas are the third most common intracranial neoplasms accounting for about 10% of all intracranial tumors in adults. These most commonly present with high prolactin levels, in 30% of the secreting tumors, and present as galactorrhea in females and gynecomastia and infertility in males. Some other common symptoms include headaches, menstrual cycle changes, behavioral changes, erectile dysfunction and weight changes. Macro adenomas due to their size ( $> 10$  mm) can compress the optic nerve structures which most frequently affects the bitemporal visual fields. Macroadenomas are usually benign and slow growing. We present a case of pituitary macroadenoma presenting with new onset visual hallucinations and memory loss .

We present a 70 year old African American female with no significant past medical or psychiatric history. Patient reported of some decline in memory about 2 years ago, which was followed 6 months later by increasing paranoia, ideas with persecutory themes and increasing suspicion. Patient was later diagnosed about a year after starting her symptoms with pituitary adenoma but refused treatment. Following this her symptoms worsen with increasing paranoia and agitation and ultimately psychiatric hospitalization for worsening psychotic symptoms. During the admission, MRI of head showed pituitary mass measuring 3.9X2.9 cm. Patient was transferred to neurosurgery and operated upon and tumor was resected. Her symptoms partially improved after surgery. Discussion: There exist a considerable overlap in psychiatric and neurological symptoms. Careful assessment should be done to rule out any organic cause of the symptoms before labeling a patient as psychiatric. As in our patient, the timeline for onset of dementia was age appropriate and visual hallucinations may be part of dementia, but a careful assessment showed the existence of an adenoma which could have been the cause of her symptoms, given the rapid onset and severity of symptoms.

The relationship of neuropathology and psychiatric symptoms must be evaluated using the key radiologic imaging tools. The importance of early imaging must be realized in a patient coming in with persistent psychiatric symptoms to rule out any organic causes of the psychiatric manifestations of neuropathology such as tumors. Neuro-imaging can give many clues to any underlying causes for the psychiatric symptoms and must be considered early in the treatment and care of the patient.

## Medical Student Clinical Vignette

**Author: Prateek Mathur**

Additional Authors: Niket Sonpal, MD  
Paridhi Malik, MS4  
Institution: Kingsbrook Jewish Medical Center

### **Title: The Difficile Diarrhea Digoxin Disaster**

The occurrence of adverse drug reactions secondary to digoxin is owed to its narrow therapeutic index; with adverse drug reactions occurring when serum concentrations exceed 0.8 mg/l. Reduced kidney function will cause digitalis to build up in the body rather than be removed normally through urine. Any disorder that disrupt kidney functioning or decreased glomerular filtration rate raise the risk for digoxin toxicity. We present a case of a 71-year-old female who presented with profound digoxin toxicity secondary to dehydration from *C. difficile* (CD) diarrhea.

A 71 year old female with a past medical history of hypertension and atrial fibrillation controlled by metoprolol and digoxin presented to the hospital with altered mental status, severe nausea, vomiting, visual disturbance, and multiple episodes of non-bloody diarrhea per day. The patient recently had a UTI and was exposed to ciprofloxacin while at her rehabilitation center for her recent knee arthroplasty. The patient's physical exam was notable for guaiac negative stools, and her labs demonstrated a potassium level 3.1 and digoxin level of 5.2. EKG demonstrated an ectopic atrial rhythm with prolonged PR interval, ST-T wave abnormalities, and shortened QRS complex. The patient was immediately started on IV hydration, metronidazole and vancomycin combination therapy as empiric therapy given her recent antibiotic exposure. *C. difficile* toxin assay was noted to be positive and after approximately one week the patient's digoxin level returned to normal. Her symptoms also slowly dissipated and her diarrhea also reduced in frequency and began she started having formed stools on day 5 of admission.

It is clear from the above case that digoxin levels are affected by changes in the volume of distribution (VOD). When the VOD is decreased, the relative concentrations of digoxin are higher and in turn toxic. Correcting the VOD through IV hydration and discontinuation of the medication are the hallmarks of therapy. It should be kept in mind by all physicians, that patients presenting with CD infections who are concomitantly on digitalis therapy are at a higher risk of adverse effects. We recommend judicious use of the inotrope and obtaining regular drug levels in patients who present with CD while on digoxin.

**Author: Hamza Murtaza**

Additional Authors: 2- Arunpreet Kahlon, MBBS, Department of Medicine, SUNY Upstate Medical University, Syracuse, NY (member ACP)  
3- Amit S. Dhamoon, MD, Phd, Department of Medicine, SUNY Upstate Medical University, Syracuse, NY  
Institution: SUNY Upstate Medical University

### **Title: When life gives you Lyme- you spread awareness**

Introduction:

Lyme disease is a tick-borne illness which is reaching epidemic proportions in north east region. I am a first year medical student at SUNY Upstate Medical University and want to share my recent first-hand encounter with this disease.

Case:

I am 25-year old male with no significant medical history. Few days ago, I began to experience diffuse joint pain, which I attributed to exercise soreness and ill-postured sleeping. I also became easily fatigued and felt dizzy whenever I stood up, which started worsening progressively. Yet again, I brushed this off as lack of energy due to fasting during the holy month of Ramadan. When a short walk left me out of breath, I became concerned and checked my pulse, which was 30 beats per minute and irregular. I went to emergency room where my EKG revealed intermittent mobitz type II/type III heart block. A temporary trans-venous pacemaker was placed and I was admitted to the cardiac Intensive care unit. I told the team about my recent trip to Acadia National Park in Maine. Although I never noticed any tick bite or rash on my body, they suspected Lyme carditis to be cause of my presentation. PCR and western blot tests came back positive for Lyme disease, and I was started on ceftriaxone empirically. Over the next few days, my heart rate began to improve, and I became independent of the pacemaker in 6 days. I was discharged the following day with PICC line and ceftriaxone for 21 days.

Discussion:

Each year, approximately 30,000 cases of Lyme disease are reported to the CDC by state health departments and are concentrated heavily in the Northeast and upper Midwest, with 96 percent of reported cases occurring in 13 states including New York and Maine. Typical symptoms include a red, expanding rash called erythema migrans(EM), chills, fever, muscle, joint pain and swollen lymph nodes. However, patients may also present with Bell's palsy and arthritis. 1% of Lyme cases present with meningitis or Lyme carditis. My case elucidates that a lack of the hallmark symptom of Lyme disease, the erythema migrans, can often lead to the underestimation and misdiagnosis of other symptoms. Although Lyme carditis is relatively rare, proper recognition is imperative for treatment as complications can be fatal. Patients treated with appropriate antibiotics in the early stages of Lyme disease usually recover rapidly and completely. In addition, it is important to spread awareness of the varying possible effects of Lyme and to encourage protection against it.

## Medical Student Clinical Vignette

**Author: Mayank Ohri MD**

Additional Authors: Abdulfatah A. Osman, MD, Kimberly Barber, PhD

Institution: Genesys Regional Medical Center

### **Title: A Unique Case of An Unruptured Aneurysm of the Right Sinus of Valsalva Presenting Solely as Bradycardia**

#### **DESCRIPTION**

This is a morbidly obese 67-year-old male with Insulin-dependent diabetes mellitus, hypothyroidism, deep venous thrombosis, pulmonary embolism, GERD, arthritis and vertigo who was referred by his PCP to the cardiology clinic for evaluation of a "slow heart rate". He denied any chest pain, shortness of breath, or dizziness.

His physical exam was unremarkable aside from bradycardia. The patient was sent with a Holter monitor that reported sinus bradycardia with recorded heart rates between 40 and 63 beats per minute (bpm).

A transthoracic echocardiogram was performed which demonstrated an aneurysm measuring 2.6 cm x 3 cm involving the right Valsalva (coronary) sinus with encroachment on the right ventricular outflow tract. The aortic valve was tri-leaflet and opened without restrictions. Mild aortic regurgitation was noted on color Doppler. The maximum transverse diameter of the aortic root at the level of the aneurysm was 6.2 cm. Surgical correction of the aneurysm was performed.

During the postoperative period, the patient's heart rate ranged from 59 to 111 bpm with no incidents of bradycardia. He was subsequently discharged and followed up in the outpatient clinic 3 months later. At that time, he had a heart rate of 68 bpm with a blood pressure of 125/74. The patient remained asymptomatic in subsequent follow-ups.

#### **DISCUSSION**

Aneurysms in the Sinuses of Valsalva are most commonly found in the right coronary sinus (70% - 94%), followed by the non coronary sinus (5% - 29%), and least commonly in the left coronary sinus (1%). Congenital aneurysms are more common than acquired. In our case, a work up of the infectious, traumatic, atherosclerotic, and connective tissue disease etiologies were negative, leaving high suspicion for a congenital SVA.

Echocardiography, Doppler studies, MRI, or CT can be used in the diagnosis of a SVA. Color Doppler ultrasonography shows turbulent flow inside of an unruptured aneurysm and can also detect flow from a fistula into a receiving chamber. If visualization of the aneurysm is suboptimal on TTE, then evaluation with transesophageal echocardiography, CT, or MRI is recommended. In our case, the 2.6 x 3.0 aneurysm of the right sinus of Valsalva protruded into the intraventricular septum, resulting in direct pressure on and inducing an inflammatory response near the electroconductive pathways in the heart. Previous cases of SVA have been associated with various arrhythmias and conduction delays e.g. tachycardia, atrioventricular heart block, and complete heart block.

In conclusion, this case exhibits a rare presentation of an aneurysm in the sinus of Valsalva with the isolated finding of bradycardia. It followed the current recommendations for workup and surgical correction; furthermore, it adds to the support of structural evaluation of bradycardia through the use of echocardiography and other imaging modalities.

**Author: Avinash Oza BS**

Additional Authors: Vincent Peyko, PharmD, BCPS  
Institution: Kingsbrook Jewish Medical Center

### **Title: A unique presentation of Pantoea agglomerans bacteremia**

*Pantoea agglomerans*, formerly known as *Enterobacter agglomerans* and *Erwinia herbicola*, is a gram negative bacteria found in the environment, but may arise from endogenous intestinal flora in hospitalized patients. Due to its new nomenclature, it is often grouped as *Enterobacter* subspecies. *P. agglomerans* commonly presents as septic arthritis or synovitis, secondary to contaminated catheters or penetrating injuries in immunocompromised patients. We did not observe the commonly associated symptoms documented in previously reported cases.

A 54 year-old female, admitted for worsening respiratory symptoms and pneumonia, presented with shortness of breath and a productive cough for one day, with the following associated symptoms: constant, aching chest pain, wheezing, rhinorrhea, sore throat, and a fever of 100.3 °F. Medical and social history consisted of asthma, chronic obstructive pulmonary disease, cigarette smoking, and illicit drug use, including intranasal inhalation of heroin and cocaine. On physical examination, a minor healing laceration on her right medial thigh, of unknown origin to the patient, was appreciated. Lungs had bilateral rales and wheezing, pronounced in the bases. Chest X-ray showed possible right lower lobe pneumonia versus atelectasis. In the ED, blood cultures were drawn and intravenous levofloxacin was administered. Upon admission, she was switched to azithromycin and ceftriaxone. Symptoms did not improve for two days, although her fever subsided to 98.7 °F by day two. On day three, white blood cell count rose to 14.9K, from 10.3K the day before and 11.2K on admission, and blood culture sensitivities showed growth of *Pantoea agglomerans* sensitive to ampicillin/sulbactam, many third generation cephalosporins (including ceftriaxone), and other commonly prescribed antibiotics, but resistant to ampicillin and cefazolin. Computerized tomography scan of the chest without contrast was consistent with chronic obstructive lung disease, and she was switched to amikacin and ceftazidime. Concurrent HIV and Hepatitis C tests came back negative. Repeat blood cultures were drawn on day three. On day four, symptoms mildly improved, and antibiotics were changed to ampicillin/sulbactam. By day five, symptoms resolved, latest blood culture was negative, recent chest X-ray showed no pulmonary infiltrates, and white blood cell count was 11.4K; consequently, she was switched to oral levofloxacin and prepared for discharge.

This patient's presentation of acute chronic obstructive lung disease exacerbation, respiratory infection, and concurrent *Pantoea agglomerans* bacteremia was uncharacteristic, as this microbe commonly affects immunocompromised patients via penetrating trauma. Subtyping *Pantoea agglomerans* under *Enterobacter* subspecies could contribute towards obscuring relevant epidemiology. Early antibiotic treatment with a fluoroquinolone and regular blood cultures may be considered for refractory respiratory infections, limiting progression to septic shock.

**Author: Jacienta Paily MS**

Additional Authors: Sherry Bhowra DO, Haitham Hassane MD, Stephen Chrzanowski MD, Henri Woodman MD

Institution: University At Buffalo - Catholic Health Internal Medicine

**Title: Unexpected Cause of Acute Liver Injury: A Case Report of Human Babesiosis**

Introduction:

Acute liver injury can be defined as the presence of abnormal liver biochemical and functional tests without initial evidence of hepatic encephalopathy. This case demonstrates important strategies in delivering high value care when approaching a patient with acute liver injury, with emphasis on obtaining a thorough travel history.

Case Presentation:

A 47 year-old Caucasian male presented to his primary care physician with a five-day history of intermittent sharp epigastric pain radiating to his left flank and up to his left chest. He had associated malaise, diaphoresis, abdominal distension, nausea, and diarrhea. In the waiting room, he felt a wave of heat, lost consciousness, and fell to the ground tremulous. He was taken by ambulance to the nearest emergency department. On admission, vital signs showed a fever of 103 F and tachycardia. His skin was jaundiced and he had a dry cough. The patient had a history of alcohol use, and had a binge of 15-16 beers just 9 days prior to this episode. Labs showed a normal WBC count with 10% bands, thrombocytopenia and elevated liver function tests (LFTs) including total bilirubin 3.6 mg/dL with direct bilirubin 0.7 mg/dL, AST 153 U/l, ALT 208 U/l, ALP 146 U/l, GGT 84 U/l, and albumin 2.9 gm/dL. The patient was admitted for acute liver injury, but at the time the differential diagnosis was broad. Among the laboratory tests ordered were blood cultures, stool cultures, stool ova & parasite, toxicology screen, hepatitis panel, CMV/EBV antibodies, celiac disease antibodies, cryptosporidium and giardia antigen, leptospirosis panel, autoimmune liver disease panel (including AMA and smooth muscle antibody), iron panel, ferritin, haptoglobin, ceruloplasmin, and alpha-1 antitrypsin. Though most of the results were noncontributory, a low haptoglobin <30mg/dl along with a urine urobilinogen of 2.0 eu/dL pointed to an underlying hemolytic process. Further discussion with the patient elucidated his love for camping and recent camping trips to northeastern U.S. A tickborne disease was suspected and a peripheral smear was reviewed, revealing a positive diagnosis of babesiosis. The patient was started on atovaquone and azithromycin and subsequently showed dramatic clinical improvement.

Discussion:

Patients with infectious babesiosis may present with elevated LFTs along with signs of fever, jaundice, and malaise. With such a nonspecific presentation, it is not uncommon to order multiple laboratory tests in search of an etiology. Since the diagnosis of babesiosis often requires a large degree of suspicion, obtaining a thorough travel history is critical when working up an acute liver injury. If the travel history suggests a potential etiology, a targeted workup should follow in order to optimize care, avoid excess expenditure, and ultimately provide high value care.

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

**Medical Student  
Patient Safety and Outcomes  
Measurement**

## Medical Student Patient Safety & Outcomes Measurement

<p><b>Author: Renee Barlev MSIV</b>                  Additional Authors: Ashrei Bayewitz, MD, Winthrop University Hospital, Mineola, New York;                  Jonah Feldman, MD, Winthrop University Hospital, Mineola, New York                  Institution: Winthrop University Hospital</p> <p><b>Title: Floating in a (Protein) C of Waste: Quantifying the Incidence of Inappropriate Protein C Level Testing in the Hospital Setting</b></p> <p>Purpose: After completing the ACP’s AAIM curriculum on High Value Cost Conscious Care, residents and medical students were asked to identify and calculate the impact of unrecognized sources of inefficiency or waste in our hospital. A group of medical students and residents, along with a supervising faculty member, identified the inappropriate hypercoagulable workup as a wasteful practice pattern that warranted further evaluation. Published reports suggest that hypercoagulability panels are often ordered inappropriately, but these reports give little indication as to the magnitude of the problem specifically in the hospital setting. Furthermore, these reports do not specify which clinical situations would be best targeted for provider education or other hospital-based QI interventions. With this project, we sought to determine if within our hospital, acute ischemic stroke and acute DVT/PE are clinical scenarios that would have a sufficient incidence of inappropriate testing to justify diagnosis specific QI interventions.</p> <p>Methods: We performed a retrospective analysis from 2009-2014 on all hospitalized patients at Winthrop University Hospital that were discharged with the ICD-9 codes corresponding to acute ischemic stroke (CVA), or acute DVT/PE. We identified whether or not each patient underwent protein C level testing, as current guidelines define Protein C testing in the context of acute thromboembolism or CVA to be inappropriate.</p> <p>Results: During the evaluation period (2009-2014), there were 3,328 unique patient admissions with the diagnosis of CVA or DVT/PE. 483 (14.5%) of these patients had protein C levels resulted, for a total of 514 resulted tests (some patients had multiple results on one admission). Of the 514 inappropriate test results, 163 were associated with the diagnosis of CVA, 199 with the diagnosis of DVT, and 162 with the diagnosis of PE.</p> <p>Conclusions: Our analysis indicates that in our hospital, inappropriate ordering of Protein C levels in hospitalized patients with acute CVA or DVT/PE is a significant problem. We believe that having identified specific clinical contexts where wasteful ordering is prevalent, we are in a better position to create targeted interventions that decrease wasteful healthcare spending and increase the delivery of high value care at our institution. Further work is needed to learn what type of interventions can best achieve these goals.</p>	<p><b>Author: Eunice Monge</b>                  Additional Authors: Isaac Dapkins MD                  Institution: Bronx Lebanon</p> <p><b>Title: Efficacy of the HIV/AIDS Medical Case Management (MCM) Program at the Bronx-Lebanon Hospital Center</b></p> <p>The purpose of this study was to assess the effectiveness of the HIV/AIDS Medical Case Management (MCM) program on patient adherence to Medical Appointments at the Bronx-Lebanon Hospital Center. Effectiveness was measured by analyzing a patient’s attendance to infectious disease appointments. The first objective was to analyze change in attendance over a period of a year. The second objective was to determine the relationship between the years a patient has been a part of the program and their most recent attendance. The final objective was to determine if a relationship existed between sex and a patient’s recent attendance. In order to accomplish this, a chart review was conducted on half the patients in the program. 109 random patients were selected, but data was collected for 100 patients. The 9 patients who were in the program for less than 15 months were excluded. The results indicated that a patient’s attendance generally decreased in a year’s time, that there is a negative correlation between time in the MCM program and adherence to appointments, and that women are more adherent to appointments than men. The cause for the reduction in appointment adherence was not fully described but was likely a failure to complete appointment reminders on the part of the MCM staff. It is also possible that barriers to accessing appointments still exist. Interventions to improve MCM staff compliance with appointment reminders, patient level interventions to improve adherence and non MCM staff interventions to improve patient appointment adherence represents opportunities for improvement.</p>
---	--

<p><b>Author: Tammy Tavdy OMS II</b>                  Additional Authors: Chris Barsi, MD; Peter Harris, MD; Rich Menaik, MD; Nicholas C. Reis, DO; Swapna Munnangi, PhD; Mikhail Elfond, PhD                  Institution: Nassau University Medical Center</p> <p><b>Title: RISK FACTORS AND MORTALITY ASSOCIATED WITH UNDERTRIAGED PATIENTS AT A LEVEL I SAFETY-NET TRAUMA CENTER: A RETROSPECTIVE STUDY</b></p> <p><b>Background</b>                  While risk factors for undertriage have been previously identified, the studies have not looked at undertriaged patients within a multi-tiered trauma system, especially in a Level I public safety net trauma center. Determining these factors is especially important at a regional safety net hospital, due to the limited availability of clinical resources and funding.</p> <p><b>Purpose</b>                  The primary objective of this study was to determine the risk factors associated with undertriage and mortality in the undertriaged patient population at a Level I safety net trauma center.</p> <p><b>Methods</b>                  A retrospective analysis was performed on all trauma patients who presented to the Nassau University Medical Center with an Injury Severity Score (ISS) &gt;15 over a two year period (2013-2014). Univariate and multivariate regression analyses were used to determine the risk factors predictive of undertriage in severely injured trauma patients and of mortality in undertriaged patients.</p> <p><b>Results</b>                  During our two year study period, 334 of 2485 admitted trauma patients presented with major trauma (ISS &gt;15) and were included in our study. From the univariate analysis, variables that were found to be independently associated with mortality in undertriaged patients include intubation status (OR=29.1, 95%CI 2.501-359.78), GCS (OR=0.721, 95%CI 0.605-0.858), ISS (OR=1.127, 95% CI 1.022-1.244), revised trauma score (OR=0.372, 95%CI 0.200-0.694), and dementia (OR=4.7, 95%CI 1.096-20.149). A multivariate regression model controlling for the confounding variables in the univariate analysis was utilized to evaluate the contribution of independent variables to undertriage. There was a positive associative trend between dementia and mortality (OR=4.695, 95% CI 0.853-25.854, P=0.076). Independent risk factors that were found to be significantly associated with undertriage in severely injured trauma patients (ISS&gt;15) included GCS (OR=1.304, 95%CI 1.210-1.405), ISS (OR=0.876, 95%CI 0.843-0.910), MVC (OR=1.726, 95%CI 1.082-2.754), falls (OR=0.33, 95%CI 0.206-0.529), revised trauma score (OR=2.436, 95%CI 1.848-3.211), ED SBP (OR=1.013, 95%CI 1.006-1.020), ED HR (OR=0.992, 95%CI 0.983-1.001), intubation (OR=0.059, 95%CI 0.018-0.196), and dementia (OR=2.383, 95%CI 0.883-6.427). When a multivariate analysis was performed to evaluate the statistically significant risk factors, dementia was found to be significantly associated with undertriage in severely injured trauma patients (OR=11.384, 95%CI 1.537-84.305, P=0.0173).</p>	<p><b>Conclusion</b>                  This study shows that severely injured trauma patients with dementia are at significant risk for undertriage. In addition, dementia was found to have an influence on mortality in undertriaged patients. Early identification of these risk factors while triaging at a Level I safety net hospital could significantly enhance the level of care provided, and may translate into improved patient outcomes and decreased mortality following severe trauma.</p>
--	--

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

**Medical Student Research**

## Medical Student Research

<p><b>Author: Johnson Ho B.S.</b> Additional Authors: Kyle B. Womack, M.D., Christopher Paliotta, B.S., and Peter J. Bergold, Ph.D. Institution: SUNY Downstate Medical Center</p> <p><b>Title: INTERHEMISPHERIC INFORMATION TRANSFER: A NEW DIAGNOSTIC METHOD FOR WHITE MATTER DISRUPTION IN PATIENTS WITH MILD TRAUMATIC BRAIN INJURY.</b></p> <p>Purpose: Mild traumatic brain injury (mTBI) yields brain deficits with variable symptomology that can only be effectively diagnosed months after injury. A new diagnostic method was developed for earlier detection of long-term sequelae post-mTBI by indirectly measuring interhemispheric transfer time (ITT). This study examined whether ITT in mTBI patients correlate with white matter abnormalities in centroaxial structures. Within 24 hours after mTBI, crossed reaction times (CRT), uncrossed reaction times (URT) and crossed-uncrossed difference (CUD) were measured in 23 patients using a laptop computer that displayed visual stimuli to either the left or right visual field of the retina. CUD is a surrogate marker of ITT.</p> <p>Methods: Patients were recruited at Parkland Memorial Hospital, Dallas, TX and scanned at the UT Southwestern Medical Center. Patients aged 18-50 years were included if they had a post-resuscitation Glasgow Coma Scale &gt; 13 and an injury mechanism compatible with mTBI. Patients with mTBI were given a peripheral visual reaction test (PVRT) within 24 hours after injury. PVRT was administered using a laptop computer that measured the time elapsed between exposure to a plus sign 31.6° on the left or right from the central point and the striking of a computer key with his/her dominant hand. The test had 50 trials and three outcome measurements were computed from the average of the final five trials: CRT, URT, and CUD. Within 7 days after injury the same cohort received a diffusion tensor-MRI (DTI) scan and a battery of neuropsychological tests: Digit Symbol, Symbol Search, Digit Span, Controlled Oral Word Test, Trails A and B, Brief Symptom Inventory and California Verbal Learning Test. Hypothesis: mTBI patients were predicted to have higher CUDs, aberrant DTI parameters and abnormal neuropsychological test scores. CUD scores in the injured cohort were predicted to correlate with aberrant DTI parameters from centroaxial white matter regions.</p> <p>Results: CUD, CRT and URT deficits, &gt; 2 standard deviations (SD) were seen in 40.9%, 68.2% and 45.5% of patients, respectively. ITT of injured patients negatively correlated with mean diffusivity (<math>p &lt; 0.001</math>, <math>r = -0.811</math>) in the posterior corpus callosum. mTBI patients with a CUD &lt; 2SD significantly differed from those with a CUD &gt; 2SD on the Stroop 1, COWAT, and obsessive-compulsive components of the Brief Symptom Inventory test.</p> <p>Conclusions: Large CRT, URT or CUD deficits are common after mTBI. ITT measures white matter integrity in the posterior corpus callosum, a brain region frequently damaged by mTBI. Patients can be stratified on the basis of ITT on the Stroop 1, COWAT and the obsessive-compulsive components of the neuropsychological tests.</p>	<p><b>Author: Tamana Kaderi</b> Additional Authors: Dr. Allen Kong Institution: University of California Irvine Medical Center</p> <p><b>Title: Red Blood Cell Age Correlation with Venous Thromboembolism in Surgical Intensive Care Patients</b></p> <p>Blood transfusions are a major part of patient intensive care with more than 40% of critical care patients receiving transfusions during their stay in the ICU. Increased red blood cell storage time has been associated with immunomodulation and impaired vasoregulation. Deep venous thrombosis (DVT) and its sequel, pulmonary embolism (PE), are the leading causes of preventable in-hospital deaths. Virchow's triad is a well-known assessment of risk factors for the development of a thrombus; one category includes hypercoagulability. We hypothesize that patients in the ICU who were given RBC transfusions with older age had a higher incidence of DVT and complications. Data were collected on trauma and non-trauma patients, ages 18 and older, who were admitted to the University of California Irvine Medical Center ICU from 2009-2015. We placed patients into 4 groups- patients who received blood that was less than or equal to 14 days old, less than or equal to 21 days old, less than or equal to 28 days old, or 29 days old or greater. We primarily compared patients who received blood greater than 28 days old with patients who received blood less than 28 days old and assessed DVT rates among these groups. Dates of hospitalization, DVT and PE results, number of RBC units given, splenic injuries, diabetes, and DVT prophylaxis were looked at to rule out any confounding variables. Statistical analysis included a multivariate logistic regression to investigate the relationship between RBC age and risk of DVT. Preliminary data analysis indicated no statistically significant relationship between RBC age and DVT (<math>p=0.5874</math>) when all other variables were held constant. We will further analyze these patients to find an inflection point where there is a trend toward significance. We hope that these findings will help improve our DVT prophylaxis protocol. Ultimately, the goal is to reduce the prevalence of deep vein thrombosis and improve patient care.</p>
---	--

## Medical Student Research

**Author: Dahlia Kenawy**

Additional Authors: Daniel Schwartz; Marc Gibber, MD  
Institution: Montefiore Medical Center; Albert Einstein  
College of Medicine

**Title: An Evaluation of Perioperative Communication  
in the Operating Room**

Background: In the operating room (OR), communication failures are often cited as at least one of the root causes of about 80% of sentinel events. To our knowledge, no study has yet investigated perioperative levels of communication in relation to knowledge of surgical steps. In this study, we compared perceptions of communication of surgical protocol in the OR between surgeons and non-surgeons to establish a baseline. We further evaluated the quality of communication by assessing non-surgeon knowledge of surgical steps of laparoscopic cholecystectomies to determine whether important information about the operation was relayed to non-surgeons.

Study Design: Surgeons and non-surgeons who were routinely involved in laparoscopic cholecystectomies were surveyed. Laparoscopic cholecystectomies were chosen because of their frequency and relatively routine nature. Surgeons were defined as attending physicians and residents, while non-surgeons were defined as any personnel in the OR who were directly or indirectly involved in the procedure. Participants were asked to rate the importance of communication between team members on a 7-point Likert scale, in addition to rating current communication levels on a scale of 1 to 10 with 10 being the best possible communication in regards to surgical procedural steps. Participants were also asked to list the steps involved in the procedure.

Results: There was no significant difference between surgeons (n = 23) and non-surgeons (n = 43) in terms of rating the importance of communication amongst team members (p > 0.50). There was a significant difference (p = 0.003) in rating current levels of communication, with surgeons (n = 23) giving an average rating of 8.9 +/- 1.2 and non-surgeons (n = 41) giving an average rating of 7.6 +/- 2.1. To evaluate the effectiveness of communication in the OR, we compared the number of surgical steps provided by surgeons (n = 8) and non-surgeons (n = 43) and found a significant difference (p = 3.7E-10), with the former averaging 13.5 +/- 1.5 steps and the latter averaging 5.4 +/- 3.1 steps.

Conclusion: Our findings are consistent with previous studies showing both inadequate communication in the OR, as well as heightened perceptions of communication by surgeons as compared to non-surgeons. This disconnect in perception may be partially explained by the differences in opinion by surgeons and non-surgeons as to what constitutes good teamwork.

**Author: Jonathan Peters University at Buffalo School  
of Medicine and Biomedical Sciences MD Candidate,  
'18**

Additional Authors: Mark Travers, PhD; Lisa Vogl, MPH  
Institution: Roswell Park Cancer Institute

**Title: Secondhand Yield of Tobacco Products: E-  
cigarettes, Cigarettes, Cigarillos, and Hookah**

While cigarette smoking prevalence has declined in youth, electronic cigarette (e-cig) use has tripled in the last year, surpassing cigarette use, and hookah use has doubled, equaling cigarette use. Secondhand smoke exposure is a direct cause of increased incidence of cardiovascular and pulmonary disease, and mortality. In light of this dramatic shift in the tobacco marketplace the goal of this study is to examine the differences in secondhand emissions between traditional and emerging tobacco products. This will determine what we expect the future public health burden to be from involuntary exposure to tobacco emissions. This project investigates the particulate (PM) and carbon monoxide (CO) emissions of combustible cigarettes, comparing them to hookah, cigarillos, and electronic cigarettes.

The authors characterized emissions using standardized puff topography settings unique for each product during 2 smoking sessions in a controlled laboratory setting for each product designed to mimic what a group of 4 people would smoke during one hour. Emissions were also measured for one hour before and two hours after each smoking session. Each smoking session consisted of 120 puffs, or about ten 1-2 second puffs every five minutes. Cigarettes, cigarillos, and e-cigarettes were smoked through a syringe to standardize 35 mL puffs. The hookah was smoked through an electric pump. Particulate matter (PM2.5, TSI SidePak), particle size distribution (TSI Aerotrak), and CO (TSI Q-Trak) were measured continuously.

Average CO emissions from the hookah were significantly greater by a factor of 4.6 than from cigarettes, 3.83 versus 0.84 ppm (p<.0001). This finding is most likely due to the presence of a coal, necessary to heat the tobacco (shisha) in the hookah. Average PM2.5 emissions from the e-cig were significantly less than from the cigarette, 7.29 versus 319.90 µg/m<sup>3</sup>, a factor of 44 (p<.0001). Yet average PM2.5 emissions from the cigarillo and hookah were 274.8 and 75.7 µg/m<sup>3</sup> respectively, 7.9 and 2.2 times higher than the EPA National Ambient Air Quality standard for outdoor particulate matter pollution, 35 µg/m<sup>3</sup>. CO emissions from the cigarillo were significantly greater than from the cigarette by a factor of 2.2 (p<.0001). Particulate matter from all products was primarily in the respirable range (<4µm), meaning they are easily breathed deep into the lungs. In light of the high levels of CO and PM emissions, hookah and cigarillos should be prohibited in indoor spaces. Further research needs to be conducted on e-cig emissions measuring a broader range of emissions and carcinogens. Nicotine emissions from these products will be investigated in future studies.

## Medical Student Research

<p><b>Author: Polina Pinkhasova</b> OMS-III Additional Authors: Satoru Kobayashi, PhD, Qiangrong Liang, MD, PhD Institution: NYIT College of Osteopathic Medicine</p> <p><b>Title: METFORMIN REDUCES MITOCHONDRIAL DEGRADATION IN DOXORUBICIN TREATED CARDIAC MYOBLASTS</b></p> <p>METFORMIN REDUCES MITOCHONDRIAL DEGRADATION IN DOXORUBICIN TREATED CARDIAC MYOBLASTS Polina R. Pinkhasova, OMS II, Satoru Kobayashi, PhD, and Qiangrong Liang, MD, PhD Department of Biomedical Sciences New York Institute of Technology College of Osteopathic Medicine, Old Westbury NY 11568, 2015 Background: Doxorubicin (DOX) is among the most effective and widely used antineoplastic agents for the treatment of a wide variety of cancers including both solid tumors and leukemias. However, its usefulness is compromised by its cardiotoxicity. It has been known that Metformin (MET) can rescue myocardium from DOX-induced damage. Given its cardioprotective properties, MET may be used in DOX-containing chemotherapy to reduce its cardiotoxic effect. Preliminary studies have demonstrated that DOX induces excessive mitochondrial fragmentation and degradation. Objective: We tested the hypothesis that MET protects against DOX-induced cardiomyocyte injury by inhibiting excessive degradation of mitochondria through the autophagy-lysosome pathway (mitophagy). Methods: H9C2 cardiac myoblasts were cultured in 10% fetal bovine serum containing medium. Cells were incubated with DOX (1 uM) for 16hrs. MET (1 mM, 3 mM, and 5 mM) were added 4hrs prior to DOX treatment. Propidium Iodide (PI) staining was used to determine DOX-induced cardiomyocyte death. Apoptotic cell death was determined by the cleavage of PARP in Western blot analysis. To evaluate the level of mitophagy, an adenovirus encoding mitophagy reporter (AdmtRosella) were infected in cells 24hrs before treatments. MtRosella is composed of a mitochondrial targeting sequence and a RFP-GFP fusion protein. Using confocal microscopy the fragmented mitochondria degraded in the lysosome were detected as red puncta where the pH sensitive GFP is quenched, while the rod-shaped mitochondria were detected as yellow in the green/red merged image. The numbers of red puncta were counted to evaluate the level of mitophagy. Results: DOX increased the number of PI positive cells and the level of PARP cleavage, which were attenuated by Metformin. In the mitophagy reporter assay, DOX increased the number of red puncta, the signature of mitophagy, while it was reversed by MET at the dose providing protection against DOX. Conclusion: Metformin protects against Doxorubicin-induced cardiotoxicity. The inhibition of mitophagy by Metformin may explain the mechanism behind cardioprotection.</p>	<p><b>Author: Sonika Raj</b> Additional Authors: Manpreet Kaur, MD Institution: Albany Medical College</p> <p><b>Title: Screening for Obstructive Sleep Apnea in Adult Psychiatry Clinic</b></p> <p>Objective: The rate of undiagnosed obstructive sleep apnea (OSA) is very high in the general population, and there is significant comorbidity between OSA and mood disorders. To further complicate this issue, many OSA symptoms, such as daytime sleepiness, are difficult to differentiate from symptoms of mood disorders. In this study, we screened patients with mood disorder symptoms that might also represent undiagnosed OSA, to investigate whether they had been identified and referred to a sleep clinic for further evaluation. We also examined the frequency at which patients at intermediate to high risk for OSA were being prescribed sedatives to manage their mood disorder symptoms. Method: 138 patients at the outpatient Psychiatry clinic at Albany Medical Center, were screened for OSA symptoms and risk factors. Participant STOP-BANG scores were used to identify those at high risk for developing OSA. Electronic medical records were reviewed to identify the medications prescribed to these patients at the clinic. Results: 115 patients had complete data for the STOP-BANG questionnaire. Within this group, 29 (25%) were found to be at high risk for OSA, and 32 (28%) were found to be at intermediate risk for OSA. Of these patients (both intermediate- and high-risk), 30 (50%) had been referred to a sleep clinic. However, only 1 of these patients was referred by a provider at the psychiatry clinic. 44% of the patients in the intermediate- to high-risk group were being prescribed sedatives by their mental health provider. Conclusions: OSA screening can be done very easily; however, in our sample, the rate of referral to a sleep clinic for further evaluation was very poor. In fact, most patients who met elevated risk criteria were not advised to follow up with a sleep specialist. We also found that in our sample, nearly half of the patients with elevated risk criteria were being prescribed sedatives by their mental health providers. Prescribing sedatives to patients at intermediate or high risk for OSA without full evaluation is not recommended, as this practice may actually worsen patients' symptoms. This study highlights the importance of raising provider awareness of OSA within psychiatric populations, and educating them about appropriate referral sources for further evaluation.</p>
---	---

**Author: Lindsay Wahl**

Additional Authors: Jennifer J Judge, Amali Epa, Thomas H Thatcher, Richard P Phipps, Patricia J Sime, R Matthew Kottmann

Institution: University of Rochester School of Medicine and Dentistry, CTSI

**Title: Tissue Transglutaminase Mediates Myofibroblast Differentiation in Primary Human Lung Fibroblasts**

Background: Idiopathic Pulmonary Fibrosis (IPF) is a progressive, fibrotic lung disease of unknown etiology that affects more than 200,000 people in the United States. There are few effective therapies and the median survival from the time of diagnosis is 2.9 years. The pathological hallmark of IPF is the transformation of lung fibroblasts into myofibroblasts, which accumulate in clusters called fibroblastic foci.

Myofibroblasts produce excess  $\alpha$ -smooth muscle actin ( $\alpha$ SMA,) calponin and extracellular matrix proteins in the lung interstitium, resulting in scar formation and compromise of lung function. One key cytokine responsible for myofibroblast differentiation is transforming growth factor (TGF) $\beta$ 1.

Our lab has shown that TG2 expression is increased in the lungs of patients with IPF, and that TG2 knockout mice are largely protected from developing pulmonary fibrosis when exposed to bleomycin, a pro-fibrotic agent. While TG2 is likely an important protein involved in the progression of IPF, the enzyme has not yet been shown to regulate myofibroblast differentiation in the lung. Here, we hypothesize that intracellular TG2 regulates expression of myofibroblast markers in human lung fibroblasts and that inhibition of TG2 will inhibit myofibroblast differentiation.

**Methods:**

To examine the effects of TG2 inhibition on myofibroblast differentiation in human lung fibroblasts, Short Hairpin (Sh) RNA lentiviral vectors targeting TG2 and a scrambled Sh-RNA were used to knockdown TG2 expression. Similarly, a lentiviral vector was used to overexpress wild type TG2 and W241A, a transamidation deficient mutant TG2. These cells were cultured and treated with TGF $\beta$ 1. Cell lysates were harvested 72 hours post-treatment. Protein expression levels of  $\alpha$ SMA, calponin, TG2 and GAPDH were measured by Western blot.

**Results:**

In normal lung fibroblasts, TGF $\beta$ 1 induced expression of  $\alpha$ SMA and TG2. Our results show that TG2 inhibition decreased the expression of calponin,  $\alpha$ SMA and TGF $\beta$ 1 induced  $\alpha$ SMA expression compared to controls. Wild type TG2 overexpression increased expression of calponin and  $\alpha$ SMA. However, overexpression of the the transamidation deficient mutant of TG2, W241A, did not increase expression of calponin or  $\alpha$ SMA.

**Conclusion:**

These data suggest that the TG2 may regulate myofibroblast differentiation in primary human lung fibroblasts, and may be an important driver of fibrosis in the lung tissue of patients with IPF. In addition, these data suggest that transamidation activity of TG2 is necessary for promoting myofibroblast differentiation in the lung.

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

**Resident/ Fellow  
Clinical Vignette**

## Resident/ Fellow Clinical Vignette

**Author: Nashwa Abdulsalam, MBBS**

Additional Authors: Nagesh Jadhav, MD

Institution: Rochester General Hospital

### **Title: Cefepime Induced Encephalopathy**

Background: Cefepime neurotoxicity has been reported in patients with chronic kidney disease. It can present with myoclonus, impaired consciousness or seizures. It is usually related to the plasma concentration of cefepime. The diagnosis of cefepime-induced encephalopathy can be difficult and is usually not well recognized.

Case: A 71 year old man with a large B cell lymphoma who was receiving radiation therapy was admitted with fever and right leg ulcer. He was found to be neutropenic with findings of right calcaneal osteomyelitis on bone scan. Wound culture was positive for MRSA. He was treated with IV daptomycin 6mg/kg daily and cefepime 2 grams every 8 hours as he had developed neutropenia as a side effect of vancomycin therapy in the past. Three days following initiation of therapy despite resolution of fever, the patient developed confusion and then obtundation. Physical examination: Blood pressure 138/62 mm hg, pulse 84/min, temperature 97.2 F. He was disorientated to time, place and person. Neurologic examination showed myoclonus with no deficits. There was a right calcaneal non healing shallow ulcer 3x4 cm in size without purulence or cellulitis. Laboratory: WBC 900u/L, Absolute neutrophil count 200, Hb 7.8g/dl, creatinine 0.8mg /dl, lactate 1.4mmol/L, ammonia 6ummol/L.

Head CT scan was normal, Lumbar puncture showed mildly elevated proteins with zero cell count. EEG was consistent with moderate diffuse encephalopathy, with no epileptiform abnormalities. Cefepime was discontinued and within two days, the patient's mental status returned to baseline, and myoclonus resolved.

Discussion: Cefepime-induced neurotoxicity, a potentially fatal complication, has been described in patients with chronic kidney disease. Although our patient had normal kidney function his encephalopathy appeared to be induced by cefepime and no other cause was found. The presence of myoclonus and the return of his mental status to baseline following discontinuation of cefepime supports this diagnosis.

Conclusion: Cefepime-induced neurotoxicity can be fatal, and it should be considered in the differential diagnosis of acute encephalopathy. Dose adjustments and attention to kidney function are important, when patients are treated with cefepime, but if encephalopathy develops with no clear cause discontinuation of the drug should be considered regardless of kidney function.

**Author: Prakash Acharya, MD**

Additional Authors: Bishow Aryal MD, Frank Tamarin MD, Prasanta Basak MD, Stephen Jesmajian MD

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

### **Title: HYPERAMMONEMIC ENCEPHALOPATHY INDUCED BY COMBINING TOPIRAMATE WITH SODIUM VALPROATE**

#### Introduction

Sodium valproate (SV) is an anticonvulsant that can increase serum ammonia levels. Topiramate is another anticonvulsant which interferes with ammonia metabolism. We present a case where the addition of Topiramate to SV resulted in hyperammonemic encephalopathy.

#### Case Presentation

A 57 year old male with a history of bipolar disorder and seizures, presented with frequent falls for 2 months. He attributed the falls to unsteady gait, which has been progressively worsening. He denied loss of consciousness, weakness or palpitations. Home medications included SV, topiramate, quetiapine and venlafaxine. He had been on the same dose of sodium valproate for 2 years, and topiramate had been started 4 months ago. Physical examination was negative for orthostatic hypotension. He had poor recent and distant memory. Heel to shin dysmetria was present, suggesting cerebellar dysfunction. Serum ammonia level was elevated, but liver function tests and SV levels were within normal limits. MRI of the brain and CT scan of the head were normal. SV and topiramate were discontinued, and the patient started on intravenous L-carnitine. His ammonia level trended down to normal within 3 days. His mental status improved considerably and he was able to ambulate well with a walker. He was discharged on L-carnitine supplementation, as well as a reduced dose of SV and topiramate.

#### Discussion

The exact mechanism of encephalopathy caused by SV is unknown. A direct cortical effect and a secondary effect from hyperammonemia have been proposed. Ammonia, a by-product of hepatic amino-acid metabolism, is converted to urea via the Krebs-Henseleit urea cycle in the liver for subsequent excretion in the urine. SV hinders ammonia excretion by inhibiting carbamoyl-phosphate synthetase I enzyme in the urea cycle, hence raising plasma ammonia levels. In addition, SV increases ammonia production in the kidneys. Topiramate increases ammonia level by inhibiting substrate formation necessary for the urea cycle in the liver. It also inhibits cerebral glutamine synthetase, which helps to detoxify cerebral ammonia by converting glutamate and ammonia to glutamine. The increased intra-cerebral ammonia leads to encephalopathy by impairing astrocyte function and causing cerebral edema. Carnitine is an important cofactor of beta oxidation in liver and long term SV therapy causes its depletion, possibly secondary to urinary loss. Depleted carnitine leads to the production of propionic acid which inhibits an essential enzyme of urea cycle. Carnitine repletion has been shown to be effective in the management of hyperammonemic encephalopathy.

#### Conclusion

SV can cause hyperammonemic encephalopathy, even with normal serum levels. Combining topiramate with SV further increases the risk of hyperammonemic encephalopathy. Ammonia level should be promptly checked in patients on these drugs who present with encephalopathy, and L-carnitine is an effective treatment in hyperammonemic states.

## Resident/ Fellow Clinical Vignette

**Author: Ahmed Al-Chalabi, MD**

Additional Authors: Ahmed Mahmood, MD

Mohammad Malekzadegan, MD

Avani Patel, M

Institution: Jamaica Hospital Medical Center, Internal Medicine

### **Title: SMALL INTESTINE MUCOSA ASSOCIATED LYMPHOID TISSUE AND HELICOBACTER PYLORI PEPTIC ULCER DISEASE. IS THERE ANY ASSOCIATION?**

H. pylori has been highly implicated as the stimulating agent in many gastric cancers, but especially in Gastric MALT lymphomas or maltomas. However, scarce literature exists about their role in the development of non-gastric MALT lymphomas.

We present a case of 51 year old female with history of hypertension who was referred from primary care physician to gastroenterologist because of complains of vague abdominal discomfort, bloating and occasional vomiting of two months duration. The patient also had an incidental 8.3 cm hypochoic structure with central shadowing at the level of the umbilicus on a pelvic ultrasound, suspicious of a dilated loop of small bowel. Further history, physical exam and lab tests were unremarkable. A computer tomography of abdomen and pelvis was ordered, while the patient was scheduled for esophagogastroduodenoscopy and colonoscopy. The computer tomography revealed markedly thickened and distended small bowel segments, with no signs of metastasis or lymph node involvement. On endoscopy, two one-centimeter antral ulcers with raised margins and a duodenal bulbar ulcer were found. Histopathological examination identified moderate to severe chronic active gastritis with abundant H. pylori-like bacilli. The rapid urease test was positive. Colonoscopy was normal apart from a tubular adenoma which was removed in its entirety. After completing Helicobacter pylori eradication therapy, a single balloon enteroscopy was performed. Here, a circumferential stricture in the middle jejunum about 3-5cm in length with abnormal mucosa and crypt pattern was identified. Biopsy samples showed a majority of small lymphocytes positive for CD20, CD 79a, CD43, Bcl2, but negative for CD10, Bcl6, CD21, CD23 and Bcll. The diagnosis of marginal zone lymphoma (MALT lymphoma) was made. Since the positron emission tomography showed no signs of metastasis, the patient was referred for laparoscopic small bowel resection. The postoperative histopathology report confirmed transmural extension of the lymphoma with normal intestinal tissue at the proximal and distal cut margins. Given the early stage of the disease (stage I, limited), the patient did not receive chemotherapy or radiation therapy. The patient's symptoms resolved completely and a repeat computer tomography six months after surgery failed to show any recurrent disease or metastasis.

Though the role of Helicobacter pylori in the development of gastric MALT lymphomas is well established, the association with non-gastric lymphoma is still unproven. In particular, antibiotic therapy rarely leads to regression of non-gastric MALT tumors. Yet a remarkable number of patients diagnosed with non-gastric MALT lymphoma (up to 45% in one study) were found to have evidence of infection with H. pylori. This suggests a role in the pathogenesis that needs further evaluation. We believe that all patients diagnosed with non-gastric MALT should undergo workup to rule out active H. pylori disease.

**Author: Adnan Aldurah, MBBS**

Additional Authors: Matthew Ferrantino, MD

Sh.Noor Husnain, MD

Institution: Unity Hospital, Internal Medicine Residency Program

### **Title: Common Symptom ! Uncommon Disease**

47 year old gentleman with hypertension and GERD presented to the pulmonary office with two years of progressive shortness of breath and non-productive cough. He was seen by his internist multiple times, treated with multiple courses of antibiotics, steroids, bronchodilators and antitussives without relief. Exertional dyspnea limited his daily activities and over two years progressed to dyspnea at rest. He is lifelong nonsmoker, with exposures to dust, soil, Freon gas, acetylene, and fumes from cutting metal.

On examination, Temp 36.8, BP 208/100, HR 77, RR 16, SpO2 74% on room air. He appeared in moderate respiratory distress with frequent paroxysms of nonproductive cough. There was mild cyanosis and digital clubbing. Lung examination demonstrated diminished breath sounds bilaterally without rales, wheezes or rhonchi. Cardiac examination was regular with normal S1, S2, and II/IV systolic murmur. Abdominal, extremity, dermatologic and rheumatologic examinations were normal.

Lab work demonstrated unremarkable CBC and chemistry. Subsequent lab work included negative ANA, ANCA, CK, Aldolase, Anti-Jo-1 antibody, Anti-Scl-70 antibody, and Anti-RNP antibody. Chest X-ray: increased interstitial markings, right greater than left. Chest CT: scattered diffuse ground-glass attenuation with superimposed interlobar septal thickening in a crazy-paving pattern.

Patient was transferred to ICU, intubated, and underwent bronchoscopy. Bronchoalveolar lavage (BAL) revealed progressive return of white opaque material with sequential lavage. PAS-staining was positive, consistent with Pulmonary Alveolar Proteinosis (PAP).

PAP is a diffuse lung disease characterized by accumulation of PAS-positive lipoproteinaceous material in the distal air spaces, with little or no lung inflammation and preserved underlying lung architecture.

PAP has three forms: congenital due to mutations in surfactant or GM-CSF receptors, secondary to high level of dust exposure, hematological malignancy or post-allogeneic bone marrow transplantation, or acquired, which is the most common and is associated with anti-GM-CSF antibodies that cause macrophage dysfunction and impaired processing of surfactant.

PAP can be diagnosed by classical radiographic findings of "crazy-paving" appearance of the pulmonary parenchyma with identification of PAS-positive material on BAL or trans-bronchial biopsy.

Treatment depends upon symptomatology. Asymptomatic or mildly symptomatic patients can be observed without treatment. For mild-moderate disease, supportive therapy with oxygen and ongoing monitoring is indicated. For moderate-severe disease, which includes significant shortness of breath, hypoxemia at rest, treatment options include whole lung lavage, GM-CSF if anti-GM-CSF antibodies are positive, or rituximab.

Our patient underwent whole lung lavage and was subsequently weaned from mechanical ventilator support. He was found to have anti-GM-CSF antibodies, and has been treated with daily GM-CSF.

Although uncommon diagnosis, PAP is a potential cause of chronic cough and exertional dyspnea. For patients with persistent cough, dyspnea and non-resolving radiographic abnormalities, early referral to pulmonology should be considered.

## Resident/ Fellow Clinical Vignette

**Author: Shaheer Ali, MD**

Additional Authors: Renjini Thomas, M.D., Waqas Malik, M.D., Roxana Lazarescu, M.D.

Institution: NewYork-Presbyterian/Queens, Flushing, NY

### **Title: A CASE OF THE MYSTERY FEVER: ADULT-ONSET STILL'S DISEASE**

Introduction: We report a case of Still's disease in a female patient with cyclical fevers, sore throat and rash initially diagnosed as bacterial pharyngitis. With elevated acute phase reactants, adult-onset Still's disease (AOSD) should be considered on the list of differentials. The Yamaguchi criteria were utilized to narrow the differential and reach the ultimate diagnosis.

Case Presentation: A 49 year old female from China, with a past medical history of pre-DM presented with high fevers and generalized weakness for one week. She reported fevers ranging from 37.8-38.8oC, which resolved by the evening. Associated symptoms included sore throat, dizziness, abdominal pain, generalized joint pain and malaise. She had emigrated from China 8 years ago and had recently gone back to visit for 1 month. The patient denied nausea, vomiting, diarrhea, known sick contacts or history of TB or exposure. The patient had a Tmax of 38.9oC and bandemia of 35%, as well as transaminitis, elevated CRP, and a serum ferritin level >33,000. She continued to have fevers (38.3-38.8oC) which did not respond to broad-spectrum empiric antibiotics. The patient also had a transient salmon colored rash on the medial aspects of her thighs bilaterally which worsened with onset of fever. Our workup which included investigating infectious sources, autoimmune disease, drug reactions, malignancy, including bone marrow biopsy, was inconclusive and antibiotics were discontinued after persistent symptoms. With the Yamaguchi criteria satisfied and most alternate diagnosis excluded, the patient was diagnosed with AOSD. She was started on naproxen and prednisone with improvement in symptoms.

Discussion: Our patient had an initially confounding presentation in the setting of recent travel, cyclic fevers and transaminitis and a negative infectious work up into conventional causes. However, it was the development of a transient salmon-colored rash which waxed with fevers that led us to investigate rheumatologic causes. However, ANA and RF were both negative. As patient met 5 of the Yamaguchi criteria for Still's disease and other etiologies were excluded, a diagnosis was finally reached.<sup>1</sup> In comparative study of the six types of criteria used to diagnose Still's, Yamaguchi criteria were found to be the most sensitive.<sup>2</sup> Although the differential for FUO is broad and is a diagnosis of exclusion, rheumatologic etiologies should always be considered in a patient that does not respond to conventional therapy. Among the causes of FUO, AOSD is the most common connective tissue disease.<sup>3</sup>

**Author: Nabeel AlQsous, MD**

Additional Authors: Quintos A. MD, Khoudari G. MD , Lyons J. MD, Lingutla D. MD

Institution: unity hospital/Rochester Regional Health System

### **Title: MORE THAN A BACK ABSCESS! EMPYEMA NECESSITANS IN THE SETTING OF PNEUMONIA AND EFFUSION**

Introduction:

Empyema necessitans is a rare complication of pleural space infections and can be confused with chest wall or back abscess. We present an interesting case of empyema necessitans which initially presented as back abscess.

Case:

A 22 year old African American male with no significant past medical history was referred to the hospital from his primary care physician's office for a possible back abscess. Patient's chief complaint was "Pus draining from his back for the last 3 months". He also had 3 month history of productive cough and intermittent chills. No history of smoking, alcohol or drug abuse. On physical examination patient had sinus tachycardia .Further evaluation revealed a 1\*0.5 cm wound in the left posterior chest wall, which was draining serous fluid. Absent air entry on the left lung base on auscultation. Laboratory work remarkable for leukocytosis. CT scan of the chest showed a left sided loculated pleural effusion with underlying parenchymal infiltrates along with left sided sinus tract extending from the skin surface at the site of the draining wound connecting with the left lung effusion. Hence the diagnosis of Empyema necessitans was made. He was initially started on IV antibiotics and subsequently underwent drainage of the chest wall abscess and resection of the sinus tract by thoracic surgery. Surgical cultures grew a rare Beta-Hemolytic Streptococcus Group B. His PPD test was negative. His clinical condition has improved gradually and was discharged home on IV antibiotics.

Discussion:

Empyema necessitans is a rare complication of pleural space infection that occurs mostly due to inadequate treatment of such infections when the infected fluid dissects through the chest wall. Early diagnosis and antibiotic therapy of pneumonia will, in most cases, make the course of the disease uncomplicated, and simple parapneumonic effusion (PPE) often resolves with antibiotic therapy alone. In 5-10% of patients, PPE becomes more complicated and leads to empyema, which might later on lead to empyema necessitans. The mortality rate among patients with empyema ranges between 5.4% and 22%

Therapy for empyema is determined by the stage. In the early exudative phase of empyema, repeated drainage may be adequate. However, during the fibropurulent phase thoracocentesis is always unsatisfactory and closed chest tube drainage is often necessary. Empyema necessities often requires surgical resection of the fistula.

Conclusion:

Our patient has been having symptoms for months that went undetected and was attributed to a back abscess. Careful history and physical examination are key for early diagnosis and would have prevented the complicated course above.

## Resident/ Fellow Clinical Vignette

**Author: Nabeel AlQsous, MD**

Additional Authors: Telfah M. MD, Khoudari G. MD,  
Lyons J. MD, Matos M. MD  
Institution: unity hospital/Rochester Regional Health

### **Title: UNILATERAL PULMONARY EDEMA MIMICKING BRONCHOALVEOLAR CARCINOMA**

#### Introduction

Cardiogenic pulmonary edema is very common and usually presents with bilateral "bat wings" appearance. Unilateral cardiogenic pulmonary edema is a rare entity and is almost always linked to severe mitral valve regurgitation. This is a case report of unilateral right sided pulmonary edema in the setting of severe aortic valve stenosis.

#### Case

A 66 year old male with a 40 pack-year smoking history, severe aortic valve stenosis and atrial fibrillation who presented to the hospital with upper GI bleed while he was on Dabigatran. While being managed for his bleed, he was complaining of progressive shortness of breath and lower extremities edema.

Imaging including a chest x-ray and CT scan showed diffuse right lung infiltrate that was suspicious for bronchoalveolar carcinoma, along with moderate right sided pleural effusion without masses. He did not have any clinical evidence of pneumonia. Thoracentesis showed transudative fluid with negative cytology for malignancy and negative fluid cultures for bacteria, AFB and fungus. Bronchoscopy was done and showed normal endobronchial exam with negative bronchoalveolar lavage for infection or malignancy.

Cardiac evaluation showed an ejection fraction of 40%, severe aortic valve stenosis, mild mitral valve regurgitation, left ventricular end diastolic pressure of 28 mmHg and pulmonary artery pressure of 48 mm Hg. The beta natriuretic peptide was elevated. Patient was started on furosemide, symptomatic improvement was noted, repeat chest x-ray showed almost complete resolution of right lung infiltrate. The patient was sent for aortic valve replacement. Later follow up CXR showed complete resolution of the infiltrate.

#### Discussion

Unilateral cardiogenic pulmonary edema is a very rare entity with higher mortality compared to bilateral cardiogenic pulmonary edema. Severe mitral valve regurgitation is almost always the valvular cause. Other etiologies might include vascular or bronchial obstruction, congenital heart disease or prolonged rest on one side. Our patient had unilateral right lung infiltrate with pleural effusion in setting of uncontrolled atrial fibrillation, severe aortic valve stenosis, and mild mitral valve regurgitation. Other possible causes of a unilateral infiltrate including pneumonia, malignancy, and pulmonary embolism were excluded. The patient improved dramatically on diuresis indicating pulmonary edema as the cause of the infiltrate.

#### Conclusion

Unilateral pulmonary edema is rare entity and associated with higher mortality secondary to misdiagnosis. It is vital to rule out malignancy and infection before considering pulmonary edema as the cause of any unilateral infiltrate on chest imaging.

**Author: Obiajulu Anozie, MD**

Additional Authors: Sharma, Anuj MD  
Hegazy, Housam MD  
Institution: SUNY Upstate Medical University

### **Title: A Rare Presentation of Infective Endocarditis Caused by Aerococcus Urinae**

Aerococcus Urinae is a relatively uncommon cause of urinary tract infections. It is typically seen in elderly individuals with underlying predisposing factors such as urinary tract pathologies, diabetes and cancer. In rare cases it has also been identified as the causative organism in septicemia and infective endocarditis.

A 51 year old male with a history of urethral stricture and fistula presented with a one week history of tachycardia, fever and chills, and confusion. His wife had also reported that her husband exhibited persistent dry heaving with one episode of coffee ground emesis. Upon admission he was found to be in atrial flutter with a heart rate ranging from 110-120 associated with a supply-demand cardiac ischemia. On examination he appeared to be in moderate respiratory distress with the use of accessory muscles. He exhibited diffuse, bilateral crackles as well as an irregular heart rhythm. Chest X-ray demonstrated pulmonary congestion which was correlated by a CT Thorax with findings consistent with pulmonary edema. Laboratory analysis showed a wbc count of  $13.2 \times 10^3/\mu\text{L}$  with neutrophilic predominance and urinalysis showed wbc count of 25 HPF with +1 bacteria. Urine cultures soon returned positive for  $> 100,000$  CFU/ml of Aerococcus Urinae which were subsequently detected on blood cultures as well. He was started on broad antibiotic coverage, placed on BiPAP and subsequently admitted to the CCU for closer medical management. TTE showed moderate aortic regurgitation however TEE was not pursued at that time due to his history of coffee ground emesis just prior to admission. Myocardial perfusion scan was also negative for any reversible areas of ischemia and his troponin leak was attributed to increased demand. He was aggressively diuresed and his heart rate was controlled with a cardizem drip. Sepsis from complicated UTI due to Aerococcus Urinae was treated with Vancomycin given the lack of antibiotic sensitivities and he was discharged on a two week regiment with close follow up with Infectious Disease. He developed progressively worsening shortness of breath and volume overload despite being discharged on a regiment of lasix and cardizem. During a follow up appointment with Cardiology he was referred back to the ED where a transthoracic echocardiogram showed a dilated left ventricle with reduced systolic function as well as a markedly dilated left ventricle. A TEE now demonstrated severe aortic regurgitation with mobile, echogenic tissue attached to the valve. Cardiothoracic surgery soon evaluated the patient and he underwent successful aortic valve replacement with a #23 Magna.

Infections due to Aerococcus Urinae are extremely rare and typically seen in elderly male patients with predisposing urological conditions. Given its potential for severe bloodstream infections with high fatality, physicians should consider endocarditis when the organism is identified in the blood.

## Resident/ Fellow Clinical Vignette

**Author: Steve Antoine, MD**

Additional Authors: Tiffany Hill

Kan Liu

Institution: Upstate medical University

### **Title: EKG changes captured during an episode of hypertensive emergency**

Hypertension is a known risk factor for congestive heart failure and coronary artery disease through a series of wear and tear which take years. Acute rise in blood pressure causing immediate end organ damage is known as hypertensive crisis. Lack of early signs of cardiac damages can lead to delay in treatment and adverse outcome. Hyperacute T-wave on EKG is known to be associated with myocardial infarction and often precede the appearance of ST-elevation or Q-waves. They are rarely seen with hypertensive crisis, their association has only been documented in experimental cases<sup>1</sup>. We present a case with EKG changes captured during an episode of hypertensive crisis and resolution in parallel with blood pressure control.

#### **CASE**

88 year old female with history of hypertension, TIA, anxiety disorder presented with slurred speech for 1 day. She did not have any other symptoms, did not complain of weakness or numbness. Her blood pressure was elevated at 183/55. Physical exam revealed an anxious elderly female with dysarthria, her lungs were clear, heart was regular rhythm without S3 or S4. The neurologic exam was otherwise normal. EKG on admission (fig1) was normal. Her cardiac enzymes were negative, CXR showed clear lungs, CT scan of the brain was negative. While in the ED she had a panic attack episode with palpitation severe anxiety and her blood pressure climbed to 214/71 with rates in the 130s. Her oxygen requirements went from room air to 3L nasal cannula. A follow up EKG was done (fig2) showed hyperacute T waves in the anteroseptal leads with non specific ST changes. CXR ordered showed cephalization of pulmonary vessels not present on admission, consistent with pulmonary edema. Patient was given sublingual nitroglycerin X2, a nitro patch and ativan for the anxiety, she was brought to the ICU for close monitoring. Her BP dropped to 144/72 after a few hours. A 3rd EKG was obtained (fig3) showing resolution of the hyperacute T-waves. Troponins trended up to 0.12 after 6 hours and 0.22 after 12 hours. Eventually the patient's oxygen requirement came back to baseline; her troponin trended down. Patient was transferred to a telemetry floor and discharged home on day3.

#### **DISCUSSION**

Acute rise in blood pressure can cause end organ damage rarely manifested clinically. This case demonstrates an anxiety triggered hypertensive crisis accompanied by neurologic and EKG changes which resolved in parallel with the fall in blood pressure. This patient also developed flash pulmonary edema and had cardiac enzymes elevation with peak consistent with the acute rise in blood pressure. Hyperacute T-waves on EKG can be associated with myocardial infarction and often precede the appearance of ST elevation; as far as we know they have only been demonstrated experimentally with hypertensive crisis<sup>1</sup>.

**Author: Bishow Aryal, MD**

Additional Authors: Ashutossh Naaraayan MD, Prakash Acharya MD, Prasanta Basak FACP, Stephen Jesmajian FACP

Institution: Montefiore New Rochelle Hospital

### **Title: DIFFUSE ALVEOLAR HEMORRHAGE SECONDARY TO WARFARIN TOXICITY**

#### **Introduction**

Diffuse alveolar hemorrhage (DAH) is a potentially life-threatening condition, presenting with hemoptysis, anemia, dyspnea and hypoxic respiratory failure. It is usually associated with autoimmune vasculitis or connective tissue disorders. DAH is an extremely rare complication of supratherapeutic anticoagulation with warfarin therapy. We present a patient who developed DAH while on warfarin.

#### **Case Presentation**

A 62 year old male with a history of CHF and right leg DVT, presented with hemoptysis of 1 week duration. He denied any fever, sore throat, dyspnea, chest pain, nausea, melena and bleeding from any other site. He was non-adherent to his outpatient follow-up. He reported taking medications from leftover prescription bottles, including warfarin. Vitals were stable. Examination of the lung revealed bilateral diffuse rales in the mid to lower lung fields. There was no evidence of petechiae or ecchymosis, and the stool was negative for occult blood. Laboratory data revealed hemoglobin 8.1mg/dL with normal WBC and platelet counts. Prothrombin time was 280.80ms, and INR 25.40. CXR showed diffuse bilateral opacities. High resolution CT of the chest showed diffuse bilateral airspace opacities with ground glass density, suggestive of alveolar hemorrhage. He was given 8 units of fresh frozen plasma and vitamin K, with gradual correction of INR and resolution of symptoms. He also received 3 units of packed red blood cells. Workup for vasculitis, including ANA, Anti-proteinase 3 antibody, myeloperoxidase antibody, was negative. HIV and hepatitis B tests were negative, and complement levels were normal. Follow up CXR showed clearing of the infiltrates.

#### **Discussion**

DAH originates from the microvasculature in the lung alveoli. Common etiologies of DAH include systemic vasculitis, connective tissue disorders, bone marrow transplantation, medications and coagulation disorders. Clinical manifestations are hemoptysis, dyspnea, respiratory failure and anemia. CXR findings showing diffuse alveolar infiltrates and CT scan revealing diffuse bilateral ground glass opacities are highly suggestive of DAH. The diagnosis may be confirmed with bronchoscopy with bronchoalveolar lavage. Warfarin related bleeding is seen in 10-16% of cases annually. Major risk factors for bleeding while on warfarin are patients with heart failure, non-adherence to therapy, elderly, presence of an acute illness and medication interactions. Non-adherence to follow-up and heart failure in our case, led to development of a supratherapeutic INR and subsequent DAH. Warfarin toxicity manifesting as DAH is extremely rare with a handful of reported cases in medical literature. Treatment consists of holding warfarin and rapid reversal of INR with fresh frozen plasma or prothrombin complex concentrate, and intravenous vitamin K therapy.

#### **Conclusion**

Close follow-up of patients on anticoagulation is of utmost importance to prevent life-threatening bleeding. Awareness about the rare possibility of DAH in patients with supratherapeutic INR and typical symptoms and radiologic findings may help initiate aggressive treatment measures and improve outcomes.

## Resident/ Fellow Clinical Vignette

**Author: Tarek Ashour, MD**

Additional Authors: Audi A, Naidu Y

Institution: Rochester Regional Health System, Unity Hospital

**Title: A case of temperate pyomyositis in a healthy young runner**

A 23 year old previously healthy male long distance runner presented to the emergency department (ED) with left hip pain of two weeks duration. He was initially examined by his sport medicine doctor. A hip x-ray was done and was unremarkable. He was treated for possible muscular sprain. He presented to the ED because his pain was gradually progressing and limiting his daily activities. He had no history of recent illness, trauma, IV drug abuse, skin breakdown, rash or insect bites. On examination, he had a temperature of 36.7, pulse of 57, respiratory rate of 18, oxygen saturation of 99% on room air and blood pressure of 120/69. He had no murmur. He had tenderness of his inner thigh muscles and lumbar area, but no swelling or erythema were noted. A psoas sign was positive bilaterally. His muscular strength was diminished in the hip flexors bilaterally. No sensory deficit was noted. He had a leukocytosis of 12.1 with neutrophilic predominance and elevated ESR of 38 and CRP of 8.428. MRI of the pelvis and lumbar spine showed multiple fluid collections within the musculature of the thighs, pelvis, and rectus abdominis muscles. He had no stigmata of endocarditis. He was diagnosed with pyomyositis. Ultrasound guided aspiration of the rectus abdominis yielded purulent fluid which grew Methicillin Sensitive Staphylococcus Aureus (MSSA). Results of the blood and urine cultures showed MSSA as well. A screening test for HIV was negative. A CT scan of the chest showed septic emboli in the lungs. A transesophageal echocardiogram revealed no valvular vegetations. The patient was treated with IV antibiotics for 42 days and his condition improved.

Discussion

Pyomyositis is an acute bacterial infection of skeletal muscle, typically involving the larger muscles of the lower extremities and trunk. It is endemic in tropical areas. By contrast, it is uncommon in non-tropical areas. Temperate pyomyositis has been described in patients who have HIV disease, diabetes, immunosuppression, IV drug abuse, and trauma, including exercise-induced microtrauma which may be the case in our patient. It has been divided in to three stages including invasive stage, purulent stage and late stage. Most of the patients are first seen in the purulent stage because of the presence of fever, chills and progressive pain. MRI is the imaging modality of choice for the diagnosis of pyomyositis. It is a life threatening condition. Diagnosis in early stages is challenging and requires high clinical suspicion. A delay in diagnosis can lead to sepsis and death. As it is an infection due to hematogenous spread, evaluation for endocarditis is necessary. Early systemic antibiotics are the mainstay of treatment and can eliminate the need for surgical drainage in selected cases.

**Author: Sayf Bala, MD**

Additional Authors: Vincent Skovira

Institution: UHS Wilson Hospital

**Title: Doxycycline and Acute pancreatitis**

introduction: Drug-induced pancreatitis (DIP) is a rare problem in medicine. Though DIP is a rare etiology of acute pancreatitis compared to other common etiologies like alcohol, gallstones, hypercalcemia, hypertriglyceridemia, infection, trauma or medical procedures like endoscopic retrograde cholangiopancreatography (ERCP), its incidence continues to rise. We report a case of a 67 year-old male with DIP following doxycycline treatment for Lyme disease. Case: 67-year-old male, with history of Diabetes and Coronary artery disease, presented with severe abdominal pain associated with nausea and vomiting. He had normal vital signs, Abdominal exam showed soft, tender abdomen in the epigastric region. Labs revealed a lipase level that was >2500U/L and triglyceride level of 105mg/dL. Patient denied alcohol use. A computed tomography without contrast of the abdomen showed evidence of edema indicative of acute pancreatitis. A right upper quadrant ultrasound was negative for gallstones or dilated biliary duct. The patient was recently diagnosed with Lyme disease and was treated with a 21-day course of doxycycline 100mg PO daily which concluded five days prior to symptoms. He was started on aggressive fluid resuscitation as well as empiric ciprofloxacin and metronidazole. With common etiologies ruled out we concluded that the patient had drug-induced pancreatitis secondary to doxycycline use.

Discussion: Drug induced pancreatitis (DIP) constitutes 1.4% of acute pancreatitis cases. In the literature, Tetracycline group including demeclocycline and minocycline were reported 5 times as the causing factor for DIP while in particular Doxycycline was reported three times as the etiology of acute pancreatitis and one time in association with ornidazole as the trigger factor.

In these cases The incidence of these event was variable ranging between 3 days to 14 days while on the treatment and 5 days after discontinuation of doxycycline. In addition to the prior reported cases our case was not associated acute laboratory or radiologic complications of pancreatitis and the clinical course showed complete resolution of the DIP with the appropriate management.

In conclusion, Our case represents the 3rd case reported in the English literature and it carried score of 5 based on Naranjo et al probability scale. Unfortunately it's not clear yet who are prone to develop DIP from doxycycline therefore physicians should be cautious in detecting early sign and symptoms of acute pancreatitis in patients who have recent exposure to doxycycline .in such circumstances doxycycline should be discontinued and re-exposure should be prohibited.

## Resident/ Fellow Clinical Vignette

**Author: Jamila Benmoussa, MD**

Additional Authors: Marie Chevenon, Minesh Nandi, Jay Nfonoyim MD, Thomas Forlenza MD  
Institution: Richmond University Medical Center

**Title: Ibuprofen Induced Thrombotic Thrombocytopenic Purpura**

Thrombotic thrombocytopenic purpura (TTP) is a multi-organ disease characterized by a pentad of symptoms (neurologic manifestation, renal disease, microangiopathic hemolytic anemia, thrombocytopenia, fever). Common causes of TTP include idiopathic, congenital, infections, drug-induced, malignancy and pregnancy. In the last 40 years despite the common use of non-steroidal anti-inflammatory drugs (NSAIDs), only two cases of TTP induced by NSAIDs have been reported. In this study, we describe a case of a 37 year-old African American male who was found unresponsive on the floor. It was later found that he ingested 30 ibuprofen pills (400 mg per pill) with a total dose of 12 grams in an attempt to commit suicide. On initial examination, the patient was confused and unable to give pertinent history. The patient had a temperature of 37.4°C, heart rate of 112 beats per minute, blood pressure of 151/81 mmHg and a respiratory rate of 20 breaths per minute. The patient was noted to have a pale conjunctivae, scleral icterus, vitiligo and left arm weakness. Upon Foley catheter placement, 100 cc of grossly bloody urine were collected. The rest of the physical examination was within normal limits. Complete blood count revealed hemoglobin of 6.3 mg/dL, hematocrit of 19.1% and platelet count of 29000/mcL. Serum electrolytes were within normal limits. The patient was treated initially in the emergency department for severe anemia secondary to hematuria and received one unit of packed red blood cells and one unit of platelets. Afterward, there was no significant rises in platelets or hematocrit level were noted. Later on, a diagnosis of TTP was more evident based on clinical findings and laboratory results; schistocytes were appreciated on the peripheral blood smear along with hemolytic anemia, severe thrombocytopenia, acute kidney injury and an altered mental status. A disintegrin and metalloprotease with a thrombospondin type 1 motif, member 13 (ADAMTS13) level was noted to be less than 3%, a low haptoglobin of <15mg/dL, lactate dehydrogenase of 2308 units/L confirming a diagnosis of TTP. TTP complications developed in this patient due to his high thrombotic state. He suffered a non-ST elevation myocardial infarction (NSTEMI) as well as a right parietal infarct. The patient was treated in the hospital where he received plasma exchange for 3 weeks, along with prednisone and rituximab. His ADAMTS13 levels and platelets normalized and the patient recovered eventually. This case illustrates an unusual cause of TTP, where ibuprofen induces antibodies against ADAMTS13. Thus, recognizing TTP induced by NSAIDs is essential to initiation of appropriate therapy and prevention of serious morbidity and mortality.

**Author: Ahmad Cheema, MD**

Additional Authors: Hafeez-ul-Hassan M.D, Abdul Mannan M.D, Maryum Hussain M.D  
Institution: Icahn School of Medicine at Mount Sinai/St. Luke's Roosevelt Hospital

**Title: A case of pulmonary complications after subcutaneous injections of Polyacrylamide Hydrogel**

Introduction:

Injectable fillers are one of the popular non surgical treatments for wrinkles and facial contouring in Europe. One such filler is Polyacrylamide hydrogel (Aquamid) which is currently approved in various countries for facial contouring and correction of HIV lipoatrophy. However, it is not FDA approved in the United States. Several adverse effects of Polyacrylamide Hydrogel have been reported in the literature, including local infection, inflammation, pain, nodule formation and delayed hypersensitivity reaction. However, no pulmonary complications have yet been reported.

Case description:

A 26 year old female with no history of smoking, oral contraceptive use or recent long travel presented to the emergency department with worsening shortness of breath, cough and substernal chest discomfort since four days. Patient was in her usual state of health four days ago when she had bilateral multiple injections of Polyacrylamide Hydrogel in the buttocks for cosmetic enhancement. A few hours after the procedure, she developed dry cough, chest discomfort and dyspnea on exertion and with conversation which gradually worsened. On arrival in the ED, patient was noted to be tachypneic to 40s with oxygen saturation of 88% on room air. Stigmata of recent injections were seen on buttocks. Chest xray showed bilateral confluent opacities in mid and lower lung zones. CT angio chest demonstrated extensive diffuse ground glass opacities bilaterally and right heart strain pattern, but no definite embolus. EKG showed Right Bundle Branch Block pattern. Patient was eventually intubated for acute hypoxic respiratory failure. Diffuse alveolar hemorrhages were seen on Bronchoscopy with no overt source of bleeding. Cytology of BAL was positive for alveolar macrophages and mixed inflammatory cells. Work up for connective tissue and autoimmune diseases was negative. Patient's condition improved with steroids and empiric antibiotics and was eventually extubated. CT chest after one month demonstrated complete resolution of bilateral opacities.

Conclusion:

Illegal and non-approved use of Polyacrylamide hydrogel can lead to serious consequences. Our case showed pneumonitis and alveolar hemorrhage after hydrogel injection in the buttocks for augmentation which likely resulted in systemic embolization. As this is the first report of pulmonary complications secondary to hydrogel injection, pathogenesis is not yet clearly understood. Possible mechanisms include accidental injection into the venous system or migration from interstitial subcutaneous tissue into the general blood stream. When patients develop acute respiratory failure after cosmetic procedures, embolism of injected material should always be in the differential diagnoses. Also, use of unapproved substances must be discouraged.

## Resident/ Fellow Clinical Vignette

**Author: Matthew Clarke, MD**

Additional Authors: Jamila Benmouss, MD, Amulya Penmetsa MS4, Philip Otterbeck MD, Farhang Ebrahimi MD  
Institution: Richmond University Medical Center

### **Title: A Case Dilemma: Inaccuracies of HbA1c in Measuring Glycemic Control in a Patient with Liver Cirrhosis**

For decades, hemoglobin A1c (HbA1c) has been the standard measure of long-range glycemic control in patients with diabetes type 2 (T2DM) and liver cirrhosis. However, there is a subset of patients in whom there will be discordance between HbA1c and blood glucose measurements, rendering it less useful in determining a patient's glycemic status.

We present a case of 61 year old Caucasian female with history of Hepatitis C-related liver cirrhosis and T2DM on metformin (HbA1c in 2013 was 13.7%), who presented with shortness of breath and increasing abdominal girth to our institution.

Physical exam revealed pallor, a grossly distended abdomen with a positive fluid wave, splenomegaly, and a protruding, yet reducible, umbilical hernia.

Initial labs showed that the patient had anemia of chronic disease, acute kidney injury, elevated liver function tests, elevated lactate dehydrogenase, admission finger stick glucose was 568 mg/dl, and HbA1c of 5.5%.

HbA1c can be calculated by averaging seven fingerstick glucose readings per day. In our patient's case, this value was calculated to be 12.08%. Given the discordance between the blood glucose measurements, Calculated HbA1c and the measured HbA1c, other possible etiologies rather than liver cirrhosis that would explain this laboratory "mismatch" (example lab error, compromised glucometer, hemoglobinopathies, or hemolytic anemia) were excluded.

Falsely low than expected HbA1c values can be expected in patients with a wide range of diseases that affect availability of glucose, glycation rate, and erythrocyte lifespan. These include the aforementioned hemolytic anemia and hemoglobinopathies, as well as renal failure and liver cirrhosis, where hypersplenism can lead to hemolysis and a shorter erythrocyte lifespan.

Other laboratory parameters have emerged as being clinically useful in measuring glycemic status in patients with diabetes and liver cirrhosis: fructosamine, glycated albumin, and a measure known as chronic liver disease-A1c (CLD-A1C). In fact, our patient had an elevated fructosamine of 415 µmol and an elevated glycated albumin of 4.7%, consistent with a hyperglycemic state over the past two to three weeks.

There are limitations, however, to each of the previously-mentioned measurements. Since CLD-A1c has not been well-studied in patients with kidney disease, this value was not as given our patient's history of acute kidney injury. In addition, fructosamine and glycated albumin are both found to be affected in patients who have proteinuria or decreased albumin levels. Typically, the best proposed methods of measuring glycemic status is frequent fingerstick glucose monitoring and HbA1c. However, this case illustrates the importance of the use of other markers to evaluate glycemic control in patient with discordance between HbA1c and blood glucose measurements; taking into account the limitations of each measurement based on an individual patient's comorbidities.

**Author: Siddhant Datta, MD**

Additional Authors: Shanchiya Ravindradas, MD  
Ann Falsey, MD  
Institution: Rochester General Hospital

### **Title: Sometimes a Runny Nose is More Than -Just a Runny Nose!!**

Background: Streptococcus mitis is prevalent in the normal flora of the oropharynx. It is generally considered to be a less virulent pathogen but it may cause life threatening infections including subacute bacterial endocarditis. Meningitis with S.mitis in the absence of endocarditis is rare. We report a case of S.mitis meningitis in which eliciting a detailed past medical history was key to making the correct diagnosis.

Case: 52 year old female with hypertension, allergic rhinitis and recurrent sinusitis presented with one day of chills, headache, neck stiffness and photophobia. On exam she was lethargic with a stiff neck and an otherwise normal exam. CT head was negative for acute bleed, infarct or sinusitis. Lumbar puncture was performed after one dose of antibiotics and CSF revealed ~6000 nucleated cells with 90% polymorphs, glucose of 26 and proteins of 446. Gram stain did not reveal any organisms. Empiric treatment was initiated with antibiotics and steroids. CSF was sterile but 2 sets of blood cultures grew Streptococcus mitis. Antibiotics were narrowed to Ceftriaxone and the patient rapidly improved. TTE and TEE were negative for valvular abnormalities and vegetations. Because of the unusual organism her history was reviewed. She again related having allergic rhinitis and recurrent sinus infections. When probed for more details she described persistent, positional clear rhinorrhea for 2-3 years, stating she could "water the plants" with her nose. Of note, she had a motor vehicle accident three years prior with a concussion. Nasal fluid analysis was positive for glucose and Beta-2 transferrin and CSF cisternogram was done which showed CSF leak in the region of the right anterior ethmoid air cells/cribriform plate. She recovered completely with a 10 day course of ceftriaxone. Repair of the leak by the neurosurgical service was performed 2 months after discharge.

Discussion: Bacterial meningitis resulting from CSF leakage secondary to trauma is an uncommon but well described phenomenon. An accurate history is paramount in making a proper diagnosis. It is important to query patients regarding their symptoms when self "diagnoses" such as sinusitis are provided. This patient tended to minimize and ascribe symptoms to illnesses with which she was familiar but when prompted, she eventually provided classic symptoms of a CSF leak to explain her unusual bacteriology. Establishing a diagnosis is important for long term prognosis in patients with CSF leak. An episode of meningitis with resultant inflammation may seal the defect, but some patients continue to leak and are at risk for recurrent meningitis. Neurosurgical evaluation should be sought for possible repair.

## Resident/ Fellow Clinical Vignette

**Author: Siddhant Datta, MD**

Additional Authors: Farrukh Abbas, MD  
Y. Abbas  
Institution: Rochester General Hospital

### **Title: Mysterious Case of Migratory Polyarthritits in Pancreatic Cancer**

Background: Cancer polyarthritits is an uncommon, para-neoplastic manifestation of some solid tumors and hematologic malignancies. It is most commonly seen with lung cancer and there have been very few cases of migratory polyarthritits in pancreatic carcinoma. It generally occurs in elderly patients and is characterized by abrupt onset of asymmetric arthritits, often involving large joints of the lower extremities, sparing the joints of the hands.

Case: A 58 year old man presented with a 2 week history of abdominal pain. CT abdomen showed a large mass involving the pancreatic tail and spleen with liver metastasis and liver biopsy confirmed adenocarcinoma of pancreas. Two days after admission, left hip pain developed associated with warmth, tenderness and restricted range of motion. Bone scan did not reveal any bony metastasis and MRI of left hip was negative for osteonecrosis. The hip pain resolved spontaneously after approximately 36 hours, but 2 days later, bilateral arthritits involving both ankles developed with pain, swelling, warmth, erythema, tenderness, restricted ROM and inability to bear weight. The patient had no previous history of arthritits or any rheumatologic disease. Workup was negative for Rapid Flu/RSV, HIV panel, GC screen, hepatitis panel, ANA and RF. Renal function was normal; uric acid was 4 mg/dl, ESR and CRP were elevated to 60 and 192 respectively. Given negative workup patient was started on NSAIDs for possible migratory polyarthritits. His arthritits improved to great extent till discharge on the 10th hospital day.

Conclusion: Migratory polyarthritits can present as para-neoplastic syndrome in pancreatic cancer patients and can be a diagnostic challenge in clinical settings. The differential diagnosis include gout, infectious arthritits, reactive arthritits, bony metastasis and avascular necrosis if the patient taking steroids. Migratory polyarthritits as Para-neoplastic syndrome in pancreatic cancer patients is rare, so its diagnosis can be missed. The condition usually responds to non-steroidal anti-inflammatory medications. If paraneoplastic rheumatism does not respond to conventional drugs, they usually regress with treatment of cancer.

**Author: Samit Datta, M.D.**

Additional Authors: Dr. Niket Sonpal  
Dr. Sherri Sandel  
Dr. Sonja Olsen  
Institution: Lenox Hill Hospital

### **Title: The Paan Pancreatitis Problem**

Paan is a commonly abused substance in Southeast Asian countries and India, which is mainly composed of the betel leaf and areca nut (1). Nicotine, caffeine, and alcohol are three common addictive substances that are more commonly used than paan, however like cigarettes the total number of additives is unknown (1). One mouse-model study showed the betel causing increased pancreatic lipase stimulation, and decreased trypsin and chymotrypsin secretion (2). In this case study, we present a case where Paan is the presumed causative agent of pancreatitis in an 18 year-old female.

An 18-year-old Bangladeshi female was recently discharged from an outside hospital with a diagnosis of pancreatitis for similar symptoms. Following discharge, patient reported the pain never fully resolved and worsened 3 days prior to presentation. Patient reported no alcohol or cigarette use, no trauma, and no recent travel. She denies any medication use, and was not on antibiotics prior to admission. She did, however, endorse paan usage for approximately 4-5 years, and has noted issues with anorexia with a 12 pound weight loss and nausea for approximately 1 year. Physical exam was notable for significant tenderness in the epigastrium. Notable labwork showed Lipase of 20251 U/L, and a normal triglyceride level. An ultrasound showed no evidence of gallstones, and a CT of the abdomen and pelvis which showed mild stranding and fluid surrounding the pancreas, and MRCP which showed acute pancreatitis, with no evidence of cholelithiasis, choledocholithiasis, or evidence of pancreas divisum. Patient was also found to be negative for HIV, VZV, CMV, and mumps.

Pancreatitis is a very severe disease that has been well described in the literature with clear evidence for risk factors. Our patient was negative for alcohol use, gallstones, hypertriglyceridemia, trauma, and medication use. While there has been one study linking the use of smokeless tobacco to pancreatic cancer in the Swedish population, there is very little research for the effects of paan use on the human body. This case report demonstrates a need for prospective studies to detail and evaluate the exposure to paan in the budding immigrant Indian and Southeast Asian population and the deleterious health effects, namely pancreatic stimulation and pancreatitis.

1. Garg A, Chaturvedi P, and Gupta PC. A review of the systemic adverse effects of the areca nut. *Indian J. Med Paediatr Oncol.* 2014 Jan-Mar; 35(1): 3-9.
2. Prabhu MS, Platel K. Effect of orally administered betel leaf on digestive enzymes of pancreas and intestinal mucosa and on bile production in rats. *Indian J Exp Biol.* 1995 Oct; 33(10): 752-6.

<p><b>Author: Evan Diamond, M.D.</b>                  Additional Authors: Evan Diamond, M.D., Roxana Lazarescu, M.D.                  Institution: New York Presbyterian Queens</p> <p><b>Title: What can happen when the Internet is your Primary Provider-- A case of a Hydrogen Peroxide Enema</b></p> <p>Background                  Today people are using the Internet as a source of information for all things medical. The use of Google, Yahoo and illegitimate medical websites has misled the public into unsafe practices. Hydrogen peroxide has been used therapeutically in a variety of settings for nearly 100 years. Clinical applications involving the gastrointestinal tract include relief of fecal impaction or meconium ileus and image enhancement in radiological procedures, although most of these practices have been abandoned.</p> <p>Case presentation                  43-year-old female with past medical history of chronic constipation and anxiety presented to the emergency department complaining of rectal pain and bright red blood per rectum for the past twelve hours. Patient reports constipation for the previous ten days. She reported administering 60 mL of 3% hydrogen peroxide solution diluted with 60 mL of tap water and followed directions from an online website. The patient immediately emptied her bowels with relief, however 3 hours later, she started having excruciating pain and bleeding. She went to an urgent care facility where they gave her unknown antibiotics, however the pain and bleeding did not subside. Physical examination revealed a soft abdomen with diffuse tenderness over the epigastrium. Rectal examination revealed diminished anal tone and bright red blood in the anus. The rest of her exam was normal. Laboratory findings were significant for leukocytosis with a left shift, and acute blood loss anemia. Computed tomography Abdomen/Pelvis was done which showed rectosigmoid wall thickening with extensive surrounding inflammatory changes consistent with colitis. The patient was managed conservatively with analgesics, antibiotics including Flagyl and Levaquin as well as given Miralax two times a day. Rectal bleeding resolved within 72 hours. The patient was discharged home with instructions to take budesonide 9 mg daily for 14 days and to follow up as an outpatient for which she had complete resolution of symptoms.</p> <p>Discussion                  In this case, the patient did not consult with a physician prior to administration of a hydrogen peroxide enema, which ultimately led to unsafe practices. Fox and Duggan (2013) found that 72% of adults who reported utilizing the Internet indicated they had looked online for health information in the past year. Fox and Duggan (2013) also found that 77% of online searches for health information came from search engines such as Google, Bing, and Yahoo, while only 13% of searches started from sites specializing in health information, such as MDWEB. Tustin (2010) found that patients who felt a lack of empathy from, or lack of quality time with, their provider were more likely to search for health information online. Thus, physicians must be wary of this and educate patients to consult with them prior to making medical decisions on their own.</p>	<p><b>Author: Passang Dolma, MD</b>                  Additional Authors: Sofia Turner (ACP Member)                  Institution: MONTEFIORE MEDICAL CENTER, WAKEFIELD CAMPUS</p> <p><b>Title: CONSIDER VALPROIC ACID INDUCED HYPERAMMONEMIA IN ACUTE ENCEPHALOPATHY</b></p> <p>Introduction                  Valproic acid (VPA) is generally a well-tolerated anticonvulsant and used in treating many types of epilepsy. However, it is associated with both neurologic and systemic side effects, one of which is valproate-induced hyperammonemia (VIH), which may cause acute encephalopathy.</p> <p>Case Presentation                  39 year-old man with history of mental retardation, autism, seizure disorder and hypothyroidism who at baseline was ambulatory and nonverbal was sent from group home after having five episodes of generalized tonic-clonic seizures. He was adherent with his antiepileptic medications (keppra and valproic acid) and had no recent infectious symptoms. Physical exam was notable for lethargy with easy arousability to verbal stimuli without focal neurologic deficits. Initial blood glucose level was 76 mg/dl and valproic acid was 92.2ug/mL (50.0-140.0 ug/mL). Laboratory studies were notable for normal electrolytes, lack of leukocytosis and normal thyroid function tests. CT head was unremarkable. He was evaluated by Neurology with recommendations to increase dose of antiepileptics. An EEG ruled out non-convulsive status. Due to continued lethargy, an ammonia level was checked which was elevated at 135 umol/L (11-35 umol/L). Lactulose therapy was initiated with subsequent improvement in mental status and return to baseline the next day. Hyperammonemia was attributed to VPA and the dose was adjusted prior to discharge.</p> <p>Discussion                  The incidence of valproate induced hyperammonemia has not been well established, with studies citing a range of 16-52%. Hyperammonemia may occur with both therapeutic and supratherapeutic concentrations of VPA. The mechanism of hyperammonemia is believed to be related to propionic acid (a metabolite of VPA), which inhibits carbamoyl phosphate synthetase, an enzyme necessary for ammonia elimination via the urea cycle. Valproate-induced hyperammonemia may occur in both acute overdose and chronic use. Symptomatic hyperammonemia from valproate therapy, referred to as valproate-Induced hyperammonemic encephalopathy (VHE), manifests as confusion, lethargy, vomiting, increased seizure frequency and may progress to coma and death. Onset of VHE can be sudden with a valproate loading dose or insidious with chronic therapy. Furthermore, encephalopathy may develop in patients previously taking VPA. However, the degree of encephalopathy is not related to serum VPA level as evidenced in our patient with a normal serum level. Possible risk factors for VHE seem to be poor nutritional status, carnitine deficiency, congenital urea cycle disorder, and other antiepileptic medication interactions. Majority of patients with VHE experience mild to moderate lethargy and recover uneventfully with rapid medication discontinuation and lactulose use. Severe cases may require urgent dialysis to treat this life threatening condition.</p> <p>Learning points                  Clinicians should consider valproate-induced hyperammonemia in patients taking VPA and presenting with lethargy, gastrointestinal symptoms and decreased level of consciousness. The mainstay of treatment for valproate-induced hyperammonemic encephalopathy remains timely diagnosis and discontinuation of VPA therapy.</p>
---	---

## Resident/ Fellow Clinical Vignette

<p><b>Author: Granati Glen, MD</b> Additional Authors: Institution: Stony Brook University, Internal Medicine</p> <p><b>Title: Does Trending Procalcitonin in the ICU Provide Effective Monitoring? or a False Sense of security?</b></p> <p>Case: A 55 year old female presented with decreased oral intake, right gluteal cellulitis, severe anion gap metabolic acidosis (anion gap = 23), severe prerenal azotemia (BUN = 94 mg/dL, creatinine = .49 mg/dL) and initial blood cultures grew methicillin resistant Staphylococcus epidermidis and methicillin sensitive Staphylococcus aureus. Subsequently the patient developed SIRS, was volume resuscitated, and became hypoxic. Chest X-ray showed pulmonary edema and bilateral airspace disease. She was upgraded to the intensive care unit (ICU) and treated with daptomycin for sepsis presumed secondary to cellulitis. Patient declined further CT imaging at that time. Procalcitonin (PCT) was trended in the ICU to evaluate the antibiotic's effectiveness in eliminating infection. PCT was 61.35 ng/ml on ICU admission and trended to 1.86 ng/ml over six days on daptomycin. On ICU day five she developed hypoxia, hypercapnia and worsening shock requiring intubation and three vasopressors. The patient's family agreed to a CT chest which revealed extensive bilateral pneumonia. Antibiotics were changed to vancomycin and cefepime to treat pneumonia. The patient worsened, PCT remained low, and she was eventually terminally extubated.</p> <p>Discussion: Procalcitonin is a peptide precursor that is increased with bacterial infections. PCT has been used to distinguish bacterial from viral infection and to differentiate noninfectious causes of SIRS from sepsis. Additionally PCT has been used to facilitate earlier discontinuation of antibiotic therapy (particularly in pneumonia). Use of PCT in ICUs and emergency departments to guide diagnosis, antibiotic treatment and illness severity has become common practice although there is little evidence to support outcomes such as length of stay or ability to detect nosocomial infection. Daptomycin has activity against gram positive bacteria, but it is ineffective in lung infection due to inactivation by surfactant. Our patient's PCT declined precipitously during daptomycin treatment while developing severe pneumonia. Literature search did not reveal evidence to suggest that trending PCT predicts antibiotic effectiveness. Our patient's decline in PCT despite developing fulminant infection suggests that perhaps daptomycin was effective in eliminating bacteria in the patient's bloodstream. This may have resulted in a decrease in PCT despite lack of effective treatment for the patient's primary source of infection (pneumonia). The case suggests that PCT elevation is more specific to bacteremia and sepsis than it is for isolated bacterial infections (e.g. pneumonia); and that declining PCT with treatment measures a decrease in bacteremia and not necessarily effective treatment of an infection's primary focus (pneumonia in this case). Our case also suggests that tracking PCT may be ineffective at detecting new infections in hospitalized patients already started on antibiotics. Lastly the case suggests that PCT may not be useful in tracking the overall effectiveness of antibacterial therapy in inpatient populations.</p>	<p><b>Author: Granati Glen, MD</b> Additional Authors: Sherise Rogers Institution: Stony Brook University, Internal Medicine</p> <p><b>Title: R-CEP Provides Favorable Outcome in Treatment of Primary Cardiac Lymphoma</b></p> <p>We present a case of a 72 year old male with a past medical history significant for paroxysmal atrial fibrillation, inferior wall myocardial infarction, and known pulmonary, renal, and cutaneous sarcoidosis who comes to the hospital for an elective atrial fibrillation radiofrequency ablation (RFA). A transesophageal echocardiogram performed prior to the procedure revealed a large tissue density within the pericardial space, adjacent to the free wall of the right ventricle and left atrium, and invading the myocardium with extension through the interatrial septum. The RFA was not performed and the patient was admitted for further workup and management of a new cardiac mass initially suspected to be cardiac sarcoidosis. Subsequent work up included a cardiac MRI which demonstrated a dense 7.5 cm by 3.5 cm mass involving the right ventricle and right atrium, wrapping around the great vessels. Cardiac MRI performed three years ago did not display such abnormality. Hence, a percutaneous endomyocardial biopsy was performed which yielded pathology findings consistent with CD30 positive diffuse large B cell lymphoma (DLBL). The patient who remained stable during this hospitalization was then discharged and obtained follow up care with an oncologist. He then underwent PET scanning, demonstrating absence of extracardiac lesions; confirming the diagnosis of primary cardiac lymphoma (PCL). Subsequently, the patient underwent two cycles of chemotherapy with rituximab, cyclophosphamide, etoposide, and prednisone (R-CEP). Post chemotherapy transthoracic echocardiogram did not visualize a mass and the patient's ejection fraction remained the unchanged at 50%.</p> <p>Discussion: Primary cardiac lymphoma is a very rare disease with an associated low prognosis. It accounts for approximately 1% of intrinsic cardiac tumors and 0.5% of extranodal non-Hodgkin lymphomas. As a variant of DLBL, it is most common among immunocompromised patients. Prognosis in PCL is less than one month without treatment with some living up to five years with treatment. Radiologic diagnosis of PCL is minimally standardized. Most tumors are discovered incidentally on imaging, while approximately 25% are found postmortem. MRI has the highest antemortem sensitivity for cardiac tumors. Standard treatment of PCL is typically R-CHOP or it's variants, with or without radiation. R-CEP is an alternative chemotherapy regimen for non-hodgkins lymphoma, utilized commonly in the elderly to decrease anthracyclin induced cardiotoxicity . What is unique about this case is that our patient, had optimal response to R-CEP with no evidence of tumor on echocardiogram following two treatment cycles. Additionally, our patient's ejection fraction remained unchanged, at 50%, after therapy. This case of complete remission with R-CEP without associated cardiotoxicity suggests that R-CEP may be a preferable treatment regimen for patients with PCL compared to standard DLBL therapy.</p>
---	---

<p><b>Author: Naveen Gnanabakthan, MD</b>                  Additional Authors: Michael Kosters, Devamohan Sivalingam, Konrad Dziamski, Brian Changlai Jr.                  Institution: SUNY - Upstate Medical University, Dept of Medicine</p> <p><b>Title: Type II Congenital Pulmonary Airway Malformation (CPAM) presenting in adulthood</b></p> <p><b>INTRODUCTION:</b> Congenital pulmonary airway malformation (CPAM) is a rare congenital anomaly involving the lower respiratory tract, with an incidence of 1 out of 8300 to 30,000 pregnancies(1). The majority of CPAMs are identified on prenatal ultrasound or are detected early in the post-natal period due to respiratory compromise(2). We present a rare case of CPAM discovered in an otherwise healthy young male.</p> <p><b>CASE PRESENTATION:</b> A 24-year old male presented with exertional dyspnea over a 4 day period. He denied any cough, fevers, weight loss or night sweats. He denied tobacco use and family history was non-contributory. Vitals and physical examination were unrevealing, however labs were notable for a mild leukocytosis. His chest x-ray (CXR) revealed a left lower lobe (LLL) infiltrate. Given his presentation, he was diagnosed with a community-acquired pneumonia and discharged home with antibiotics. Despite adequate treatment and an additional course of antibiotics, his symptoms persisted over the following 3 months and a repeat CXR was unchanged. A thoracic CT angiogram showed a 9 cm LLL lesion with a small cyst on the periphery, which had both pulmonary arterial supply and venous drainage without significant airspace disease, suggesting a possible congenital lesion. He then underwent resection of the LLL mass. Histology revealed a relatively well demarcated lesion characterized by multiple bronchiole-like structures, suggestive of a type II CPAM. Post-operatively, his dyspnea improved significantly.</p> <p><b>DISCUSSION:</b> Type II CPAM consist of 15-20% of all CPAMs(3). In addition to pulmonary involvement, type II CPAMs are often associated with other organ system anomalies, which were not present in our patient. The diagnosis of CPAM can usually be made through radiographic imaging. His dyspnea and failed response to multiple antibiotic regimens lead to the discovery of CPAM, unusually diagnosed in what had been a healthy adulthood. The relatively late presentation of his symptoms was likely related to the size of the lesion, which may have been too small to have caused respiratory dysfunction during the post-natal period. Although malignancy potential of type II CPAM is low, resection of the lesion is recommended in symptomatic patients.</p> <p><b>CONCLUSIONS:</b> CPAMs are typically discovered during the gestational or post-natal period, but rarely present in adulthood. The case highlights the importance of considering CPAM in young adults with a persistent CXR infiltrate that is unresponsive to repeated antimicrobial treatments.</p> <p>Reference #1 : Shanti CM, Klein MD. Cystic lung disease. <i>Semin Pediatr Surg</i> 2008; 17:2.</p> <p>Reference #2: Laberge et al. Outcome of the prenatally diagnosed congenital cystic adenomatoid lung malformation: a Canadian experience. <i>Fetal Diagn Ther</i> 2001; 16:178-86.</p> <p>Reference #3: Stocker JT, Drake RM, Madewell JE. Cystic and congenital lung disease in the newborn. <i>Perspect Pediatr Pathol</i> 1978; 4:93</p>	<p><b>Author: Umut Gomceli, MD</b>                  Additional Authors: Srijia Vangala MD, Paul J Kelly MD, Manisha Singh MD                  Institution: BRONX LEBANON HOSPITAL CENTER</p> <p><b>Title: An Unusual Case of Ototoxicity with Use of Oral Vancomycin</b></p> <p><b>Introduction:</b> Systemic absorption of oral vancomycin is poor particularly because of the size of the molecule and its pharmacokinetics. It has an elimination half life of 5- 11 hours in patients with normal renal function. We present a rare case of ototoxicity after oral vancomycin administration and detectable serum vancomycin levels 24hours after cessation of vancomycin.</p> <p><b>Case Presentation:</b> A 42 year old woman with history of hypertension, diabetes mellitus and previously treated Clostridium difficile colitis presented with abdominal pain and diarrhea for 2 weeks. Clostridium difficile infection was confirmed with PCR and patient had a normal renal function. Initially metronidazole was started but changed to oral vancomycin 125 mg every 6 hours due to intolerance. After three doses of oral vancomycin, patient reported lightheadedness, bilateral ear buzzing and whistling sensation and decreased hearing described as a sensation of "clogged ears". Patient reported to emergency room because of worsening of these symptoms. Vancomycin dosing was reduced to every 8 hours, but symptoms persisted. On day 3, vancomycin was discontinued with progressive resolution of symptoms over the next 12 hours. The serum random vancomycin level on day 4 was detectable at 2.181g/dL, 24 hours after last dose. Temporal association of patient's symptoms and improvement with cessation of therapy along with a detectable vancomycin level indicates systemic absorption of oral vancomycin with resultant ototoxicity.</p> <p><b>Discussion:</b> The potential for absorption of oral vancomycin is not well described and is attributed to compromised intestinal epithelium allowing for increased drug absorption. Some studies suggested that oral vancomycin may result in therapeutic or even potentially toxic levels of serum vancomycin in patients with impaired renal function. Ototoxicity may be transient or permanent side effect of vancomycin therapy and is related to high serum levels. Symptoms usually resolve after decreasing the dose or cessation of vancomycin. No detectable serum vancomycin levels were found in 98% of the patients treated with oral vancomycin in a prospective study. Our case is very interesting because despite normal renal function the patient developed ototoxicity and systemic absorption of the drug was confirmed with a measurable vancomycin level 24 hours after drug was discontinued.</p> <p>Oral vancomycin is generally considered a drug that is not systemically absorbed. Rare cases of toxicity and systemic absorption have been related to renal insufficiency. This case reflects that some patients may be more susceptible to increased systemic absorption of oral vancomycin. The possibility for ototoxicity should be considered and discussed with patients while prescribing oral vancomycin since it can potentially be permanent.</p>
--	--

## Resident/ Fellow Clinical Vignette

**Author: Kulothungan Gunasekaran, MBBS**

Additional Authors: Shashank Kotakonda MBBS, Ahmad Chaudhary MD, Michael Bauer MD, Edward Bischof MD  
Institution: Mary Imogene Bassett Medical Center

### **Title: Thick walled lung cavity as the sole presentation of Granulomatosis with Polyangiitis (Wegener's Granulomatosis)**

#### Introduction:

Thick walled cavity in Granulomatosis with Polyangiitis is not uncommon. It usually presents with other classic symptoms but isolated cavity is unusual. Here we describe a patient with thick walled lung cavity as sole presenting feature of Granulomatosis with Polyangiitis, who was on TNF inhibitor for Psoriatic arthritis.

#### Case:

58 year-old female with history of Psoriatic Arthritis (on Methotrexate, Adalimumab) and Pyoderma Gangrenosum with multiple non-healing skin ulcers presented to clinic with flu-like symptoms, cough and blood tinged sputum for one week. Augmentin was prescribed for presumed pneumonia. Chest X-Ray showed thick walled large cavitory lesion in the right upper lobe and nodules in her left lung. Augmentin was switched to Clindamycin and a pulmonary referral was made. Bronchoscopic washings were negative for malignancy. Infectious work-up for bacteria including tuberculosis and fungi remained negative. She underwent biopsy of the left lung nodule, histopathology of which revealed extensive necrotizing suppurative granulomatous inflammation consistent with Granulomatosis with Polyangiitis. PR-3 ANCA serology was strongly positive supporting the diagnosis. Her renal function was normal and weight remained stable. She was treated with Rituximab and Prednisone and responded well with improvement in her symptoms.

#### Discussion:

Cavities are relatively uncommon in many autoimmune diseases except Granulomatosis with Polyangiitis. It is a systemic vasculitis that almost always involves the upper or lower respiratory tract. Patients usually present with fever, migratory arthralgias, malaise, anorexia and weight loss. The frequent lung manifestations are nodules and infiltrates. Pulmonary cavities have also been observed by computed tomography in 35 - 50% of patients. As most patients with autoimmune diseases are treated with potent immunosuppressive agents, infectious etiologies for cavitory lesions should be thoroughly investigated. In our patient, we ruled out all infectious causes. She did not have any other symptoms or signs of Granulomatosis with Polyangiitis. This raises the question if chronic anti-TNF therapy for Psoriasis altered the presentation. TNF is critical for granuloma formation by up-regulating adhesion molecules that participate in cellular recruitment and lymphocyte activation, particularly macrophages. Review of the limited literature available showed no benefit to adjunctive anti-TNF (Etanercept) therapy for Granulomatosis with Polyangiitis, including remission maintenance.

#### Conclusion:

Granulomatosis with Polyangiitis should be suspected in patients with thick walled cavity who are treated with anti-TNF agents even in the absence of classic symptoms and signs. Further clinical studies are needed to evaluate the role of anti-TNF agents in this disease.

**Author: Sushilkumar Gupta, MD**

Additional Authors: Shyam Shankar MBBS., Karishma Kitchloo MD., Arun Kumar Arumugam Raajasekar MBBS.  
Chen Ioana MD., Abhinav Binod Chandra MD.  
Institution: Maimonides Medical Center

### **Title: Neurosarcoidosis Induced Longitudinal Extensive Transverse Myelitis**

#### Abstract:

Transverse myelitis is an uncommon neurological emergency encountered in daily practice. Longitudinal extensive transverse myelitis (LETM) is defined as a spinal cord lesion that extends over 3 or more vertebrae. We describe a 53 year old male with sudden onset of urinary retention and bilateral lower extremity paraplegia and diagnosed to have LETM secondary to neurosarcoidosis. Standard therapy with intravenous corticosteroids and plasmapheresis did not show clinical improvement.

#### Case presentation:

A 53 year old male presented to the ED with complaints of sudden onset of urine retention for one day and weight loss for three months. His past medical history was significant for hypertension and peripheral vascular disease. He denied fever, weakness of extremities, sensory deficit and respiratory symptoms. Routine labs were unremarkable except for a BUN of 41mg/dl and Creatinine of 2.5mg/dl. Patient was admitted with the preliminary diagnosis of acute renal failure secondary to obstructive uropathy. The patient developed sudden onset bilateral lower extremity weakness the morning after admission. MRI thoracic and lumbar spine with IV contrast demonstrated an increased signal intensity extending from T3-T12 region with central cord expansion suggestive of LETM. Inflammatory markers such as Rheumatoid factor, ds-DNA, lupus anticoagulant, c-ANCA, p-ANCA, Beta 2 glycoprotein were all negative. Neuromyelitis Optica (NMO) specific aquaporin 4 antibody was absent. Infectious etiology was ruled out after HIV, HTLV1&2, treponemal Ab, blood and urine cultures were negative. CSF analysis revealed leukocytosis with lymphocytic pleocytosis (96%), normal glucose level, elevated protein (85mg/dl) and elevated Immunoglobulin G level 136.6 mg/dl. CSF was sterile, cytology negative, oligoclonal bands absent. Serum ACE and calcium levels were normal. CT Chest showed subcarinal and mediastinal lymphadenopathy. Biopsy of the lymph nodes showed non caseating granuloma consistent with sarcoidosis, malignancy was ruled out. During the hospital stay, patient received a short course of high dose intravenous steroids and plasmapheresis but did not have expected response. The patient was discharged to physical rehabilitation center and was lost to follow up.

#### Discussion:

LETM is a characteristic feature of NMO, but spinal cord lesions can also occur with other autoimmune disease such as Sjogren syndrome, Sarcoidosis, SLE, Behcet disease, infectious process and neoplastic diseases. 5-15% of patients are found to have sarcoidosis with CNS involvement, of which LETM is not the initial presentation and has been sparsely reported in the literature. Although biopsy is considered the gold standard, the diagnosis is often primarily made on imaging studies like CT/MRI. The mainstay of management of neurosarcoidosis includes high dose steroids and plasmapheresis. Newer studies suggest a possible role of anti-tumor necrosis factor alpha (TNF a) for clinical management of neurosarcoidosis induced LETM.

<p><b>Author: Hafeez Hassan, MD</b>                  Additional Authors: James Tak, MD                  Syed Waqas Haider, MD                  Institution: Icahn school of medicine at Mount Sinai St Luke's Roosevelt Hospital center</p> <p><b>Title: Isolated right ventricular myocarditis: a rarely reported case</b></p> <p>INTRODUCTION:                  Myocarditis is the inflammation of the myocardium, presentation of which ranges from nonspecific systemic symptoms (fever, myalgia, palpitations, or exertional dyspnea) to fulminant cardiac failure and sudden death. Myocarditis can be seen in a variety of clinical conditions including viral illnesses and autoimmune diseases but myocarditis affecting right ventricle is rarely found. A review of literature showed only 4 previous reports, all diagnosed at autopsy, in which diagnosis was not suspected in vivo. Isolated right ventricular myocarditis was first described by Hayes in 1961 and later by Mancio in 2013.</p> <p>Case:                  23-year-old male with no past medical history who presented to emergency room with a non-exertional sharp left sided chest pain, preceded by two days of nonspecific flu-like symptoms (fatigue, cough, sinus congestion and rhinorrhea), with no sick contact, recent travel, recent hospitalization or any family history of heart disease.</p> <p>He was hemodynamically stable and physical exam was benign. Initial labs were normal with the exception of mild leukocytosis and initial troponins of 6.464 (reference range 0-0.034 ng/ml) which trended up to 197.00.</p> <p>Chest radiograph revealed no air way disease or cardiac enlargement. The EKG showed sinus tachycardia with no ST or T wave changes. Early echocardiogram revealed mild right ventricle hypokinesis. The right and left ventricular ejection fractions were 40% and 60%, respectively with no valvular abnormality. Urgent Cardiac catheterization was done which did not show any coronary artery disease. Gadolinium enhanced MRI was done which showed diffuse edema and transmural late gadolinium enhancement of the wall of the right ventricle extending into the RV outflow tract with slightly depressed RV function, consistent with isolated RV myocarditis. Troponin level started trending down next day and leukocytosis resolved.</p> <p>Complete work up of the patient failed to reveal any specific cause of myocarditis. His respiratory panel was negative for viral pathogens as cause of respiratory illness leading to isolated right sided myocarditis. He was discharged with high dose NSAIDs. His repeat MRI after 8 weeks showed resolution of the RV wall edema and improvement in ventricular ejection fraction.</p> <p>A diagnosis of isolated right ventricular myocarditis was made on the basis of clinical, echocardiographic and Cardiac MRI findings. With early diagnosis and treatment, the patient's condition and right ventricular function improved although complete work up of the patient failed to reveal any specific cause of myocarditis.</p> <p>CONCLUSIONS:                  Isolated right ventricular myocarditis should be suspected in a patient with depressed right ventricular function without left ventricular involvement on echocardiography and Cardiac MRI, elevated cardiac enzymes and no evidence of coronary artery disease. Early diagnosis and treatment should be prompted to improve right ventricular function and to prevent progression to a more serious sequel and death.</p>	<p><b>Author: Jeffrey Hoeksma, MD</b>                  Additional Authors: Berstein, Paul MD                  Institution: Rochester General Hospital</p> <p><b>Title: AN UNLIKELY PAIR: CONCURRENT MALT LYMPHOMA AND SARCOIDOSIS</b></p> <p>Lymphoma is rarely associated with sarcoidosis. We present a patient with MALT (mucosa-associated lymphoid tissue) lymphoma who presents with new onset sarcoidosis.</p> <p>67 y/o with 20 year history of untreated MALT lymphoma of parotid and lacrimal glands presented with new-onset elevation of serum calcium to 13 mg/dL and decreased eGFR. In the year prior to admission, he had lost 25 lbs, and noted decreased strength, night sweats, and worsening cough. Imaging revealed marked splenomegaly with abdominal and hilar lymphadenopathy. Bone marrow and lymph node biopsy showed granuloma without progression of lymphoma. The patient was noted to have elevated angiotensin converting enzyme and 1,25 dihydroxyvitamin D (1,25(OH)2D) consistent with sarcoidosis. Treatment with prednisone resulted in weight gain, resolution of cough, reduction in adenopathy, and normalization of serum calcium and eGFR at 3 months.</p> <p>Patients with sarcoidosis are well-known to have a greater than 5-fold relative risk of developing lymphoma. The proposed mechanism involves increased dendritic cell antigen presentation to CD4+ cells within a granuloma, leading to excessive CD4+ activation. The decreased CD8+ inhibitory cell population within the same granuloma may allow T-cell clones to escape regulation and develop lymphoma. Both lymphoma and sarcoidosis present with hypercalcemia and renal dysfunction due to elevated 1a-hydroxylase activity of macrophages, leading to elevated 1,25(OH)2D. Case studies have described sarcoidosis after a diagnosis of lymphoma, with a median time of diagnosis ~ 3 years after initial presentation. It is believed dysfunction in the immunoregulatory environment of lymphoma may lead to development of sarcoidosis. This is one of only a few reported cases of sarcoidosis after a diagnosis of lymphoma.</p>
---	--

## Resident/ Fellow Clinical Vignette

**Author: Adedapo Iluyomade, MD**

Additional Authors: Manpreet Singh Sabharwal, Vishal Tolia, Praneet Wander  
Institution: Mount Sinai St. Luke's - Roosevelt Hospital Center

### **Title: A Case of Embolic Stroke Secondary to Libman-Sacks Endocarditis**

#### INTRODUCTION

Systemic Lupus Erythematosus (SLE) is an autoimmune connective tissue disease that affects nearly every major organ system in the human body. The most characteristic cardiac manifestation of SLE is Libman-Sacks endocarditis. In 1924, a description of the atypical, sterile, verrucous vegetations classically found in this form of endocarditis was first published. All cardiac valves and endocardial surfaces can be involved, however mitral and aortic valves are most commonly affected.

#### CASE

A functionally independent and cognitively intact 72 year old female known to have Benign Hypertension, Type 2 Diabetes Mellitus on Glyburide and Sitagliptin, SLE diagnosed 25 years prior and currently treated with Azathioprine and Prednisone presented to a tertiary care hospital with a three hour history of altered mental status and disorientation. On presentation, patient was found to be afebrile, normotensive, with normal heart rate, rhythm and respirations. On physical exam, she was noticeably drowsy and confused, with a systolic murmur and non-focal neurological exam. Initial assessment yielded a working diagnosis of opioid toxicity considering collateral history. Non-contrast head CT was negative for hemorrhage or infarct. CT angiography of the head and neck revealed diffuse atherosclerotic disease of bilateral internal carotid arteries. After admission to General Medicine, 24-hour telemetry monitoring yielded no arrhythmogenicity; however the patient continued to experience a decline in mental status. An electroencephalogram (EEG) was negative for epileptiform activity, and an extensive metabolic workup yielded largely benign results. MRI of the brain was then obtained, revealing numerous foci of hyper-intensity in the bilateral occipital and cerebellar regions, right midbrain, left paramedian pons and left thalamus. Stroke team consultation was initiated, and transthoracic echocardiogram (TTE) obtained. Results of the TTE were largely benign, without sign of patent foramen ovale or overt embolic nidus. A subsequent transesophageal echocardiogram (TEE) was significant for a mobile 8x5mm vegetation on the anterior leaflet of the mitral valve. The patient was then transferred to the Neurological Intensive Care Unit for closer monitoring, and a repeat MRI was unchanged. A total of four sets of blood cultures obtained during the hospital course returned negative for microorganism growth. A diagnosis of nonbacterial thrombotic endocarditis was ultimately determined. Of note, review of systems was negative throughout duration of stay.

#### CONCLUSION

Valvular abnormalities, especially Libman-Sacks vegetations, are commonly found in patients with SLE. Moysakis et al found characteristic valvular lesions in 11% of SLE patients studied. Further review of the medical literature suggests that Libman-Sacks vegetations may affect as many as half of all persons with SLE. Early identification and management can aid clinicians in preventing its morbid, and potentially fatal, sequelae. Additionally, this case highlights the importance of obtaining a TEE in the evaluation of cerebrovascular accidents despite the presence of alternate, potentially confounding, findings.

**Author: Marjan Islam, MD**

Additional Authors: Raymond Chai MD, Azita Khorsandi MD  
Institution: Mount Sinai Beth Israel

### **Title: A Rare Case of Coalescent Mastoiditis in a Young Adult Male with Left-sided Ear Pain**

#### BACKGROUND:

Coalescent mastoiditis is a rare complication of otitis media, occurring once in every 10000 cases, with the vast majority described in the pediatric population. Due to their proximity to the posterior cranial fossa, an infection of the mastoid air cells can spread to the central nervous system. While the incidence of coalescent mastoiditis has declined dramatically in the era of intravenous antibiotics, it remains a medical emergency.

Complications include meningitis, facial nerve palsy, sinus thrombophlebitis, intracranial abscess, and subdural empyema.

#### CASE PRESENTATION:

A 45 year-old male with history of chronic ear infections presented with altered mental status in the setting of acute otitis media. He developed left-sided ear pain 2 days prior to admission, which progressed to headache, neck stiffness and photophobia. Vital signs on admission were HR = 102, BP = 165/91, T = 98.0, RR = 18, and exam was significant for disorientation, nuchal rigidity and drainage of pus from the left ear. CT of the head revealed a left-sided focal coalescent mastoiditis, with associated pneumocephalus and midline shift, secondary to a subdural empyema. It also exposed a large defect of the tegmen tympani, the bony plate separating the cranial and tympanic cavities. Neurosurgery was emergently consulted, and the patient underwent a left parietal craniectomy with evacuation of the empyema. Vancomycin, cefepime and metronidazole were started. Post-operative complications included seizures, thrombosis of the left sigmoid sinus, and an acute infarct of the left centrum semiovale. The patient was seen 2 months after discharge as an outpatient, where, with the exception of a mild residual right-arm weakness and compliant of left-ear tinnitus, he exhibited complete neurologic recovery.

#### DISCUSSION:

Coalescent mastoiditis is a rare complication of otitis media, usually preceded by breakdown of the bony tegmen and progression to a suppurative infection of the mastoid air cells. Complications such as subdural empyema and pneumocephaly can occur. Because of its high morbidity, clinicians should consider coalescent mastoiditis in an adult who presents with otitis media and altered mental status, and should be aware the clinical signs can resemble uncomplicated bacterial meningitis. Causative organisms include streptococcus pneumoniae, neisseria meningitidis and staphylococcus aureus. Initial treatment should include broad-spectrum antibiotics, as identification of a specific etiology via culture can be difficult. Surgical mastoidectomy must be considered if there is concern for infected bone, with the preservation of hearing often requiring surgical reconstruction of the acoustic anatomy or implants with ossicular prostheses. Tegmen defects, which may be congenital or due to progressive erosion from chronic infections, can predispose to coalescent mastoiditis. It is likely that in our patient, an acute otitis media exacerbated a pre-existing tegmen defect that had developed from previous recurrent ear infections. Cerebrospinal otorrhea, coalescent mastoiditis and subdural empyema then ensued.

## Resident/ Fellow Clinical Vignette

**Author: Julie Islam, MD**

Additional Authors: Mauricio Gonzalez-Arias MD1, Serena Fermin2, 1 Department of Medicine, Metropolitan Hospital Center, New York, New York New York Medical College, Valhalla, New York 2Touro College-School of Health Sciences, Bay Shore, New York  
Institution: Metropolitan Hospital- NYMC

**Title: A CASE REPORT: PSEUDO-MEIGS SYNDROME PRESENTING WITH DEEP VEIN THROMBOSIS AND A STAGHORN CALCULUS OF THE IPSILATERAL SIDE**

**Introduction:**

The common triad seen in Meigs syndrome is a benign ovarian tumor, ascites, and pleural effusion. When the mass originates from the other pelvic organs it is known as Pseudo-Meigs. We present an especially rare case in a premenopausal woman who also had deep vein thrombosis and a staghorn calculus of the ipsilateral side.

**Case Report:**

36 y/o African American female presented with left-sided pleuritic chest pain associated with shortness of breath and fatigue for 2 weeks. After being seen in a different hospital's ER, she was found to have a loculated left-sided pleural effusion. She was given analgesics and referred to an outpatient clinic. She had no past medical history aside from a left lower extremity DVT in 2010 that was treated with a short course of Coumadin. She also described heavy menses for the last six years.

On examination, she was in no acute distress and had normal chest symmetry with poor respiratory effort. There was dullness to percussion up to the mid left lung field. Initial labs revealed severe iron deficiency anemia with a hemoglobin of 6.6mg/dl and hypoalbuminemia. Chest CT showed a large left loculated pleural effusion as well as ascites. Incidentally, a left sided staghorn calculus was found. Further studies also revealed an elevated CRP at 43. An US guided thoracentesis was done, removing 400 cc of exudative fluid. Pleural fluid ADA level was negative. Further work-up was also pertinent for CA 125 level, elevated at 122 (normal <33). A pelvic ultrasound showed a 7.5 cm large, intramural myoma, along with a moderate amount of pelvic ascites and an enlarged left ovary.

**Discussion:**

Pseudo-Meigs Syndrome is an anomalous presentation often confused with Meigs. Less than 50 cases have been reported in the US. Commonly benign, it can mimic malignancy with elevation of tumor marker CA-125. Though the pathophysiology still remains up to debate, one theory elucidates a probable inflammatory effect by the release of inflammatory mediators and breakdown products causing fluid accumulation in both abdominal and chest cavities. Until now, one case has been reported to have also presented with venous thrombosis. It can be hypothesized that this increased inflammatory state may lead to hypercoagulability and clotting. It remains unclear where the staghorn calculi, a rarity in its own right, fits into this clinical picture. However, we can postulate that an alteration in anatomy caused by a fibroid mass in combination with a hypermetabolic state caused by the tumor itself, produced the right environment for struvite stone formation in the presence of a subclinical infection.

**Author: Amer Issa, MD**

Additional Authors: Mathhar Aldaoud MD, Ryan Connell MD.  
Institution: Rochester Regional Health System/ Unity Hospital

**Title: THE KISSING DISEASE THAT CAN BREAK YOUR HEART**

**Background:**

Epstein-Barr virus (EBV) myocarditis is a rare manifestation of infectious mononucleosis.

**Case Presentation:**

Previously healthy 23 year-old male presented to the emergency department with subjective fever, sore throat, fatigue and progressive shortness of breath. Four weeks earlier he had similar symptoms and was treated with penicillin for presumed streptococcal pharyngitis with no significant improvement. Vital signs were significant for temperature of 38.9 C, respiratory rate of 22 breaths/min, pulse rate of 115 beats/min, BP of 141/87 mm Hg and SpO2 of 71 % on room air. Physical examination revealed tachypnea with labored breathing, mildly enlarged tonsils, crackles in both lung bases, regular rate and rhythm, normal S1 and S2, with no murmurs or rubs, mild splenomegaly, maculopapular rash on his chest and extremities. Laboratory studies revealed elevated white blood cells count with neutrophilia, B-Type natriuretic peptide of 269 pg/ml (normal 0-100 pg/ml), Troponin of 1.40 ng/ml (normal <= 0.04 ng/ml), Alanine Aminotransferase (ALT) of 192 U/l (normal 1-44), Aspartate aminotransferase (AST) of 70 U/l normal (14-39) and Creatinine of 0.75 mg/dl normal (0.7-1.3 mg/dl). Arterial blood gases were as the following: PH of 7.43, PaO2: 60 mmHg, PaCO2: 33 mmHg, HCO3 21 mmol/l.

Electrocardiogram showed normal sinus rhythm with inverted T waves in inferolateral leads. Chest X-ray showed bilateral pleural effusion with no infiltrate. Chest CT angiogram revealed bilateral small pleural effusion, interstitial prominence in both upper lobes likely represent pulmonary vascular congestion and no evidence of pulmonary embolism. Echocardiography demonstrated moderate concentric left ventricular hypertrophy with diffuse hypokinesis and estimated ejection fraction of 20-25%. Epstein-Barr virus (EBV) antibodies titers: EBV viral capsid antigen IgM antibodies: >160 u/ml (normal <36), EBV viral capsid antigen IgG antibodies > 25 u/ml (normal <18), EBV nuclear antigen IgG antibodies > 3 u/ml (normal <18), EBV early antigen IgG antibodies 7.9 u/ml (normal <9) with a positive heterophile antibody test result. Patient was treated for hypoxia secondary to systolic congestive heart failure with oxygen supplementation and diuretics. Patient's respiratory status improved gradually and he was off oxygen by day five of admission. Post discharge he was seen by cardiologist, had an angiogram which showed no coronary artery disease, patient was thought to have EBV-induced myocarditis. Repeated Echocardiography in one week showed normalization of systolic function.

**Discussion:**

Myocarditis defined as inflammation of the myocardium. It has been recognized as a cause of congestive heart failure. Many viruses have been implicated as a cause of myocarditis. One of these viruses is EBV which can have diverse clinical manifestations. Significant cardiac involvement is rarely described. This case showed how EBV can cause myocarditis as part of the infectious mononucleosis disease spectrum.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Safeera Javed, MBBS</b> Additional Authors: Srinidhi Jayadevappa MD, TS Dharmarajan MD, MACP Institution: Montefiore Medical Center Wakefield campus</p> <p><b>Title: NORMAL MCV IN B12 DEFICIENCY IS A PROMPT FOR FURTHER EVALUATION OF ANEMIA</b></p> <p>Introduction Pernicious anemia (PA) is an autoimmune disease characterized by intrinsic factor antibodies and B12 malabsorption. Masking of the macrocytic expression of megaloblastic anemia by co-existing thalassemia, iron deficiency anemia and chronic illnesses is reported. Presented is a case of PA with severe B12 deficiency, with MCV in normal range.</p> <p>Case 28 years old female presented with acute on chronic abdominal pain and vomiting. She reported chronic burning epigastric pain, now worse, aggravated by stress and spicy foods, relieved by fasting and proton pump inhibitors. Labs confirmed severe anemia and pancytopenia with Hb 4.6; WBC 3.9; Platelets 89,000 and RDW of 45. Indirect hyperbilirubinemia, reticulocytosis, raised LDH and low haptoglobin was consistent with hemolysis and normal MCV suggested normocytic anemia; however further evaluation revealed B12 level &lt;30 pg/ml (211- 946); normal folic acid; elevated MMA and homocysteine level; and positive IF and parietal cell antibodies. Endoscopy confirmed chronic gastritis and focal intestinal metaplasia. Based on a diagnosis of PA, B12 injections were initiated. The MCV was concerning as it was not elevated as expected in B12 deficiency, prompting further evaluation of anemia. High ferritin and low transferrin iron binding levels excluded iron deficiency; anemia of chronic disease was now considered. Hb electrophoresis confirmed normal HbA2, excluding Beta thalassemia, but patient was heterozygous positive for alpha thalassemia mutation.</p> <p>Discussion: The diagnostic value of MCV is overestimated in practice, when utilized as a screening parameter to diagnose B12 deficiency. B12 deficiency may be clinically asymptomatic or present with hematological and neurological manifestations. Anemia and macro-ovalocytosis are often absent. Deficiency can co-exist with other hematological abnormalities that may abate macrocytosis; such as concurrent iron deficiency anemia, anemia of chronic disease (ACD) or a- or b- thalassemia. Our patient was anemic with a normal MCV, leading to a belief that it was anemia of chronic disease vs alpha thalassemia trait. Of interest, elevated methylmalonic acid and homocysteine levels precede a decrease in B12 levels and decline in hematocrit, emphasizing the importance of these measurements in the diagnosis rather than relying on B12 assays or MCV alone.</p> <p>Lessons Learnt: 1. MCV should not be used as a sole screening parameter to consider B12 deficiency. 2. Anemia requires a comprehensive investigation for all causes, even in those with B12 deficiency.</p> <p>Reference: Oosterhuis WP et al. Diagnostic value of the MCV in the detection of vitamin B12 deficiency; Scand J Clin Lab Invest 2000;60:9-18. Dharmarajan TS et al. Vitamin B12 deficiency: Recognizing subtle symptoms in older adults. Geriatrics. 2003; 58(3):30-38</p>	<p><b>Author: Safeera Javed, MBBS</b> Additional Authors: Mohammed Makkiya, MD; Bennal Perkins, MD Institution: Montefiore Medical Center Wakefield campus</p> <p><b>Title: MOYAMOYA SYNDROME: A RARE PRESENTATION OF VZV ENCEPHALOPATHY</b></p> <p>Introduction: We report a case of moyamoya syndrome (MMS) in a patient diagnosed with Varicella encephalopathy. Although MMS has been reported in children with VZV vasculopathy, this is the first written case reported in an adult patient with VZV encephalopathy.</p> <p>Case: 23 year old female with diabetes mellitus presented with 2 weeks of left face twitching associated with slurred speech and weakness of left upper extremity. Examination was remarkable for mild nystagmus on lateral gaze and decreased strength in left upper extremity. MRI revealed right frontal cortical lesion, suggestive of infarct. MRA head revealed proximal occlusion of both left and right MCAs. Cerebral angiogram confirmed narrowing and extensive collateral vessels; features most consistent with moyamoya spectrum vasculopathy. A complete autoimmune, hematologic and infectious work up was conducted. Antinuclear antibody (ANA), Anti-neutrophilic cytoplasmic antibody (ANCA), C3/C4 and Antiphospholipid antibody were negative. Cerebrospinal fluid (CSF) analysis was negative for VZV PCR, but positive for VZV IgG, suggestive of VZV encephalopathy. She was started on Acyclovir and was evaluated for potential revascularization.</p> <p>Discussion: Moyamoya disease either exists as an isolated idiopathic disease entity, or can be associated with infections, autoimmune and hematological disorders. Inflammation and autoimmune response associated with infections lead to a vasculopathy which characterize the pathophysiologic mechanism for angiopathy seen in MMS. MMS has been reported after many viral and bacterial infections. Over the last several decades, VZV has emerged as an important cause of intracranial vasculopathy. The clinical spectrum of VZV vasculopathy has expanded to include TIA, ischemic and hemorrhagic stroke and aneurysms. While vasculopathy is a well-recognized complication of VZV encephalopathy, VZV as a cause of MMS has rarely been reported. Our patient presented with cerebrovascular events which were attributed to MMS. This diagnosis was likely secondary to VZV encephalopathy. The diagnosis of VZV vasculopathy, however, is not always straightforward. CSF VZV PCR is not a sensitive test for diagnoses of VZV encephalopathy, in contrast to HSV encephalitis where PCR is very sensitive and specific for diagnoses. This is likely due to the chronic and protracted clinical course of VZV mediated neurologic disease. Detection of anti-VZV IgG antibody in CSF is a more sensitive indicator of VZV vasculopathy than VZV DNA.</p> <p>Conclusions: 1. Moyamoya disease is a rare angiopathy that requires a thorough investigation to determine etiology. VZV must be considered as a potential cause. 2. In VZV encephalopathy, anti-VZV IgG antibody in CSF is the test of choice to diagnose disease. Therefore, it is imperative to obtain samples for both VZV DNA and VZV IgG analysis to make this diagnosis.</p>
--	---

## Resident/ Fellow Clinical Vignette

**Author: Chandana Javvaji, MBBS**

Additional Authors: Pongwut Danchaivijitr MD. Roswell Park Cancer Institute, Buffalo, NY.  
Saby George MD. Roswell Park Cancer Institute, Buffalo, NY.

Institution: University at Buffalo Catholic Health System Internal Medicine Training Program

### **Title: SERUM PROSTATE SPECIFIC ANTIGEN LEVEL REDUCTION AFTER VARICELLA-ZOSTER VIRUS INFECTION AND TREATMENT IN PROSTATE CANCER**

Introduction: A case of metastatic Castration Resistant Prostate Cancer(mCRPC) whose Prostate Specific Antigen(PSA) levels dropped remarkably after a reactivated varicella zoster virus(VZV) infection treated with Acyclovir.

Case description: 69-year-old male was diagnosed with biopsy proven (Gleason score 5+4=9) prostate adenocarcinoma with metastasis to the bone and lymph node. His initial PSA level was 10.45 ng/ml. Androgen deprivation therapy was started with Leuprolide, which led to a PSA nadir of 0.59 ng/mL after 3 months of treatment. Follow up PSA level after 7 months of treatment increased to 1.63 ng/ml. CT and bone scans showed worsening of bony metastases. His serum testosterone was found to be at castration level. Due to his high Gleason score disease, chemotherapy with Docetaxel was started. After 7th cycle of chemotherapy, his PSA level increased to 7.60 ng/ml. Docetaxel was then discontinued, as radiographic imaging showed worsening bony metastases with multiple new lesions. 2 weeks after discontinuation of Docetaxel, patient had Varicella zoster virus infection on left T10 dermatome. He was treated with Acyclovir 800mg five times daily with marked improvement. As he has extensive bone only disease, it was decided to start Radium-223 as his next line of treatment for prostate cancer. 3 weeks after his Shingles treatment prior to starting Radium-223, his PSA level was noted to be down to 1.66 from 7.6 ng/ml. Patient then proceeded with the first dose of Radium-223. PSA level decreased further to 0.14 ng/mL 6 weeks post VZV infection. Patient completed 6 cycles of Radium-223 with slight improvement of his bone pain. PSA level during last follow up (5 months since his PSA values started to decline) was 0.32 ng/ml.

Discussion: In this case of mCRPC which progressed on Docetaxel, the PSA unexpectedly plunged after an episode of VZV infection treated with Acyclovir and has not had any increment since then. To date, this is the second description of PSA reduction in prostate cancer in relation to VZV infection. There was a prior case study reported in European association of Urology in 2009. Acyclovir is converted to acyclovir monophosphate by virus-specific thymidine kinase then further converted to acyclovir triphosphate by other cellular enzymes. Acyclovir triphosphate inhibits DNA synthesis and viral replication by competing with deoxyguanosine triphosphate for viral DNA polymerase and being incorporated into viral DNA. Thymidine kinase is also present in herpes simplex virus. There is a current phase I/II trial using intra prostatic injection of adenovirus/herpes simplex thymidine kinase gene plus oral Valacyclovir in the treatment of prostate cancer. Although the mechanism is unclear, our observation suggests a possibility of VZV infection/Acyclovir treatment/combination of both may stimulate immune response, which may lead to PSA reduction and change the course of prostate cancer with prolonged survival.

**Author: Srinidhi Jayadevappa Meera, MD**

Institution: Montefiore Medical Center , Wakefield Campus

### **Title: Disseminated Varicella Zoster Presenting as Myopericarditis in an immunocompetent male**

Srinidhi J Meera MD (ACP Associate), TS Dharmarajan MD, MACP, AGSF

Introduction

Varicella zoster (VZ) infection seldom presents with life-threatening complications in immune-competent adults. Disseminated VZ infection is uncommon. Although pneumonitis, hepatitis and encephalitis are encountered, cardiovascular manifestations are rare in healthy adults.

Case

44 year old male with no significant past history presented to the ED with dyspnea, dry cough, unintentional weight loss over 3 months, and diffuse muscle soreness, no fever and chills.

In the ED: BP 136/86, heart rate 95/minute, respiratory rate 22/minute, afebrile. EKG: T wave inversion in lead III and 1 mm ST elevation in V2, V3. Troponin was 0.89ng/ml (N:0.00-0.10) and CPK 5165 U/L. SGOT was 96U/L(N:13-50U/L) and SGPT 113U/L. He had pleuritic pain and bilateral diffuse crackles. CT Angiography thorax showed opacities and pulmonary vascular congestion. Initiated IV antibiotics for community-acquired pneumonia. Troponin trend was 0.89 -> 0.78? 0.71, Echocardiogram: normal ventricular wall motion with left ventricular ejection fraction of 65 %. Myocardial perfusion scan normal. Myocarditis associated with pericarditis was suspected. ANA, Rheumatoid Factor, Strep pneumo, legionella, amylase, lipase, HIV, hepatitis panel, CRP, ANCA, Anti Jo, anti Mi 2 antibodies were negative. He became better in days. With suspicion for autoimmune disorder, outpatient evaluation was planned but he was lost to follow up.

He returned in 2 weeks with worsening symptoms and elevated troponin, CPK of 4845 and diffuse ST elevations on EKG. He had worsening muscle weakness with inability to turn in bed. Biopsy of new onset skin eruptions confirmed multinucleated giant cells (Tzanck smear) suspicious for varicella. He developed hypoxic respiratory failure with ARDS requiring mechanical ventilation. CT thorax showed new bilateral lung nodules suggesting varicella pneumonitis. Varicella IgM and IgG were positive. EMG with muscle biopsy confirmed inflammatory myopathy. Acyclovir with steroids helped improvement and discharge.

DISCUSSION

Cardiac involvement in VZ is rare in an immunocompetent patient. Reported cardiac manifestations of varicella include pericarditis, myocarditis, endocarditis, pericardial effusion, cardiac tamponade, arrhythmias, and heart block. Features of myopericarditis or pericarditis can mimic acute coronary syndrome, with the distinction subtle. Our patient had elevated troponin and diffuse ST segment elevations, with negative stress test. Once coronary artery occlusion is excluded, management of myopericarditis involves therapy with nonsteroidal anti-inflammatory drugs, monitoring myocardial function, and addressing LV dysfunction or arrhythmias. There may be a role for antiviral agents, immunoglobulins or pacemaker.

Lessons Learnt:

• Myopericarditis is a rare manifestation of disseminated varicella zoster and occurs even in immunocompetent adults.

Reference:

Petrun B et al. Disseminated varicella-zoster virus in an immunocompetent adult. Dermatology Online Journal. 2015; 21(3):10

## Resident/ Fellow Clinical Vignette

**Author: Srinidhi Jayadevappa Meera, MD**

Additional Authors: Dr Sofia Turner ,Safeera Javed  
Institution: Montefiore Medical Center Wakefield  
Division

**Title: LETHARGY AS THE SOLE PRESENTING SYMPTOM OF ACUTE CORONARY SYNDROME – AN OFTEN UNDERDIAGNOSED ENTITY LEADING TO INCREASED IN-HOSPITAL MORTALITY**

LETHARGY AS THE SOLE PRESENTING SYMPTOM OF ACUTE CORONARY SYNDROME – AN OFTEN UNDERDIAGNOSED ENTITY LEADING TO INCREASED IN-HOSPITAL MORTALITY

Srinidhi J Meera MD (ACP Associate), Sofia Turner MD (ACP Associate), Safeera Javed MD(ACP Associate)  
Montefiore Medical Center Wakefield Campus, Dept of Internal Medicine, New York, NY

### Introduction

Acute coronary syndrome (ACS) presents without chest pain in a significant proportion of patients, particularly females, diabetics, and elderly. Most common presenting symptoms are dyspnea, diaphoresis, nausea, vomiting, and syncope. ACS patients presenting without chest pain often have adverse outcomes secondary to misdiagnoses and under-treatment. These patients may have delays in seeking medical care, less aggressive therapies, and increased in-hospital mortality.

### Case Presentation

57-year-old man with DM1 and cocaine and marijuana abuse presented with one day of sudden onset lethargy. He denied chest pain, dyspnea, diaphoresis, or palpitations. Initial blood pressure was 81/49 mmHg, heart rate was 106 beats/min, respiratory rate was 16 breaths/min and oxygen saturation was 100% on room air. Physical examination was otherwise unremarkable. Labs revealed blood glucose 472mg/dL, anion gap 23mEq/L, serum pH 7.25, positive serum acetone and urine ketones consistent with diabetic ketoacidosis (DKA). Urine drug screen was negative. DKA was treated with fluids and insulin drip with improvement in blood glucose and anion gap though lethargy persisted. A 12 lead EKG revealed new deep T wave inversions in anterior leads and troponin was elevated to peak 1.48ng/mL. Diagnosis of non-ST elevation myocardial infarction (NSTEMI) was made and he received Plavix, Aspirin, Atorvastatin and Heparin drip. Echocardiogram revealed severe mid-septal hypokinesis and mild apical hypokinesis with preserved ejection fraction. He underwent urgent coronary angiography, showing 70-80% stenosis of the left anterior descending coronary artery for which drug eluting stent was placed that resolved lethargy.

### Discussion

Atypical presentation of acute and chronic ischemic heart disease in diabetic patients is under-investigated despite extensive research into coronary artery disease. In Diabetics, Atherosclerotic plaques develop earlier, advance faster and are more diffuse. These factors contribute to a two to four-fold increased risk of cardiovascular events. Diabetic patients with silent myocardial ischemia have evidence of diffuse abnormality in metaiodobenzylguanidine (MIBG) uptake, suggesting that sympathetic denervation may be linked to abnormalities in pain perception. A multicenter retrospective study reviewing STEMI patients found that those with atypical symptoms had longer pre-hospital delays, longer ER wait times, were less likely to receive early reperfusion therapy, and had higher one month mortality as compared to those with classic chest pain.

This case demonstrates that ACS can present with atypical and nonspecific symptoms in diabetic patients and clinicians must have a high degree of suspicion to accurately diagnose and provide urgent treatment to improve outcomes.

### References

Brieger D, Eagle K, Goodman S, et al. Acute Coronary Syndromes Without Chest Pain, An Underdiagnosed and Undertreated High-Risk Group. Chest. 2004;126 (2):461-469

**Author: Kegan Jessamy, MBBS**

Additional Authors: Fidelis O. Ojevwe, Sekou Rawlins  
Institution: SUNY Upstate Medical University

**Title: Clostridium septicum, an unusual link to a lower gastrointestinal bleed**

Clostridium septicum is a highly virulent pathogen which is known to have association with colorectal malignancy, acute myeloid leukemia, myelodysplastic syndrome, immunosuppression, diabetes mellitus and cyclical neutropenia. Clostridium septicum is responsible for 1.3% of all clostridial infections with reported mortality rates close to 60% which demonstrates its high virulence. Primarily, it may present as disseminated clostridial infection in the form of septicemia, gas gangrene, and mycotic aortic aneurysms.

In our case, we present a 62 year-old female who had necrotizing fasciitis of her left thigh and groin. Computed tomography imaging of her left lower extremity was significant for air in her posterior thigh muscles which extended to her gluteus maximus and knee. She was promptly started on intravenous antibiotics and taken to the operating room where she had disarticulation of the left lower extremity to minimize spread of tissue infection. Surgical wound culture was positive for C. septicum.

Her post-operative course was unremarkable until she was noted to have frank blood in her fecal management tube on post-operative day 12. Colonoscopy done on that day revealed a fungating polypoid, sessile and ulcerated partially obstructing large mass in her cecum, consistent with well differentiated invasive adenocarcinoma. She underwent laparoscopic right hemicolectomy and had a negative workup for metastatic disease.

We would like to highlight the importance of early colorectal cancer screening in minimizing the occurrence of undetected tumors which provide an adequate environment for C. septicum leading to localized and/or remote infection. To minimize the development of these infections which occur in the context of colon cancer, physicians should be aware of the association of C. septicum and colon cancer.

## Resident/ Fellow Clinical Vignette

**Author: Omar Jilani, MD, MBA**

Additional Authors: Shaza Khan, Vijay Kanakadandi M.D., Jawad Ahmad M.D., F.R.C.P.  
Institution: Mount Sinai Beth Israel Hospital

### **Title: MACRO-AST: A NORMAL EXPLANATION FOR ABNORMAL LIVER FUNCTION TESTS**

#### Introduction

Elevated levels of AST can be seen in patients with liver, cardiac and skeletal muscle disease. An isolated elevation of AST suggests a diagnosis of macro-aspartate aminotransferase (macro-AST). We describe a case of macro-AST in a middle-aged adult that initially caused a diagnostic dilemma due to concurrent use of nutritional supplements but essentially was due to a lack of awareness of this entity.

#### Case report

A 38-year-old Caucasian man presented for a routine physical for insurance purposes. He denied any complaints. He admitted to social alcohol use but denied other toxic habits. He was only taking supplements including fish oil 1 gram twice a day, vitamin E 400 IU in the morning and 200 IU in evening, folic acid 1 mg twice a day and "Super Green", a powder containing anti-oxidant extracts from several vegetable sources. He denied drug allergies. His physical examination was unremarkable.

Laboratory evaluation was significant for an elevated serum AST of 314 IU/L with a normal ALT of 20 IU/L. The remainder of his lab-work including all other LFTs was normal, the patient was asked to stop taking dietary supplements. A repeat follow-up AST level remained elevated. He tested negative for viral hepatitis, muscle disorders, as well as metabolic and autoimmune liver diseases. Imaging was unrevealing. He was referred to the hepatology clinic for a second opinion and possible liver biopsy. Since there was no evidence of underlying liver disease and only AST elevation on repeated testing, the patient was evaluated for the presence of macro-AST. A polyethylene glycol (PEG) precipitation assay was sent. It revealed 88.2% precipitable activity with a post-PEG precipitation AST level of 16 IU/L, confirming the presence of macro-AST.

#### Discussion

Macro-AST complexes are formed from self-polymerization or binding to immunoglobulins, leading to a false positive elevation of AST. The persistently elevated AST with a negative work-up for liver and muscle disease led to the possibility of macro-AST. Macroenzymes are serum enzymes that have either self-polymerized with each other or with larger protein molecules such as immunoglobulins (IgG and IgA). Due to their decreased renal clearance compared to normal enzymes and increased molecular weight, these macro-enzymes accumulate in the blood and result in erroneously elevated AST levels on routine laboratory assays. A brief review of published cases of macro-AST shows that most individuals are asymptomatic. The diagnostic tests for macro-AST are available only in a few laboratories, further adding to the delay. The delay may cause psychological stress, increase health care costs and expose patients to unnecessary risks. Our patient was initially denied life insurance as the abnormal lab result was attributed to alcohol abuse. This case highlights the importance of awareness of this entity to avoid misdiagnosis and undue patient stress.

**Author: Ismail Jimada, Resident**

Additional Authors: Dr. Nelli Fromer.  
Oksana Symczyk.  
Kinjal Patel  
Institution: Wyckoff Heights Medical Center

### **Title: Double Hit Lymphoma in a Patient with Acute Bilateral Ophthalmoplegia.**

Diffuse large B Cell lymphoma (DLBCL) is the most common type of lymphoma and is generally highly curable. With the advent of molecular testing, a subset of DLBCL has been classified as double hit lymphoma, which is characterized by the presence of c-myc gene rearrangement t(8;14) as well as BCL2 translocation. Patients with double hit lymphoma tend to have a more aggressive clinical course and are less responsive to conventional chemotherapy. We report a case of a 57 year old female who presented with bilateral ophthalmoplegia due to compression of cranial nerves III, IV and VI by a parasellar mass. Biopsy of the pituitary mass revealed morphological features intermediate between Diffuse large B-cell lymphoma and Burkitt's lymphoma with the presence of c-myc gene rearrangement t(8;14) on FISH analysis as well as BCL2 translocation, consistent with double hit lymphoma. Cerebrospinal fluid cytology was positive for lymphoblasts. She was treated with an intensive chemotherapy regiment of R-Hyper-CVAD which consists of rituximab, cyclophosphamide, vincristine, doxorubicin and dexamethasone. She also received intrathecal methotrexate. However the patient died after completing the first cycle of chemotherapy due to infectious complications. The presenting clinical signs and symptoms in patients with these aggressive lymphoma subtypes tend to be unusual, such as can be seen in our patient with cranial nerve palsies and ophthalmoplegia. Despite much progress in the treatment of hematologic malignancies the mortality rate in patients with double hit lymphoma remains high and new treatment modalities are needed to improve outcomes.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Arunpreet Kahlon, MBBS</b> Additional Authors: Wajihuddin Syed, Amrita Dhillon, Anuj Sharma, Amit Sharma, Dana Savici Institution: SUNY Upstate Medical University</p> <p><b>Title: Development of Fanconi syndrome after addition of Ledipasvir/sofosbuvir in a patient on Tenofovir</b></p> <p>Introduction: Tenofovir is a nucleoside reverse transcriptase inhibitors (NRTI) which is a part of highly active antiretroviral therapy(HAART) regimen. It is a well described cause of Fanconi syndrome(FS). Ledipasvir/Sofosbuvir(Led/Sof) is an antiviral medication currently used for treatment of hepatitis C infection. We describe a case where FS precipitated in a patient on HAART therapy for years after he was started on Led/Sof.</p> <p>Case: 63 year old male with past medical history of HIV, Hepatitis C infection, myocardial infarction(MI), was admitted to ICU with acute coronary syndrome and electrolyte abnormalities. For his HIV, he was receiving Kaletra, efavirenz and tenofovir for last 10 years and was recently diagnosed with hepatitis C infection and for which he was started on Led/Sof therapy 3 months ago. On admission patient was found to have potassium of 2.3 meq/l, bicarbonate of 6meq/l, venous blood ph of 7.0 and pCO<sub>2</sub> of 23mmhg. Since he was taking lactulose at home that was giving him loose stools, all electrolyte abnormalities were initially attributed diarrhea. Aggressive potassium and bicarbonate replacement were started. His serum ph, potassium and bicarbonate levels failed to improve with continuous infusion of sodium bicarbonate and repeated potassium supplementation for over 36 hours. Urine studies revealed urine ph of 6 while being on sodium bicarbonate drip. Urine glucose and amino acid levels were severely elevated. Transtubular potassium gradient (TTKG) was 11.98 suggestive of renal potassium wasting. A diagnosis of Fanconi's syndrome was made. Urine did not show paraproteinemia. Unfortunately patient died from complications of electrolyte abnormalities and MI on third day of hospitalization.</p> <p>Discussion: Tenofovir is cleared renally by body and can accumulate in proximal tubules where it can inhibit replication of mitochondrial DNA(mtDNA), leading to cessation of oxidative phosphorylation in cells. This stops all active transporters in cells leading to Fanconi's syndrome. ERADICATE and ION-4 trials, which evaluated effect of Led/Sof on HCV patients co-infected with HIV, showed that Led/Sof can increase serum Tenofovir levels. Although none of the patients in these trials developed Fanconi's syndrome, a theoretical increased risk exists. To our knowledge, we are reporting one of the first cases of Fanconi's syndrome occurring in a patient on a combination of Led/Sof and Tenofovir. It is not clear whether addition of Led/Sof in this patient, who was on Tenofovir for last 10 years had a role in development of Fanconi's syndrome or not but the possibility cannot be ruled out. It should also be noted that when Tenofovir was introduced in 2001, Fanconi's syndrome was not observed in early clinical trials and was eventually discovered through isolated case reports and observational studies, highlighting the importance of such case reports.</p>	<p><b>Author: Rujuta Katkar, MD</b> Additional Authors: Neal Shah, SmitaBakhai, SharavananThevanayagam, Gregory Gudleski Institution: SUNY Buffalo, University at Buffalo</p> <p><b>Title: Quality of Care of Diabetic Patients with Severe Mental Illness (SMI) in an Academic Hospital Based Primary Care Clinic</b></p> <p>RATIONALE Higher prevalence of diabetes and cardiovascular risk factors has been reported in patients with SMI (serious mental illness i.e. schizophrenia/bipolar disorder by psychiatry) due to suboptimal care received when compared to non-SMI patients. To test this hypothesis, we did a retrospective case control study by chart review of diabetes patients with and without SMI in primary care clinic in academic setting with 40 residents.</p> <p>METHODS Chart review of 89 SMI and 248 non-SMI patients was done between January 1, 2012 and December 31, 2013. Inclusion criteria were age 18-75 years and diabetes for at least one year treated in our clinic. SMI patients were further subdivided into those residing in group homes (SMI-GH n=18) and non-group homes (SMI-NGH n=71). Group homes are staff supervised community homes where medications are given to patients under observation and care coordination is done for psychiatric and primary care appointments. NCQA (National Committee for Quality Assurance) DRP (diabetes recognition program) 2012 based outcomes (HbA1c, Blood pressure, LDL, eye and foot exam, nephropathy assessment and smoking status) and process measures such as alcohol/substance abuse, medications and health care utilization measures were measured and compared between these groups. One way ANOVA for continuous variables and Chi squared tests for categorical variables was used. A significance level of 0.05 was used for all analyses.</p> <p>RESULTS Better HbA1c&lt;7 [p&lt;.0004] and LDL&lt;100 [p&lt;.001] control was observed in SMI-GH patients vs SMI-NGH and non-SMI patients. Higher smoking [p&lt;.038] and alcohol [p&lt;.049] prevalence was observed in SMI patients irrespective of group home status. Lower prevalence of hypertension[p&lt;.001] was noted in the SMI-GH patients compared to SMI-NGH or non-SMI patients; however lower proportion of hypertensive patients were on ACE/ARB therapy in both SMI groups compared to non-SMI groups. Percentages of eye and foot exams were similar among the 3 groups. Nephropathy assessment by checking microalbuminuria was lesser in the SMI-GH patients. Use of statin, aspirin, anti-hyperglycemic and insulin medications was also similar among the 3 groups. SMI-NGH patients had significantly higher number of average missed clinic appointments as well as emergency room visits than both SMI-GH and non-SMI patients.</p> <p>CONCLUSIONS Diabetes care of SMI patients is not suboptimal to non-SMI patients in our clinic based on above outcomes. Evaluating continuity of care with Primary care provider which further enhances compliance and doctor patient relationship can be helpful to decide the reason for no difference in the care between two groups. Subdividing SMI by group home status showed even better results possibly due to increased compliance. This may help develop a cost effective community based model to reduce morbidity and mortality from diabetes complications and cardiovascular outcomes. Limitation of this study was small sample size of group home patients.</p>
---	---

## Resident/ Fellow Clinical Vignette

**Author: Daniela Kloos, MD**

Additional Authors: Lazarescu Roxana, MD

Institution: New York Presbyterian Queens

### **Title: Where the clots can develop? A case of arterial thrombosis in a patient with Crohn's disease**

Introduction: Hypercoagulability is a well-known phenomenon in inflammatory bowel disease (IBD). Venous thromboembolism is common in these patients. On the other hand, arterial involvement is rarely documented. We are presenting a case of arterial thrombosis in a patient with Crohn's disease

Case presentation: 49 year old male with HBV on viread, Crohn's disease diagnosed in 1990's on pentasa presents with 1 month history left thigh/calf claudication that was worsened to ischemic rest pain and very short distance claudication. Per patient his Crohn's has been well controlled and denied any significant diarrhea, abdominal pain, or bloody stools however did lose 10 pounds over few weeks. On admission his physical exam was significant for cool left foot and absent bilateral distal pulses; however he had bilateral femoral pulses +2, right popliteal pulse +2, Left popliteal pulse. Initial lab work was significant for leukopenia and microcytic anemia. MRA lower extremity showed occlusion of the right popliteal artery at the joint space and occlusion of the left superficial femoral artery in the proximal thigh. He was started on a heparin drip for the lower extremity arterial occlusions. Rheumatology, Hematology, GI, Cardiology consulted for further workup. No vascular intervention was recommended. Extensive rheumatologic and hypercoagulable workup was initiated and was negative for: vasculitis, SLE, RA, antithrombin III deficiency, cryoglobulinemia, antiphospholipid syndrome, factor v leiden mutation, factor II deficiency, polyarteritis nodosa, protein C deficiency, nephrotic syndrome and was heterozygous for the MTHFR C677T mutation. Patient was rule out for arterial fibrillation and the transthoracic echocardiogram didn't reveal any intracardiac thrombus, masses or vegetations. Patient was bridged to Coumadin prior to discharge.

Discussion: Hypercoagulability is a well-known phenomenon in active inflammatory bowel disease and can lead to thromboembolic events. It has been documented and demonstrated that being affected by IBD results in an approximately three fold risk of developing venous thromboembolic event compared to the general population. The most common listed factors include genetic and immune abnormalities, disequilibrium between procoagulant anticoagulant factors, as well as the endothelial damage as an IBD triggering factors has been underlined VTE in IBD is characterized by a high recurrence rate and is usually associated with the disease activity. On the other hand, arterial thrombosis in IBD is very rare. Also, in our case the patient didn't have any recent Crohn's flare and the disease was under control. This is an important aspect to remember as there is not much literature about the relationship of extra intestinal manifestation and disease activity. The thromboembolic phenomenon in IBD raises an even more challenging question for the clinician since there are no guidelines on treatment and prevention.

**Author: Arber Kodra, MD**

Additional Authors: Antonio Pires, MD

Jay Wong, MD

Steven Mendel, MD

Institution: Lenox Hill Hospital

### **Title: A Rare Case of Pneumococcal Vertebral Osteomyelitis**

Purpose: In this case, we report the rare finding of pneumococcal vertebral osteomyelitis in setting of bacterial meningitis.

Case Report: The patient is a 61 year-old male with a past medical history of hypertension who initially presented in the emergency department with fever, chest pain, and tachycardia. He was found to have a left lower lobe infiltrate on chest x-ray. The patient was treated for sepsis secondary to community-acquired pneumonia. His blood cultures grew *Streptococcus pneumoniae* (S. pneumoniae) susceptible to ceftriaxone. His repeat blood cultures were negative, and he was discharged home on a course of cefpodoxime. The patient returned one month later with fever to 103.5°F, altered mental status, nuchal rigidity and back pain. A lumbar puncture revealed a white blood cell count of 2680/L (60% neutrophils, 20% lymphocytes). The patient was started empirically on ceftriaxone, vancomycin, and ampicillin for suspected meningitis. MRI of the cervical, thoracic and lumbar spine demonstrated spondylodiscitis at the C5-C6 and T5-T6 levels and fasciitis at the L4-L5 level with a posterior epidural phlegmon and an adjacent 2mm abscess. Blood cultures were again positive for S. pneumonia, sensitive to ceftriaxone. CSF culture returned negative. Infectious Disease Service recommended 8 weeks of ceftriaxone. The patient received a peripherally inserted central catheter and was discharged on ceftriaxone with close follow up.

Discussion: Spinal and paraspinal infections from S. pneumoniae are rare. The literature suggests that infections caused by pneumococci, sickle cell anemia, bony trauma and heavy alcohol intake may be predisposing factors in pneumococcal vertebral osteomyelitis. In cases in which the diagnosis of vertebral osteomyelitis has been established with clinical/imaging findings, isolation of the causative agent and specific treatment are of utmost importance in limiting the morbidity and mortality of the disease. It is important to note that in the case of the patient's initial presentation of pneumonia with pneumococcal bacteremia, there are no Infectious Diseases Society of America (IDSA) guidelines regarding treatment. There have been no controlled trials on the optimal duration of antibiotics for the treatment of invasive pneumococcal infection in the setting of lower respiratory tract infection. It is important that more work is done to optimize the duration of therapy.

Conclusion: This case shows that vertebral osteomyelitis, albeit rare, can complicate the course of pneumococcal bacteremia without a history of invasive spinal procedures, back injury or other co-morbidities. The possibility of pneumococcal vertebral osteomyelitis should be considered in a patient with pneumococcal meningitis since this will alter the duration of antibiotic treatment. The infrequency with which it is encountered makes Pneumococcal Vertebral Osteomyelitis a formidable diagnostic challenge.

## Resident/ Fellow Clinical Vignette

**Author: Pallavi Kopparchy, MBBS**

Additional Authors: Viveksandeep C Thoguluva, MBBS; Mariam Alexander, MD; Amitha Padmanabhuni, MBBS; Rajeev Sharma, MBBS.  
Institution: SUNY upstate

### **Title: A case of periodic paralysis as the presenting feature of Grave's disease**

Introduction: Thyrotoxic periodic paralysis (TPP) is a potentially lethal complication of hyperthyroidism characterized by muscle paralysis and hypokalemia. This condition affects ~2% hyperthyroid patients of Asian descent, particularly males. Patients with TPP have an underlying predisposition for activation of Na/K-ATPase activity, either directly by thyroid hormone or indirectly via adrenergic stimulation, insulin, or exercise which causes intracellular shift of potassium. We report a case of periodic paralysis and work up revealing Grave's disease.

Case: A 30-year-old male of Korean descent presented to emergency department with profound lower extremity weakness. Stated that he felt weak after a heavy dinner and alcohol ingestion the night before and on waking up the next morning, could not move his trunk and lower extremities. On further questioning, reported experiencing similar episodes of weakness as a teenager with several occurrences since then. Attacks typically occurred early in the morning and resolved in 3-4 hours. He is unsure of family history of episodic paralysis, as he is an adopted child. His primary care physician initially worked him up for myasthenia gravis, multiple sclerosis but was unable to diagnose the etiology for his weakness. On review of systems he reported episodes of palpitations, diaphoresis, anxiety, heat intolerance, weight loss of 50 pounds over 6 months along with hyperdefecation.

Physical examination confirmed profound proximal muscle weakness in all limbs. Deep tendon reflexes were diminished. Rest of the neurological exam was normal. Thyroid was diffusely enlarged and was firm in consistency. He had tachycardia but no signs of heart failure. Laboratory studies showed a normal blood count, normal electrolyte levels except for severe hypokalemia with serum potassium level of 1.8 mmol/ L. Given his Asian descent and hypokalemia with periodic paralysis, thyroid function tests were obtained. His TSH was <0.01 IU/ml (ref range: 0.34-5.6) and free T4 was 5.10 ng/dl (ref range: 0.58-1.64). Diagnosis of TPP was made. He was initially treated with propranolol and intravenous potassium that resulted in resolution of lower extremity paralysis. RAI uptake and scan showed homogenous markedly increased uptake and thyroid stimulating immunoglobulin of 597% which was consistent with Grave's disease and was started on Propyl thiouracil. He was advised to avoid strenuous exercise, heavy carbohydrate meals and alcohol. He eventually opted for surgery and had near total thyroidectomy done.

Conclusion: Even though TPP is commonly seen in Asian men and is rarely seen in non-Asians, it is now seen more common in Western countries due to immigration. Diagnosis at initial presentation is often delayed because of the subtleness of clinical features of thyrotoxicosis and the similarities of the paralysis with other more common conditions. Early diagnosis prevents serious cardiopulmonary complications. TPP is a curable disorder that resolves when euthyroid status is achieved.

**Author: Dmitry Kozhevnikov, DO**

Additional Authors: Evan Levine, DO  
Bushra Mina, MD  
Institution: NSLIJ- Lenox Hill Hospital

### **Title: An Uncommon Cause of Hypercapnic Respiratory Failure**

Neuromuscular disorders are a well known entity in the differential diagnosis of hypercapnic respiratory failure. Myotonic dystrophy is one of the many rare neuromuscular disorders that can result in hypercapnia. Myotonic dystrophy presents in a variety of different ways, as it affects many different organ systems. The wide variety of clinical presentations and indolent symptomatology make the diagnosis extremely challenging for a practitioner. Our case is particularly unique because of the advanced age of the patient, and the slowly progressive and subtle nature of the disease, which allowed the patient to remain undiagnosed for decades.

75 year old female with history of myelofibrosis and HTN presented with generalized weakness and worsening dyspnea. Initial presentation was notable for severe somnolence after being found on the floor at home. Our patient was in hypercapnic respiratory failure with initial ABG showing pH 7.14, PCO2 147mmHg, HCO3 49mmol/L, O2 96% on room air. She was intubated and successfully extubated two days later, with subsequent improvement in PCO2, yet still requiring intermittent noninvasive mechanical ventilation. CT and MRI of the brain were negative for pathology. Chest Xray was suspicious for right basilar infiltrate, and CT chest confirmed the finding consistent with aspiration, without evidence of intrinsic lung disease. Incidentally, there was retained barium in the esophagus, suggestive of esophageal dysmotility.

Clinical suspicion was high for a neuromuscular disorder as the etiology of her hypercapnia. Once her mental status improved, she endorsed history of esophageal spasm requiring multiple balloon dilatations and early onset cataracts. Exam findings of proximal muscle weakness, temporal wasting, sunken eyes, frontal baldness, and percussion myotonia were noted. Fluoroscopic evaluation of her diaphragm was negative for evidence of paralysis, and EMG was performed. History, exam and test results were all consistent with the diagnosis of myotonic dystrophy.

The most common cause of hypercapnic respiratory failure is intrinsic lung disease. Without a diagnosis or radiographic evidence of restrictive or obstructive disease, less common etiologies of respiratory failure were considered.

Myotonic dystrophy is a multi-system disease that can present during childhood or adulthood. It is a disease process that should be considered as a differential diagnosis for a patient presenting with respiratory muscle weakness resulting in hypercapnic respiratory failure. Evidence of aspiration pneumonia and atelectasis may be found on radiographic imaging in these patients, as weakened facial and respiratory muscles predispose them to aspiration.

Due to the indolent course and large constellation of symptoms, our patient was treated for individual conditions throughout her life without a unifying diagnosis. This not only highlights a rare pathology that practitioners may have difficulty diagnosing, but also stresses the importance of a thorough history and physical examination which can lead to the discovery of a unifying diagnosis.

<p><b>Author: Tsering Lama Tamang, MBBS</b>                  Additional Authors: First Author: Tsering Gyalpo Lama Tamang                  Second Author: Marina Shuster                  Third Author: Abhinav Chandra                  Institution: Maimonides Medical Center</p> <p><b>Title: PRIMARY HEPATIC OSTEOSARCOMA: A RARE CAUSE OF PRIMARY LIVER TUMOR</b></p> <p>Introduction: Osteosarcoma is the most common bone malignancy, usually arising from skeletal lesions. Extra-osseous osteosarcomas are rare, accounting for approximately 4% of all osteosarcomas. A literature review yields very few cases of osteosarcoma primary arising from the hepatic parenchyma.</p> <p>Case report: We present a case of 54-year-old man with history hepatitis C and cirrhosis who presented with 5 days of progressive right upper quadrant pain. Magnetic resonance imaging (MRI) of abdomen and pelvis was performed that demonstrated a 4.4-cm x 4.8-cm x 4.8-cm right hepatic lobe mass with large area of necrosis and peripheral enhancement. The subsequent liver biopsy showed few cores of tumor comprised of fibroblastic malignant cells producing lace-like osteoid matrix. Rare osteoclast-like giant cells and mitotic figures were also seen. Osteosarcomatous foci in other part of body were excluded by performing extensive physical examination and radiologic imaging and biopsy of knee for suspicious lesions, which was negative for malignancy. Hence, a primary osteosarcoma was diagnosed. The patient underwent portal vein embolization in preparation for surgical resection of the right liver lobe. He was admitted 6 weeks after embolization with dyspnea and abdominal distension and expired due to abdominal hematoma and pulmonary embolism.</p> <p>Conclusion: Based on the rarity, lack of consensus in treatment and dismal prognosis, extra-osseous osteosarcoma should be considered a separate entity from osseous osteosarcoma. To date it appears surgical resection with adjuvant chemo-radiation is the best treatment choice, although due to the rarity of the disease, no evidence based treatment protocols exist. More data and research is needed in this rare and understudied malignancy.</p>	<p><b>Author: Olaoluwatomi Lamikanra, MBBS</b>                  Additional Authors: Vimbai Nyemba MD, Edward Chapnick MD                  Institution: Maimonides Medical Center  <b>Title: Raoultella Planticola associated with pyelonephritis and nephrolithiasis</b></p> <p>Introduction: Raoultella planticola is a Gram negative, aerobic, non-motile encapsulated bacillus. It is an environmental bacterium primarily found in water, soil and fish but there have been 16 reported cases of human infections. We describe the first reported case of Raoultella planticola bacteremia with septic shock secondary to a urinary tract infection.</p> <p>Case Presentation: A 76-year-old male presented with a 5-day history of hematuria, dysuria, chills and fever (103.3F). The fever was persistent and accompanied by chills and rigors. The patient also had macroscopic hematuria. Past medical history included recurrent nephrolithiasis and surgery for stone retrieval 40 years before. Medications on admission included ciprofloxacin, tamsulosin and nebulolol.</p> <p>On admission the patient was alert and oriented with vital signs: temperature 103.3F, heart rate 130/min and blood pressure 80/56mmHg. Bright red blood was visible at the urethral orifice. He was admitted to the ICU and treated with IV fluids, norepinephrine, ceftriaxone and gentamicin. The initial leucocyte count was 4,400/mm<sup>3</sup> with 88.5% neutrophils, which increased to 22,000/mm<sup>3</sup> with 17% band forms on the 2nd day of admission. Urine microscopy revealed packed red blood cells, moderate bacteria and epithelial cells. Computerized tomographic scans of the abdomen done on the 2nd hospital day showed urinary obstruction and bilateral hydronephrosis and the non-distended bladder had a stone measuring 1.51cm.</p> <p>Urine cultures showed gram negative rods which were identified as Raoultella planticola. Blood cultures drawn on admission showed gram negative rods on day 2, identified as Raoultella planticola on day 6. Antibiotic treatment was changed to meropenem and amikacin on day 5 because of persistent fever.</p> <p>The patient received 2 weeks of meropenem and amikacin. He had a transurethral resection of the prostate (TURP) with abscess drainage two weeks after his last hospital stay and was discharged home on post-operative day 3.</p> <p>Discussion: Raoultella planticola was initially described as Klebsiella planticola in 1981 yet the first case of a human infection with this organism was reported in 1984.<sup>1</sup> Infections with Raoultella planticola have been reported in patients with &gt;50% being community acquired.<sup>2</sup> Our patient had a community acquired infection with a risk factor for multidrug resistance being outpatient antibiotic. Raoultella planticola should be recognized as a cause of UTI in patients presenting from the community with significant comorbidity.</p> <p>Conclusion: We report a case of UTI and bacteremia secondary to Raoultella planticola.</p> <ol style="list-style-type: none"> <li>1. Freney J, Fleurette J, Gruer LD et al. Klebsiella trevisanii colonization and septicemia. Lancet 1984; 8382: 909.</li> <li>2. Puerta-Fernandez S, Miralles-Linares F, Sanchez-Simonet M et al. Raoultella planticola bacteremia secondary to gastroenteritis. Clinical Microbiology &amp; Infection May 2013;19(5):E236</li> </ol>
--	---

## Resident/ Fellow Clinical Vignette

<p><b>Author: Kevin Gerard Lazo, D.O.</b> Additional Authors: Samit K. Datta, MD, Robert Graham, MD Institution: Department of Medicine, Lenox Hill Hospital</p> <p><b>Title: Three strikes and he's not out! A Third Primary Malignancy Mimicking Acute Appendicitis</b></p> <p>Appendiceal mucinous tumors comprise a spectrum of tumors of the appendix - all of which have the potential to metastasize and induce appendiceal rupture. They commonly present by mimicking symptoms of acute appendicitis. To our knowledge, their incidence in conjunction with other malignancies is unknown. We present the case of a patient with three primary malignancies, the third being a low grade mucinous neoplasm, that presented as acute appendicitis. Our patient is a 60 year old male with metastatic gastric cancer and hepatocellular carcinoma (HCC) due to hepatitis B virus. He presented with a two-day history of acute right flank pain radiating to the groin with associated chills. The patient had recently been treated for a nephrolithiasis-related urinary tract infection. He returned to the clinic one week later with worsening right lower quadrant pain (RLQ), fevers, and chills. A CT Scan showed hydronephrosis and a dilated appendix. The patient was subsequently admitted and treated for appendicitis with 3 days of IV antibiotics only - alleviating his symptoms. He was later discharged with an additional 7 days of antibiotics. The patient's RLQ pain did not resolve, and he was readmitted with a repeat CT showing a dilated 1.1 cm minimally inflamed appendix with appendicolith. The patient underwent a laparoscopic appendectomy for acute appendicitis. Histopathology showed a low-grade appendiceal mucinous neoplasm. The patient had no evidence of disease during his outpatient follow-up appointment. This case is unique in that it is the presentation of a patient with three primary malignancies, one of which was an appendiceal tumor. These neoplasms comprise 0.2%-0.4% of all appendectomy specimens. One study has shown that the incidence of two or more primary malignancies is 6.3%. Another study has shown the incidence of three or more primary malignancies to be 0.1%. There are no other case reports to our knowledge of an appendiceal tumor as a second or third primary tumor. In addition, this case serves as a reminder that appendiceal tumors, while uncommon, should be on the differential for right lower quadrant pain.</p>	<p><b>Author: Evan Levine, DO</b> Additional Authors: Jeremy Jacobson MD, Daniel Lieberman MD, Nicholas Skipitaris MD Institution: Lenox Hill Hospital</p> <p><b>Title: Dabigatran, an Inciting Agent in Hemorrhagic Pericardial Effusion</b></p> <p>Determining the etiology of a pericardial effusion is often a diagnostic challenge. In this case, a novel anticoagulant was found to be the likely culprit for a patient with hemorrhagic pericardial effusion. The increasing use of novel anticoagulants makes this case critically relevant in evaluating such patients. An 81 year old male presented with profound weakness, lethargy, and anorexia for 3 days. He denied fever, chest pain, palpitations or shortness of breath. There was no history of recent trauma, MI, or infectious illness including tuberculosis. There was also no history of recent surgical or interventional procedure involving the heart. His past medical history was significant for paroxysmal atrial fibrillation and untreated small cell lymphoma. Home medications were significant only for chronic dabigatran therapy. On arrival to the emergency department, the patient was found to be in moderate respiratory distress. Physical exam revealed a fast and irregular heart rate, jugular venous distention and mild pitting edema of his lower extremities bilaterally. Serum chemistry, CBC, coagulation studies, and cardiac profile were within normal limits. His Pro-BNP was elevated. EKG showed atrial fibrillation with rapid ventricular response. CXR showed cardiomegaly. The patient was admitted to the telemetry unit where a bedside echocardiogram showed a large pericardial effusion without tamponade physiology. Subsequent pericardiocentesis yielded 600 ml of dark bloody fluid from the pericardial sac. The fluid analysis showed high RBCs and increased LDH, both consistent with a hemorrhagic effusion. Fluid and peripheral blood was sent for gram stain, culture, AFB, cytology and flow cytometry. All tests for rheumatologic, infectious, and malignant causes for pericardial effusion were negative. CT of the chest, abdomen, and pelvis failed to reveal any evidence of malignancy. Repeat echocardiogram showed resolution of the pericardial effusion post procedure. The patient was discharged on rate control medications and following a Enoxaparin bridge, he was maintained on coumadin for anticoagulation. Currently the patient is being followed as an outpatient with no evidence of reaccumulation of the effusion. This case introduces Dabigatran as the cause of a lone pericardial effusion. Prior studies illustrate the importance of considering alternative causes of pericardial effusion in the setting of a malignancy. With all laboratory and radiographic evidence leading away from malignancy as the origin of this effusion, we were prompted to consider other causes. An extensive literature search led us to consider dabigatran as the culprit in our patient. A Naranjo likelihood score &gt;5 further supported our clinical inclination. This case highlights the potential for serious complications with Dabigatran therapy. Additionally, as the indications and use of novel oral anticoagulants continues to rise, the emphasis to identify adverse drug effects associated with their administration becomes essential.</p>
---	--

## Resident/ Fellow Clinical Vignette

**Author: Jason Ling, MD**

Additional Authors: Sandeep Mallipattu, MD

Institution: Stony Brook University Hospital

### **Title: A UNIQUE CASE OF NEPHROTIC SYNDROME AND GLYCOSURIA**

Introduction:

We report a case of a 25 year old Caucasian male with nephrotic syndrome secondary to Focal Segmental Glomerulosclerosis (FSGS) who presented with anasarca, decreased glomerular filtration rate, and nephrotic range proteinuria consistent with FSGS. An intriguing aspect of this case is that the patient was noted to have persistent glycosuria without diabetes mellitus.

Case Description:

A 25-year-old Caucasian man with history of nephrotic syndrome secondary to FSGS presented to the nephrology clinic for anasarca. At the age of 15, the patient was evaluated for nephrotic syndrome and underwent a kidney biopsy which showed FSGS, classic type. He was treated with Cellcept for approximately 3 years until the patient lost his medical insurance and was lost to follow up for approximately 6 years. The patient returned with progressive anasarca and weight gain.

Physical examination showed blood pressure of 156/84 mmHg and body mass index of 30.2 kg/m<sup>2</sup>. Laboratory investigation was significant for the following values: serum creatinine, 4.9 mg/dL (433 &#181;mol/L); serum albumin, 1.5 g/dL (15 g/L); serum phosphate 5.7 mg/dL; serum bicarbonate 24 mmol/L; total cholesterol, 368 mg/dL; glucose 106 mg/dL and hemoglobin A1c (HgbA1c), 5.0%. Urinalysis showed 500 mg/dL (28 mmol/L) of glucose and 300 mg/dL (17 mmol/L) of protein.

A repeat kidney biopsy was performed and the findings were consistent with FSGS.

Methods: Because the patient had glycosuria despite an absence of hyperglycemia, we wanted to investigate if the patient had decreased expression of the renal sodium-glucose transporter SGLT2, which is responsible for glucose reuptake in the kidneys. We used a polyclonal anti-SGLT2 rabbit antibody and immunofluorescence to measure the SGLT2 intensity level with ImageJ, NIH imaging software. We compared the intensity level to two control patients, one without glomerular disease and one with FSGS without glycosuria. We also initiated a workup for Fanconi syndrome to determine if the cause was secondary to proximal tubular dysfunction.

Results: The patient's percentage of the area that stained for SGLT2 was 8.78% and 2.64%, which was significantly lower compared to our control group of 32.03% (patient without glomerular disease) and 23.33% (FSGS without glycosuria). The intensity of the SGLT2 expression was reduced as well. His urine was significantly positive for amino acids.

Discussion:

The patient had lower SGLT2 expression which is suggestive of a lower maximum transport for glucose. The diagnosis of Fanconi syndrome in this patient was unclear because although the patient has aminoaciduria, the patient did not have any other signs like acidosis or hypophosphatemia. Upon review of the literature, there was no link between Fanconi syndrome and nephrotic syndrome, however, there were several case reports that demonstrated patients with nephrotic syndrome who had glycosuria and aminoaciduria.

**Author: Yonathan Litwok, MD**

Additional Authors:

Institution: North Shore Long Island Jewish Medical Center

### **Title: A College Student's Academic Decline: Early Signs of Limbic Encephalitis**

Introduction: Anti-NMDA receptor encephalitis is a rare diagnosis, which can present with a wide variety of symptoms. This etiology should be considered in the differential of a patient presenting with mental status changes. A failure to properly identify it can have lethal consequences for the patient.

Case Scenario: A 19 year old female with no medical history, pursuing an engineering degree at a competitive university, developed behavioral changes in the middle of her college semester. Two weeks prior to her arrival at our hospital she was unable to sleep more than 1-2 hours per night and began eating only one meal per day. These changes forced her to drop out of one of her classes. Ten days prior to admission she developed a migraine headache and nausea. That same day, the patient had an episode of confusion in which she was unable to recognize a close friend. Six days prior to admission the patient's mother heard her fall and found her face down on the floor. The patient was unresponsive with her eyes closed for 2-3 minutes, with no incontinence or abnormal movements. The patient was presumed to have had a seizure although workup that day at an outside hospital was negative. The patient was discharged in a confused state and her family brought her to our institution.

Workup at our institution for her encephalopathy was negative for an infectious process or toxic etiology. As the patient's confusion and delirium worsened, paraneoplastic and autoimmune etiologies were considered. CT abdomen, confirmed with vaginal ultrasound revealed a 19.2 cm complex cystic and solid mass arising from the left adnexa. The patient's serum and CSF were positive for NMDA receptor antibodies. She underwent left salpingo-oophorectomy on hospital day 7. Pathology demonstrated high grade immature teratoma. Immunotherapy was initiated on post-op day 3. The patient was treated with corticosteroids, intravenous immunoglobulin (IVIG) and plasmapheresis.

The patient's mental status remained unchanged until she was three weeks post-op. At that point the patient's speech increased and her ability to concentrate improved. The patient's insomnia began to resolve as well. Eight weeks after discharge the patient had returned to her normal mental status.

Discussion: This patient had a very rare case of limbic encephalitis secondary to an ovarian teratoma. Symptoms often include psychiatric manifestations and behavioral changes, but can also include somatic complaints and seizures. Limbic encephalitis is caused by an auto-antibody (in this case NMDA) which leads to inflammation of the limbic system. Prompt intervention, including removal of the tumor, IVIG, plasmapheresis, and corticosteroids can lead to full recovery, as seen in our patient.

## Resident/ Fellow Clinical Vignette

**Author: Minghao Liu, MD**

Additional Authors: Lydia Kruge, Rita Louard  
Institution: Montefiore Medical Center, Moses and Weiler Division

**Title: The Perfect Storm**

A 68-year-old woman with history of hyperthyroidism developed tachycardia, fever and altered mental status after percutaneous transluminal cardiac angioplasty (PTCA). She initially presented to the emergency room with abdominal pain, shortness of breath, shoulder pain and vomiting for one day, with no fever. She appeared cachectic and had proptosis, an enlarged and smooth thyroid, and warm, moist skin, with no edema. EKG revealed ST elevations in II, III, and aVF. She underwent PTCA with placement of 3 drug-eluting stents to the right coronary artery. Six hours later, she was delirious and tachypneic, requiring intubation. Family members revealed her hyperthyroidism was intermittently treated prior to admission. Heart rate was 142 beats per minute, temperature was 102.5 degrees Fahrenheit, and capillary blood glucose was 179 mg/dL. There was no acute pathology on Computed Tomography of the head without contrast. White blood cell count was 15.6 k/microliter and cultures showed no growth. TSH was <.005 microU/mL and free T4 was 7.08 ng/dL (normal 0.8-1.7 ng/dL). Thyroid stimulating immunoglobulin index was 454% (normal <140% of baseline). She was diagnosed with thyroid storm and treated with hydrocortisone, metoprolol and methimazole followed by potassium iodide. Agitation resolved the next day and she was extubated. Two days after treatment, she briefly developed atrial fibrillation with rapid ventricular response which ceased with uptitration of metoprolol. She regained her baseline mental status over the next two weeks.

Thyroid storm is a rare and life-threatening condition characterized by symptoms of severe thyrotoxicosis including tachycardia, hyperpyrexia and altered mentation. In addition to high levels of T4 and T3 and low levels of TSH, laboratory abnormalities can also include mild hypercalcemia, hyperglycemia, elevated liver tests, and high or low white blood cell count. In patients with a history of hyperthyroidism, thyroid storm may be precipitated by trauma, infection, surgery, or iodine load - such as the iodinated contrast media used during cardiac catheterization in our patient.

Autonomy of thyroid function from the normal feedback loop by which high levels of T3 and T4 inhibit TSH is key to the mechanism of iodine-induced thyrotoxicosis. In Graves disease, activating antibodies against TSH receptors on the thyroid gland make the gland independent of stimulation from native TSH. Autonomous thyroid tissue may also arise from chronic stimulation with TSH (often in iodine-deficient settings) and subsequent mutations. When the hyperfunctioning thyroid tissue encounters an excess of iodine, the substrate for thyroid hormone formation, the ensuing increase in production of thyroid hormone can lead to a hypermetabolic state with mortality rates of 10%-30%.

Patients with thyroid storm require ICU monitoring. Treatment includes thionamides to inhibit new thyroid hormone synthesis followed by iodine to block thyroid hormone release, glucocorticoids, beta-blockers and bile acid sequestrants.

**Author: Alexandra Llyod-Smith, MD**

Additional Authors: Shashank Agarwal, Alexandra Llyod-Smith, Jaydeep Bhatt  
Institution: New York University

**Title: A case of Guillain-Barre syndrome following concussion**

Background: Guillain-Barre syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy which follows an identifiable precipitating event in two thirds of cases. GBS has been described following viral infections, immunization, various types of trauma including intracranial and general surgical procedures, orthopedic operations, and spinal anesthesia and acute head trauma. Our case is unique as our patient had GBS developing after sustaining mild traumatic brain injury resulting in concussion.

Case Report: An 18-year-old male sustained a concussion after being hit in the head with a ball (had helmet on) while playing baseball. He developed vestibular symptoms, nausea, vomiting and headache and presented to a neurologist who prescribed nortriptyline. CT scan was negative for intracranial pathology. 4 months after the head trauma, he developed one week of bilateral foot numbness, gait difficulty and imbalance with flapping feet. Neurological exam revealed distal vibratory and proprioceptive sensory loss decreased tone and absent leg reflexes. Serology for toxic metabolic derangement and anti-ganglioside antibodies were negative. Infectious disease serology including Campylobacter jejuni, HIV, CMV, EBV, VZV, hepatitis B, C and syphilis were negative as were stool cultures for Campylobacter jejuni. Cerebrospinal fluid analysis revealed albuminocytological dissociation (0.65 g/L). Electrodiagnostic testing was abnormal with evidence of an acute to sub-acute severe demyelinating sensorimotor large fiber polyneuropathy.

Discussion: This case demonstrates a possible association between concussion and the development of Guillain-Barre syndrome. Our patient showed the typical clinical features of GBS with the development of progressive limb weakness, areflexia and distal sensory loss. The diagnosis was confirmed by the characteristic albuminocytological dissociation and electrophysiological findings of an acute to sub-acute severe demyelinating sensorimotor large fiber polyneuropathy. Although the occurrence of GBS following acute head trauma including subdural hematomas, facial fractures, brachial plexus injuries has been previously reported, concussion by itself as a precipitating cause of GBS has not been described. Possible pathogenesis includes myelin basic protein (MBP), known to be immunogenic and can induce demyelinating disease in a variety of animal species. Levels of myelin basic protein in serum and cerebrospinal fluid of patients who have suffered head injury or undergone neurosurgery is elevated. It is possible myelin basic protein or some other neuronogial protein released into the circulation following injury induced the production of anti-myelin antibodies, causing a demyelinating neuropathy.

Conclusion: This case demonstrates a possible relationship between concussion and GBS. To our knowledge there have been no reported cases of GBS presenting months after sustaining a mild head injury that resulted in a concussion. Prior reports of GBS occurring after acute head injuries including subdural hematomas, facial fractures and brachial plexus injuries and possible pathophysiological mechanisms further support this association.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Sara Rose MacLeod, DO</b> Additional Authors: Nabeela Khan MD, Haider Khadim MD, Henri T Woodman MD Institution: Catholic Health System-Sisters Hospital</p> <p><b>Title: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS: A PECULIAR COURSE</b></p> <p>Introduction: Hemophagocytic Lymphohistiocytosis is a rare condition in which lymphocytes and macrophages phagocytose red and white blood cells as well as platelets. With a highly stimulated, ineffective immune response to antigens, life-threatening cytokine storms and inflammatory responses result. It is typically considered to be a rapidly progressive disease and is often fatal without treatment.</p> <p>Case Presentation: A 69 y/o Caucasian male with a past medical history of CAD s/p CABG, repaired abdominal aortic aneurysm, hypertension, laryngeal cancer s/p radiation, and chronic mild thrombocytopenia presented to the emergency department ten months ago. He was complaining of weight loss with weakness and was demonstrating pancytopenia; WBC 1.3, Hgb 11.5 and platelets 24. No infection was identified. A bone marrow biopsy was done which showed hypercellular marrow, increased macrophages, hemophagocytosis, increased interstitial T/NK cell activity and no evidence of leukemia/lymphoma. Hemophagocytic Lymphohistiocytosis was considered at this time. Interestingly, he had stable clinical course with supportive care and was discharged home with outpatient hematological follow up. A repeat CBC two months later showed significant improvement in blood counts; WBC 3.5, Hgb 14.1 and platelets 121. The clinical picture no longer supported the diagnosis of HLH suggested by bone marrow morphology. Ten months later, he now presents with hypotension, fever, mental status changes and no identifiable infection. Laboratory test show ferritin level &gt;15,000, elevated LFTs, hypertriglyceridemia, splenomegaly and profound pancytopenia; WBC 0.5, Hgb 10.2 and platelets 16. A repeat bone marrow biopsy exhibits increased histiocytes with hemophagocytosis similar to the findings on prior bone marrow morphology. He was diagnosed with HLH meeting five of the eight diagnostic criteria. At that point he was started on HLH-94 protocol with dexamethasone and etoposide.</p> <p>Discussion: The aggressive nature of untreated Hemophagocytic Lymphohistiocytosis necessitates timely diagnosis and treatment. A diagnosis can be made by identifying molecular defects or by displaying at least five diagnostic parameters. Diagnostic parameters include fever, splenomegaly, cytopenias, hypertriglyceridemia, hemophagocytes in peripheral blood or bone marrow, elevated ferritin, elevated soluble CD25 and low/absent NK-cell activity. The disease course of this particular case is unusual, specifically the long remission followed by relapse. This disease is known to be rapidly progressive when untreated. It is worthwhile to note that some patients may have a remitting and relapsing course and earlier identification and treatment are very important.</p>	<p><b>Author: kaushik Mandal, Resident Physician</b> Additional Authors: sidhertha podder, Umair Jandga, Shetra Sivamurthy Institution: jamaica hospital medical center</p> <p><b>Title: Rare presentation of orbital Plasmablastic lymphoma with oral cavity involvement in a HIV negative patient.</b></p> <p>Introduction: Plasmablastic lymphoma(PBL) is classified by WHO as HIV associated lymphoma of oral cavity. However, it has been reported in non HIV patients with extra-oral site involvement. PBL therefore represents a new distinct subtype of diffuse large B- cell lymphoma (DLBCL).</p> <p>Case description: A 71-year-old Nigerian male presented with complaint of swelling and proptosis of left eye for a period of 6 months. Initially he had diminished vision in the left eye and it started to protrude gradually with complete loss of vision. Later developed throat discomfort with palatal swelling. PMH was significant only for hypertension. Patient was non-smoker, non-alcoholic, and no family history of malignancy. On examination, vitals unremarkable. Severe proptosis of the left eye, a fleshy visible mass protruded anteriorly, superiorly, inferiorly and laterally was seen. Decreased visual acuity with diminished ROM of the extraocular muscle, bilaterally reactive pupil with afferent pupillary defect in left present. Three small cervical lymph nodes and diminished oropharyngeal airway was noticed. Chest, Cardiovascular, abdominal and rest of the neurological examinations were normal. Initial labs showed microcytic anemia: Hb/Hct: 9.9/47, elevated BUN/Cr: 57/2. CT images revealed left orbital, palatal, sublingual, floor of oral cavity masses. Further, MRI demonstrated a lobular mass (5.9 cm X 2.6 cm X 2.9 cm) within the superolateral quadrant of the left orbit which displaced the globe, the optic nerve, superior rectus and lateral rectus muscles, no intracranial extension. Also enlarged nasopharyngeal soft tissue and an abnormal lobulated palatal mass measuring 4 cm X 4.7cm X 2.5 cm were noticed.</p> <p>Relative decreased T-2 signal in MRI suggested diagnosis towards lymphoma. Subsequently, biopsy from the soft palate lesion revealed uniform population of large cells with a moderate amount of cytoplasm and a nucleus. Immunohistochemical study showed atypical cell phenotype with markers positive for CD45, CD79a, CD138, and MUM-1. Some of the large cells showed weak cytoplasmic kappa positivity. A small subset was weakly positive for CD30 and CD20, which is overall characteristic of Plasmablastic lymphoma. CSF analysis was negative for malignant cells, so were EBV, HIV serology. Chemotherapy was started with R-CODOX regimen. Clinical remission was achieved in 1 week. 2 weeks later, second cycle chemotherapy with IVAC regimen was started, repeat CT showed significant diminished tumor size. Left eye vision started to improve and returned to normal during the subsequent during follow up.</p> <p>Discussion: PBL is often described as aggressive NHL with median survival of 6 months. Our case did not fit any of the above subtype described by Colomo et al and showed marked improvement with chemotherapy. This reinforces that PBL needs further revision for identification of prognostic criteria. Morphological, immunophenotypic, and clinical feature should be taken into consideration to diagnose PBL and treatment should be individualized.</p>
---	---

## Resident/ Fellow Clinical Vignette

<p><b>Author: Zoonra Mashriqi, MD</b> Additional Authors: Ali Mahjoub, MD, Harvey Dosik MD Institution: New York Methodist Hospital</p> <p><b>Title: PERICARDIAL EFFUSION AND CARDIAC TAMPONADE IN A PATIENT WITH HYPEREOSINOPHILIC SYNDROME</b></p> <p>Cardiac tamponade is a rare initial presentation in Hypereosinophilic Syndrome. Usually, there is endomyocardial involvement as opposed to pericardial involvement and it occurs later in the course of the disease. A 50 year old male with no significant past medical history came into the emergency department with chest pain. Laboratory results showed peripheral leukocytosis with an absolute eosinophil count of 35,000 and elevated cardiac enzymes. An EKG showed normal sinus rhythm with low voltage. The patient was subsequently admitted to Telemetry. He underwent a bone marrow aspiration with biopsy which revealed a FIP1L1-PDGFR fusion gene consistent with Hypereosinophilic syndrome. His echocardiogram revealed an ejection fraction of 52% with a moderate, 18 mm pericardial effusion causing moderate right ventricular and right atrial collapse, consistent with moderate tamponade physiology. The patient remained hemodynamically stable and was scheduled for an elective pericardial window with pericardial biopsy. A total of 1,840 milliliters were drained from the pericardial space during the procedure. The pericardial fluid showed 55% eosinophils and pericardial biopsy revealed mesothelial lined fibroadipose with focal infiltration by eosinophils. The patient was started on Imatinib 100 mg and Prednisone 100 mg for treatment of Hypereosinophilic Syndrome. He was discharged with outpatient follow up. The patient is currently in remission.</p> <p>Idiopathic Hypereosinophilic Syndrome (IHES) is defined as a multi-organ disease caused by absolute eosinophilia greater than 1,500. Approximately 40-70% of these cases have cardiac involvement, which is the major cause of morbidity and mortality. However, the frequency of pericardial involvement is estimated to be less than 10%. In a prospective study, 3 out of 55 patients had pericardial effusion with IHES. None of these patients had evidence of cardiac tamponade. In another study, 9 out of 51 patients with IHES developed pericardial effusion. A review of the medical literature utilizing PubMed showed that 9 articles and 23 reported cases were published with pericardial effusion in IHES. Eight out of the 23 case reports had pericardial effusion as the initial manifestation. Only one patient out of the 23 case reports had cardiac tamponade. Since cardiac manifestations vary widely with Hypereosinophilic Syndrome, being aware of pericardial involvement is vital for prompt diagnosis and aggressive initiation of treatment in a potentially fatal disease.</p>	<p><b>Author: Deming Mi, MD</b> Additional Authors: Amina Mookshah, MD; Ali Ahoui, MD; Samuel Bavli, MD; Polina Khanina, MD Institution: St John's Episcopal Hospital</p> <p><b>Title: THYROID STORM PRESENTING WITH ALTERED MENTAL STATUS</b></p> <p>Introduction Thyroid storm is a potentially life-threatening condition that is usually triggered by major stress events such as infection, trauma, or surgery. Patients usually present with manifestations of thyrotoxicosis, such as fever, tachycardia, hypertension, and arrhythmia. In this report, we describe the case of a patient in thyroid storm who presented with altered mental status.</p> <p>Case A 48-year-old African-American female who was diagnosed with hyperthyroidism about 8 years ago and who was noncompliant with methimazole therapy was found to have altered mental status and subsequently was found unresponsive at home by family member. In the ED, the patient was lethargic and confused. She was found to have fever (105F), pulse 160, and cellulitis in the right lower leg. Blood pressure was somewhat low, with systolic blood pressure in 90s-100s and diastolic blood pressure in 40s-50s. Lab showed elevated WBCs with increased band neutrophils, and coagulopathy. Initially, meningitis was suspected, but that was subsequently ruled out by normal lumbar puncture and brain CT scan. CT of lower extremities showed moderate subcutaneous fluid and swelling in the right lower extremity without deeper structure involvement. The patient was treated with antibiotics for right lower extremity cellulitis. Approximately 18 hours after admission, she was noted to have persistent tachycardia with BP 90/60. ECG showed atrial fibrillation, which was resistant to diltiazem and digoxin IV push. The patient had no prior history of atrial fibrillation. She was given electrical cardioversion twice with 100 and then 200 Joules, without success. She returned to normal sinus rhythm spontaneously 2-3 hours later. Subsequent labs showed TSH &lt; 0.02 mIU/mL, free T4 of 5.74 ng/dL (0.78-2.19) and free T3 of 7.75 pg/mL (1.8-4.6). Serum cortisol was 15.2 mcg/dL. She was treated for thyroid storm with propylthiouracil 400 mg Q8h, potassium iodide, thiamine, stress dose of hydrocortisone, and propranolol. Her mental status improved rapidly, tachycardia resolved, and no new episode of atrial fibrillation was noted. Propylthiouracil and propranolol were tapered as the thyroid function tests improved and heart rate stabilized. Propylthiouracil was discontinued, and the patient was maintained on methimazole.</p> <p>Discussion Thyroid storm typically presents with fever, tachycardia, and often cardiac arrhythmia. But, as this case illustrates, it can also present with altered mental status when complicated by other comorbidities. Thiamine is a cofactor for several enzymes in important metabolic pathways including alpha-ketoglutarate dehydrogenase in TCA cycle. The depletion of thiamine during the hypermetabolic state of thyroid storm may contribute to the development of altered mental status.</p>
---	--

## Resident/ Fellow Clinical Vignette

**Author: Raiko Munankarmi, M.D.**

Additional Authors: Mostafa Alfshawy, Pallavi Pothuri, Vincent Rizzo.

Institution: Icahn School of Medicine at Mount Sinai / Queens Hospital Center

**Title: A Rare Cause of Orthopnea: Tracheomalacia**

Introduction:

Orthopnea is shortness of breath that occurs when lying flat. Orthopnea is often a symptom of left ventricular failure and/or pulmonary edema and pulmonary diseases like asthma, chronic bronchitis, sleep apnea or panic disorder. We present a unique case presenting with orthopnea following radiotherapy for lung cancer that proved to be due to tracheomalacia.

Case Report:

A 68 year old male with a history of lung cancer status post left lung resection in 2001 presented with sudden onset shortness of breath for 3 days. Lying flat or speaking made him feel short of breath. He recently completed 10 sessions of radiotherapy for relapse of right upper lobe lung cancer one month prior to presentation. Initially, CT scan of the chest was unable to be obtained as the patient could not lie flat. He was treated with bronchodilators and empirical therapeutic Enoxaparin for suspected pulmonary embolism because of risk factors. Later CT chest was done with a 20 degrees "propped-up" position with both the expiratory and inspiratory phases. CT scan revealed a patent trachea in inspiration and anterior bowing of the posterior distal trachea with expiration, suggesting tracheomalacia proximal to the carina with cardiac and mediastinal shift to the left side. Given the extensive history of lung disease and recent radiotherapy, the decision was made not to undergo surgical intervention and stenting. Two months after initial presentation, the patient presented with acute respiratory distress and was intubated and started on antibiotics for health care associated pneumonia after CT revealed right lung consolidation. He was admitted to medical ICU service for 24 days without improvement and developed severe sepsis ending in septic shock and death.

Discussion:

Tracheomalacia is a tracheal weakness characterized by exaggerated narrowing during expiration. Tracheomalacia is one of the rare causes of non-specific respiratory symptoms such as dyspnea, cough, and wheezing, and is often misdiagnosed as asthma, COPD exacerbation and/or pulmonary embolism. Postpneumonectomy syndrome is one of the late complications of pneumonectomy which results from extreme shift and rotation of the mediastinum towards the empty hemithorax with subsequent compression of the airway. Prolonged airway compression may lead to tracheo-bronchomalacia, which is often evident after correction of postpneumonectomy syndrome. In our case, the patient had a left mediastinal shift with tracheomalacia evident without surgical correction, which developed approximately 15 years after left pneumonectomy. It was unclear why tracheomalacia presented after such a long duration, but recent radiotherapy to the chest may suggest that the radiation therapy was the most likely precipitating factor for such a complication.

Conclusion:

Tracheomalacia is a rare cause of orthopnea that should not be missed, especially in patients with a history of interventions that include pneumonectomy or radiation therapy.

**Author: Ashutossh Naaraayan, MD,MBBS**

Additional Authors: Vipul Pareek MD, Noah Kornblum MD, Ronald Rice MD, Bijal Amin MD, Prasanta Basak MD, Stephen Jesmajian MD

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

**Title: SWEET SYNDROME IN AML - A DIAGNOSTIC DILEMMA**

Introduction

Sweet syndrome was first described in 1964. We present a case where the diagnosis was challenging because of concern for an underlying infection.

Case report

A 79-year-old-man with hyperlipidemia and myelodysplastic syndrome presented with fever, chills and an enlarging plaque on the right flank for a week. There was no history of recent travel, trauma or tick bite. Vitals recorded were: HR 92/min, Temperature 103.4 F, BP 109/71 mmhg and RR 14/min. Skin lesion was 5 x 3cm, tender, indurated erythematous plaque with central excoriation. Serous discharge grew polymicrobial flora. Labs revealed: WBC 1.7x 10<sup>3</sup> cells/dL, absolute neutrophil count 700 cells/dL, Hgb 8.3 grams/dL and platelets 6,000 per/dL. Peripheral smear showed increasing myeloid blasts. He received cefepime, clindamycin and vancomycin and was discharged after eight days, on cefpodoxime and minocycline. Supportive treatment included transfusion of blood products and granulocyte-colony stimulating factor (G-CSF). Seven days post-discharge, he was readmitted with fever and persistent pain over the evolving skin lesion; 4x2 cm central eschar with surrounding 8x8 cm of indurated, dusky, tender skin without discharge. Two satellite lesions were noticed and biopsied. He was treated for neutropenic fever with cefepime and vancomycin. Surgical debridement was done. Bacterial cultures from blood and the plaque, fungal stain and culture and AFB staining were negative. Skin biopsy showed neutrophil rich perivascular and interstitial dermatitis with dermal edema consistent with sweets syndrome. A bone marrow biopsy was performed which revealed 47% myeloid blasts. Treatment with Cytarabine and Idarubicin (7+3) regimen for AML, lead to resolution of fevers within 2 days.

Discussion

Sweet syndrome or acute febrile neutrophilic dermatosis presents with fever, neutrophilic leukocytosis and tender, red, papules, nodules or plaques which respond to corticosteroids. Skin biopsy exhibits dense, neutrophilic infiltrate into the papillary dermis. It is classified into "classic, malignancy associated (MASS) and drug-induced variants. MASS is observed with both solid and hematologic malignancies, especially AML. MASS can present before, after or concurrently with the diagnosis of malignancy. Proposed etiologic factors include Yersinia infection, inflammatory cytokines (Interleukins and endogenous G-CSF), exogenous G-CSF and antineoplastic agents.

MASS can manifest in the presence of neutropenia. Neutropenia, rash and fever can indicate sepsis of dermal origin, making the diagnosis challenging. Potential underlying infection precludes the use of corticosteroids and oral therapy with colchicine or dapsone is recommended. In the present case, multiple cultures failed to reveal an infection and antibiotics were ineffective.

Definitive chemotherapy with 7+3 regimen lead to resolution of fevers, validating the clinical and histo-pathological diagnosis of Sweet syndrome.

## Resident/ Fellow Clinical Vignette

**Author: Yogeeta Naidu, MD**

Additional Authors: Vasv Singh;MD

Ziad Alkhour;MD

Institution: Unity Hospital, Rochester Regional Health System

**Title: CHEST PAIN AND ECG CHANGES ASSOCIATED WITH ACUTE CHOLECYSTITIS: AN UNUSUAL CASE OF CARDIO BILIARY REFLEX**

**Introduction:**

Chest pain with ECG changes is often a life threatening presentation, indicative of acute coronary syndrome. However, non-cardiac clinical conditions may lead to ECG changes mimicking cardiac ischemia. We describe a case of 64 year old male with chest pain and dynamic ECG changes likely due to acute cholecystitis.

**Case Presentation:**

A 64 year old male presented to ED with a 6-hour history of sudden onset, lower mid-sternal, non-radiating chest pressure associated with diaphoresis. No other significant co-morbidities. Vitals were stable except for blood pressure of 187/108 mm Hg and the results of respiratory, cardiovascular and gastrointestinal examination were normal. Complete blood count, Comprehensive metabolic panel were within normal limits and troponin-I was 0.05, then 0.03. ECG showed new T-wave inversions in leads I, aVL, V2, V3 and V4. With concern of possible acute coronary syndrome, cardiology was consulted and treatment initiated with sublingual nitroglycerine, aspirin, clopidogrel, low molecular weight heparin, metoprolol and statin. Echocardiogram showed normal ejection fraction with no wall motion abnormalities. Next day, patient developed fever with leukocytosis of 17,000 cells/cumm, and invasive cardiology workup was deferred for possible infection. Further work-up included pancultures (urine, blood and sputum) and chest x-ray which showed right basilar opacity. Within 24 hours, blood cultures grew gram-negative rods and patient was started on IV piperacillin-tazobactam and azithromycin for possible pneumonia. On day 3, total bilirubin increased to 2.7 mg/dL. Ultrasound abdomen showed no gall bladder pathology. Chest pain continued along with fever, leukocytosis and hyperbilirubinemia. Eventually, HIDA scan suggested cystic duct obstruction and acute cholecystitis. IV piperacillin-tazobactam and metronidazole were started, & patient underwent emergent percutaneous cholecystotomy. Post-surgery, chest pain resolved & clinical condition improved. ECG done after 2 weeks was normal and nuclear stress test was negative for ischemia.

**Discussion:**

Vagally mediated cardiobiliary reflex is the presumed mechanism that may lead to ECG changes in acute cholecystitis. There have been very few case reports of patients with cholecystitis presenting with ECG changes, these ECG changes usually resolve after the acute phase of illness. Physicians should be familiar with these associations and keep in mind while evaluating patients with similar presentations, Awareness is crucial to ensure appropriate diagnostic investigations and to avoid incorrect cardiac management.

This case report is unique in that our patient had no cardiac history, presented with ECG changes mimicking acute coronary syndrome and was later diagnosed with acute cholecystitis.

**Author: Roshni Naik, MD**

Additional Authors: Parikh P

Institution: Mount Sinai Beth Israel

**Title: Acute Generalized Exanthematous Pustulosis Resulting from Amoxicillin Graded Dose Challenge**

**Introduction:** Acute Generalized Exanthematous Pustulosis (AGEP) is a drug reaction characterized by onset of diffuse erythema covered by sterile pustules. Fever and neutrophilia is often present. Organ involvement is uncommon, but some report lymphadenopathy. The onset of the symptoms after drug administration varies from few hours to 3 weeks. We report the first case of AGEP that has been resulted from penicillin graded dose challenge.

**Methods:** Case Presentation

**Results:** We present a 35-year-old female with a remote history of an unknown reaction to penicillin many years ago who presented for a penicillin allergy testing. The patient had a negative prick and intradermal testing to both penicillin G and pre-pen, which were all negative with a positive histamine control and negative diluent control. The patient tolerated a graded dose challenge with no symptoms of amoxicillin 500 mg at 1/100 of the dose, 1/10 of the dose, and then the full dose with an hour in between each dose. Thirty-six hours later, the patient developed diffuse erythematous rash and tender right auricular and cervical lymphadenopathy. She was started on prednisone (1mg/kg). After two days, the rash worsened with pustule development, and she developed fevers. Laboratory results revealed a normal complete blood count with neutrophilia of 93.3%. EBV capsid IgM was negative and antinuclear antibody was elevated at 1:160 titer. Anti-dsDNA and anti-histone antibodies were negative. After ten days, the patient had resolution of symptoms.

**Conclusions:** Penicillin allergy testing is useful in diagnosing IgE mediated penicillin allergy in patients with an unclear or remote history of penicillin allergy. Her testing was negative since her allergy to penicillin is not IgE mediated but a delayed hypersensitivity. No standardized testing exists for delayed reactions and these medicines should be avoided if the history indicates. In this patient, prior history was unknown. This shows the importance of penicillin challenge following a negative skin test for the small percentage of patients who have an atypical reaction.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Angelica Nocerino, MD</b> Additional Authors: Nazish Ilyas, MD North Shore LIJ-Lenox Hill Hospital Institution: North Shore-LIJ Lenox Hill Hospital</p> <p><b>Title: IT DOES NOT HAVE TO BE A CATASTROPHE</b></p> <p>Introduction: Antiphospholipid syndrome is an autoimmune condition that creates a hypercoagulable state. Catastrophic Antiphospholipid Syndrome (CAPS) is a rare and life-threatening form of antiphospholipid syndrome characterized by acute vascular thrombus of at least three different organ systems, with a mortality rate of 50%.</p> <p>Case Report: A 48 year-old female with a history of hypertension presented to our institution with a chief complaint of chest pain. Vital signs on presentation were significant for tachycardia to 110 bpm. Laboratory workup revealed an elevated troponin, and an ECG demonstrating ST segment elevations in leads V1-V3. The patient was admitted for treatment of STEMI and started on a heparin drip. Catheterization showed a distal RCA thrombus, and an angioplasty was performed. Overnight, she became hypoxic, requiring intubation. A CT Angiogram of the chest demonstrated bilateral pulmonary emboli. The next day, the patient developed acute abdominal pain, for which an abdominal and pelvic CT angiography revealed liver and renal (iliac artery) infarcts. Given the rapidity and diffuse spread of thrombi, a hypercoagulable workup was undertaken. Antiphospholipid antibody was found to be positive, and she was treated for CAPS with a heparin drip, IV steroids, and plasmapheresis. During treatment, she remained hemodynamically stable with no further thrombotic events. She was discharged on Coumadin. Upon outpatient follow up at one month, patient was found to still have antiphospholipid antibodies with no further thromboembolic events.</p> <p>Discussion: Only 1% of patients with APS develop CAPS. Given this infrequency, an online registry was established in 2000 by the European Forum to document all of the cases of CAPS globally, currently with 280 reported cases. According to the International Congress on Antiphospholipid Antibodies Task Force, the following four criteria must be met to diagnose CAPS: (1) involvement of 3 organs, systems, and/or tissues (2) development of manifestations within a 1-week span (3) presence of antiphospholipid antibodies on two occasions 6 weeks apart (4) histological evidence of intravascular thrombosis. The major organ systems involved during a catastrophic episode are renal (71%), pulmonary (64%), central nervous system (62%), cardiovascular (51%), skin (50%), and hepatic (33%). Treatment for CAPS centers around inhibiting the inflammatory and thrombotic state through the use of anticoagulation, corticosteroids, intravenous immunoglobulins, and plasma exchange. With the appropriate treatment regimen, mortality decreases from 53% to 33%. In the acute setting, heparin is used with a transition to lifetime oral anticoagulation. High-dose steroids such as intravenous methylprednisolone is used for first three days, followed by oral prednisone. Plasma exchange with IVIG rapidly removes antibodies. Rituximab, defibrotide, and eculizumab can be used in refractory symptoms.</p> <p>Conclusion: CAPS is a rare disease diagnosed by widespread thrombotic disease and associated with high mortality rates. Diagnosis requires clinical suspicion and appropriate treatment vastly improves mortality.</p>	<p><b>Author: Christelle Judith Nong Libend, M.D</b> Additional Authors: Robert Busuego, Mohamed Chowdhry Institution: St John's Episcopal Hospital</p> <p><b>Title: Cardiac tamponade complicating severe hypothyroidism</b></p> <p>Pericardial effusion has been frequently described in hypothyroidism; this is attributed to increased capillary permeability and subsequent leakage of fluid high in protein into the pericardium. Although a relatively common incidence (3-6% in mild and 80% in severe and long standing hypothyroidism), it so rarely leads to cardiac tamponade that a search in PUBMED as of 2011 had only 81 case records.</p> <p>We present the case of a 52-year-old woman with past medical history of hypothyroidism, uterine fibroids, pericardial effusion, psychosis, delusional disorder, noncompliance with medications and refusing medical treatment who presented to the emergency department complaining of vaginal bleeding, abdominal pain and dizziness. Physical examination was significant for a BP of 154/60 mmHg, a pulse rate of 60 bpm, no thyroid enlargement, a large suprapubic mass and bilateral 2+ lower extremities pitting edema. TSH was &gt;100 mIU/ml, FT4 0.14 ng/dl, Total T3 0.69 ng/ml, albumin of 3.0 g/dl, total protein of 6.4 g/dl and Hgb level 5.9 g/dl. An ECG revealed low voltage. CXR showed an enlargement of the cardiac silhouette.</p> <p>In regard to her refusal of treatment, she was evaluated by psychiatry and was deemed to have no capacity to make medical decision and patient's NOK was involved in all medical decisions. During her stay, she developed obstructive nephropathy and was transferred to the ICU in order to receive blood transfusion under sedation and hysterectomy. As part of her preoperative workup given her history of pericardial effusion, an echocardiogram demonstrated a normal LVEF 55%, circumferential pericardial effusion (larger than 3 weeks ago) the widest diastolic pericardial space measured is 2.2 cm and partial diastolic collapse of right atrium, right ventricle and pulmonary artery consistent with early phase of pericardial tamponade.</p> <p>The patient underwent an urgent pericardial window with partial pericardiectomy. A tense and slightly thickened pericardium was found, 400 ml of serous fluid with a protein content of 3.7 was drained. The fluid sent for culture was negative and cytology analysis was negative for malignant cells. Patient was also started on levothyroxine.</p> <p>Two days later, she additionally underwent total abdominal hysterectomy and bilateral ureteral stent placement. The rest of the hospital course remained uneventful however complicated by her psychosis toward treatment and a CT done prior to discharge showed only trace pericardial effusion and TSH before discharge had decreased to 20 mIU/ml.</p> <p>The accumulation of fluids in body cavities in hypothyroidism has been widely reported, however the occurrence of cardiac tamponade is rare. It usually lacks the typical signs and symptoms just like our patient. The infrequency of this complication is possibly explained by the slow accumulation of fluid and early diagnosis. Our case highlights that hypothyroidism left untreated can lead to this rare but potentially fatal complication.</p>
---	---

## Resident/ Fellow Clinical Vignette

**Author: Odianos Obadan, MD**

Additional Authors: Lia Thomas, MD; Deborah Schron, MD; Raymond J. Pastore, MD  
Institution: St. Johns Episcopal Hospital

### **Title: A Rare Case of Hodgkin's Lymphoma in Accessory Spleen**

#### Introduction

Hodgkin lymphoma (HL) exclusively in the accessory spleen has been seldom reported in the literature. We report a case of a HIV positive man with Classic Hodgkin lymphoma with B symptoms and positive Epstein Barr Virus (EBV) LMP.

#### Case report

A 51 -year-old Nigerian man with undisclosed HIV status, non-compliant with antiretroviral therapy presented with complaints of fever, abdominal pain, jaundice, bone pains, diarrhea and weight loss of 2 years duration. He denied history of use of hepatotoxic or intravenous drugs. His workup done in Nigeria, India and Dubai included a bone marrow biopsy which revealed hypocellular bone marrow with fibrosis and plasmacytosis. As his symptoms worsened, he decided to seek treatment in the United States. Initial physical examination was unremarkable but his mental status deteriorated. Laboratory tests showed pancytopenia, elevated liver enzymes, coagulation profile and HIV positive (CD4 count 235 cells/mm<sup>3</sup> and undetectable viral load). Hepatitis, malaria parasite tests and cerebrospinal fluid tests were negative. CT abdomen showed accessory spleen and hepatomegaly. Laparoscopic wedge liver biopsy and excision of accessory spleen was done. Pathology of accessory spleen revealed Classical Hodgkin lymphoma, mixed cellularity type, CD15 +ve, CD30 +ve, Fascin +ve, MUM-1 +ve, PAX 5 +ve, EBV LMP positive in atypical cells. He showed symptomatic and laboratory improvement on antiretroviral therapy and was referred to an Oncology Center for ABVD (Adriamycin, Bleomycin, Vinblastine, Dacarbazine) treatment with outpatient follow up.

#### Discussion

HL is the most common non AIDS defining malignancy in HIV patients. The nodes are commonly involved (75%) while spleen is the most common extranodal site (20%). This case is unusual because lymphoma was only seen in the accessory spleen. Though incidence of AIDS defining cancers has declined, the incidence of HL in AIDS has increased, possibly due to the use of combination antiretrovirals and therefore improved immunity. Nearly all cases in HIV patients are associated with EBV (70-80%), B symptoms, and histologically, half of cases are mixed cellularity as seen in the patient above. EBV is suggested as an important etiological factor in the development of HIV associated HL. The incidence of HL peaks at CD 4 counts between 150 to 199 and HL with CD4 counts less than 200 associated with a poorer prognosis. Currently, ABVD is the standard of treatment for AIDS related HL as well as HL.

**Author: Helen Okoh, MD**

Additional Authors: Sandeep Singh MD ,Spencer Langevin MD ,Susan Sanelli-Russo MD, Adriana Abrudescu MD  
Institution: Queens Hospital Center

### **Title: A CASE OF SYSTEMIC LUPUS ERYTHEMATOSUS PRESENTING AS MILLER-FISHER VARIANT OF GUILLAIN-BARRE SYNDROME**

A 41year old female with no past medical history presented to the Emergency Department with worsening lower extremity weakness and swelling for 3 months. She also complained of 2 days of Right eye swelling with diplopia and blurry vision and 1 day history of inability to walk. On physical examination ,there was no evidence of skin rashes or joints inflammation , there was bilateral lower extremity pitting edema with decreased motor strength in the proximal upper and lower extremities . Reflexes were equal and symmetric in upper extremities but diminished in lower extremities .Cranial nerve examination revealed anisocoria Right more than Left , with no nystagmus .

4 days after admission the patient's neurological status worsened and was intubated for airway protection. Due to absent reflexes in addition to progressive lower extremity weakness, ophthalmoparesis with inability of the eyes to cross the midline bilaterally, GBS was suspected. Nerve conduction studies confirmed the diagnosis. Patient was treated with plasma exchange for 5 sessions, with no improvement at the same time Hemodialysis was started due to acute renal failure . SLE was confirmed by ACR criteria, renal biopsy showed diffuse proliferative lupus nephritis. Pulse SoluMedrol therapy 1gm daily for 3 days was started. Cyclophosphamide 500mg every 2 weeks together with IVIG 0.4g/kg/day were started. Cyclophosphamide was terminated after the 3rd dose due to pancytopenia ,fever with pneumonia and worsening of the sacral decubitus. In view of SLE with positive antiribosomal P protein and lack of improvement in the neurological/GBS symptoms 2months after the onset of disease the 2nd course of IVIG was started 2g/kg divided over 5days. Significant daily improvement in motor function and reflexes started to occur. Patient was discharged home 131 days after initial presentation on prednisone 20mg daily . Patient was seen as an outpatient within 1 month post discharge with no significant residual motor or sensory deficits, walking without support, and asymptomatic for weakness. Guillian Barre as the Initial presentation of SLE has been reported in only a few cases, varying responses has been noted with each patient encounter and even now no universal treatment guidelines have yet been established . A 2nd course of IVIG was given in this patient after the initial dose because of the absence of neurological improvement after the first course of IVIG , and plasmapheresis despite the fact that serologically SLE improved . Significant and profound recovery was noted. SLE complicated by concurrent GBS with no neurological improvement after IVIG, Plasmapheresis or Cyclophosphamide should prompt consideration of a 2nd course of 5 days of IVIG with concomitant steroid use.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Cesar Orellana, MD</b> Additional Authors: Cesar Orellana, MD; Amit M Sharma, MBBS; Anna Orellana, MD; Priyanka Pitroda, MD; Tasaduq Fazili, MD. Infectious Disease, SUNY Upstate Medical University, Syracuse, NY. Institution: SUNY Upstate Medical University</p> <p><b>Title: A Gastrointestinal Diagnosis you may not Have Considered: Intestinal Spirochetosis</b></p> <p>Background/Purpose: To raise awareness about this little-known gastrointestinal disease entity, which may be misattributed to irritable bowel syndrome.</p> <p>Intestinal spirochetosis [IS] was first described in 1967. It is a condition defined histologically by the perpendicular attachment of anaerobic spirochetes (most commonly <i>Brachyspira aalborgi</i> and <i>brachyspiral pilosicoli</i>) to the apical surface of colorectal epithelial cells, which appears as a false brush border. IS manifests as various, chronic gastrointestinal symptoms such as diarrhea, constipation, alternating bowel habits, abdominal pain, and bloody stools. The epidemiology of IS is described only sporadically, e.g., prevalence in general populations in Japan, Sweden, and Norway are reported as 0.4%, 2%, and 2.5%, respectively. There is an association with diarrhea predominant irritable bowel syndrome and a higher prevalence in Human Immunodeficiency Virus (HIV) seropositive persons.</p> <p>Methods: A 39-year-old man was referred to our clinic for left lower quadrant [LLQ] abdominal discomfort and a sensation of fullness for more than 1 year. This was mild-to-moderate in intensity, intermittent, diffuse, and non-radiating. He also strained to defecate and denied improvement of discomfort after defecation. Chronically, he had had 1-2 loose stools per day without hematochezia, melena, or mucopurulence.</p> <p>Results: On exam, he had normal bowel sounds, no mass, distention, tenderness to palpation (including rebound), or guarding. Dullness to percussion was noted in the LLQ. Stool culture and ova and parasite studies were unrevealing. He had no fecal leukocytes. An HIV screen was negative. <i>Clostridium difficile</i> toxin was not detected. A colonoscopy revealed only internal hemorrhoids. A random colonic biopsy demonstrated benign histology and excluded microscopic colitis. Tissue transglutaminase IgA and IgG antibodies were not detected. Probiotics, fiber supplementation, and increased water intake failed to improve these gastrointestinal symptoms.</p> <p>Finally, a hematoxylin-eosin stained high-power section of the surface of colonic epithelial cells revealed a prominent basophilic fringe appearing along the luminal surface. Warthin-Starry stain for spirochetes highlighted the microorganisms along the brush border as a dark brown/black line. Metronidazole 500mg PO QID for 10 days resolved this condition.</p> <p>Conclusion: Evaluation for IS may be warranted in those with otherwise unexplained chronic gastrointestinal symptoms because this is an entirely treatable condition.</p>	<p><b>Author: Nick Pakzad, MD</b> Additional Authors: Kiranjit Uppal, MD William Stuart, MS IV Institution: Stony Brook University Hospital - Department of Internal Medicine Program</p> <p><b>Title: Normal Serum Lipase in the Setting of DKA Masking the Diagnosis of Acute Pancreatitis</b></p> <p>Guidelines for diagnosing acute pancreatitis require two out of three criteria: classic clinical symptoms, serum amylase or lipase levels above three times the upper limit of normal, or characteristic findings on imaging. In addition to classical clinical findings, the vast majority of patients with pancreatitis have lipase elevation as a result of acinar cell inflammation and leak, rendering imaging unnecessary. Elevated serum lipase has a sensitivity of 99% for acute pancreatitis, making it a reliable diagnostic marker. In rare instances, however, acute pancreatitis can have an atypical clinical presentation with strikingly normal lipase levels.</p> <p>A 41 year old male with a past medical history of diet-controlled Diabetes Mellitus Type II presented with one week of general malaise associated with nausea and vomiting. In the Emergency Department, vital signs showed a blood pressure of 147/104, heart rate of 122, respiratory rate of 20, and a temperature of 97.4F. Physical exam was significant for profound lethargy, dry mucous membranes, tachycardia, and mild left lower quadrant tenderness to palpation. Labwork revealed hyperglycemia in the 400s associated with an anion gap. The patient was diagnosed with Diabetic Ketoacidosis (DKA) and started on an insulin drip with subsequent resolution of hyperglycemia and closure of anion gap. The patient was planned for transition from insulin drip to subcutaneous insulin, however though the labwork appeared to be improving and the patient had only mild tenderness to palpation of the left lower quadrant, the etiology of the DKA remained unclear. In addition, the patient continued to be lethargic and tachycardic. The patient's clinical status led to further workup for an inciting factor, including serial serum amylase and lipase levels over several days which consistently remained within normal limits. A CT abdomen and pelvis was performed, which revealed extensive stranding seen surrounding predominantly the tail and head of the pancreas, consistent with severe acute pancreatitis. Additional labwork revealed triglycerides to be 2969. The patient was subsequently started on aggressive intravenous hydration and given nothing by mouth, as well as continued insulin drip use to lessen serum triglyceride burden. Once the management for pancreatitis had been initiated, the patient had significant improvement of symptoms. Given the atypical characteristics of the patient's abdominal exam (mild left lower quadrant tenderness), the patient's pain was initially attributed to the common nonspecific pains seen in DKA. The underlying severe acute pancreatitis was further masked in the setting of normal serum lipase, making this a diagnostic challenge for physicians. This case suggests that patients presenting with DKA of unclear etiology may warrant a more aggressive investigation for the inciting factor, even if traditionally accurate serum markers are within normal limits. This is an instance where utilizing a low threshold for imaging can improve patient outcomes.</p>
---	---

## Resident/ Fellow Clinical Vignette

**Author: Aakriti Pandita, MD**

Additional Authors: Gaganjot Singh, Viveksandeep Thoguluva Chandrasekar  
Institution: SUNY Upstate Medical University

**Title: Levodopa/Carbidopa Continuous Release (CR) precipitating Serotonin Syndrome in a patient receiving tube feeds.**

Due to erratic absorption of Levodopa/ Carbidopa CR through tube feeds, IR (immediate release) form is used in these patients. A decrease in dose is known to cause Neuroleptic Malignant Syndrome (NMS) while an increase is likely to result in Serotonin Syndrome (SS). A 55 y/o male with intrathecal baclofen pump for spastic quadriplegia from basilar artery stroke, presented with yellowish discharge from the skin overlying the pump. Despite being nonverbal, patient responded with slight nodding or eye blinking. ?

Though afebrile with stable vitals he was started on vancomycin and levofloxacin for 4-6 weeks due to high suspicion for infection. In anticipation of pump removal, intrathecal dose was decreased and oral baclofen 20 mg q6h added. On day 4 following pump removal there was transient increase in spasticity responding to increased oral baclofen (30mg q6h) and valium 2mg BID. This was followed by quick resolution. Home medications were continued: Oxycodone for pain, Amantadine and Levodopa/ Carbidopa 25/100 q6h for dystonia.?

Day 12, the patient was noticed being startled frequently, with increasing flushing and diaphoresis; tachypneic, tachycardic with SBP between 150-199's as well as low-grade fever (<38.3C). WBC count was 15.2 and there was mild metabolic acidosis with lactic acid of 2.1. EKG showed ST segment up sloping in V4, V5, V6 with some troponin leak trending up to maximum of 0.22, while his CK was initially elevated at 213, increasing to 350 two days later. He had troponin leak thought to be due to type 2 NSTEMI from sepsis. A CT abdomen ruled out an abscess and his blood cultures showed no growth.

A CTA thorax didn't show any evidence of pulmonary embolism. On day 15, it was noticed that patient was getting Levodopa/Carbidopa CR crushed through this PEG tube from his day of admission. CR was switched to IR form with addition of Cryproheptadine, following which his symptoms drastically improved. No source of infection was located and patient responded to presumptive treatment for SS. The dose of IR form was then decreased and following resolution he was sent home.?

Changes in drug formulation or route can precipitate SS. Administration of the Levodopa/Carbidopa IR through tube feeds has been reported to precipitate Neuroleptic Malignant Syndrome (NMS) relating to protein content of tube feeds (by decreasing absorption of the drug.) Pharmacy errors are quite common with regards to switching continuous and immediate release form of this drug. In addition there are situations like this case where none of the diagnostic criteria's for SS can be applied and patients need to be treated presumptively.

**Author: Narmadha Panneerselvam, M.B.B.S.**

Additional Authors: Amanda Engle Pharm D; Carol Beechy MD; Connie Jastremski MS,RN; Kulothungan Gunasekaran MBBS; Eric Bravin M.D.  
Institution: Bassett Medical Center

**Title: PCA for Refractory Dyspnea: Old technology, Novel Strategy**

Introduction:

Effective palliation of dyspnea in patients with advanced heart, lung, and neoplastic disease requires that clinicians interpret patient reported symptom severity, life expectancy, and psychosocial factors in the context of underlying causes and comorbid conditions when selecting appropriate pharmacological and non-pharmacological treatment. Palliative care guidelines from the National Comprehensive Cancer Network (NCCN), American Thoracic Society (ATS), American College of Chest Physicians (ACCP), and American College of Physicians (ACCP) recommend oral or parenteral opioids for severe dyspnea in advanced disease. Despite these recommendations, the role of Patient Controlled Analgesia (PCA) for delivery of parenteral opioids in this setting has not been evaluated. We describe successful palliation of severe intermittent dyspnea via morphine PCA.

Case Presentation:

A 76 year old male with transfusion refractory myelodysplastic syndrome (MDS) with severe anemia, moderate chronic obstructive pulmonary disease, and systolic heart failure was admitted with dyspnea, multifactorial in etiology. He had slight dyspnea (Modified Borg scale 2-3) at rest, and was maximally dyspneic (scale 10) with slight movement. He was initially trialed on standard oral morphine with breakthrough intravenous (IV) nurse administered boluses, along with treatment for CHF and COPD. Despite these measures, he continued to have breakthrough episodes of severe dyspnea, that were distressing and anxiety provoking. To overcome the delay from symptom onset to medication administration and effect, the patient was started on morphine PCA 2 mg bolus with lockout 30 minutes without basal dose, along with oral long acting morphine. We noticed a remarkable subjective and objective (maximal Modified Borg scale 3-4) improvement in both frequency and severity of dyspneic episodes, baseline dyspnea, and associated anxiety with 6-8 demands delivered per 24 hours.

Discussion:

While PCA is commonly used for pain control, only a single case report mentions its use in control of dyspnea. According to ACCP, dyspnea is considered analogous to the perception of pain and consists of sensory (intensity) and affective (unpleasantness) dimensions, with similar cortical processes, both resulting in human suffering. While concern for the safety of opioids is relevant, no studies have identified excess mortality associated with appropriate use of opioids for dyspnea. Since dyspnea can be anxiety provoking, empowering patients with the ability to quickly alleviate dyspnea may have additional anxiolytic impact. Utilizing PCA for dyspnea provides patients with an element of control in the face of an illness and environment which is out of their control.

Conclusion:

PCA use for control of dyspnea may provide a more effective tool for patients suffering from severe intermittent dyspnea not relieved by other traditional treatment modalities.

## Resident/ Fellow Clinical Vignette

**Author: Narmadha Panneerselvam, M.B.B.S.**

Additional Authors: Kulothungan Gunasekaran MBBS; Sowmya Chidurala MBBS; Saeda Fatima MBBS; and Venu Thirukonda MD.

Institution: Bassett Medical Center

**Title: Metastatic Infiltration of The Psoas: A site less traveled.**

Introduction:

Metastatic involvement of the psoas muscle can occur uncommonly in pulmonary, gastrointestinal, and genitourinary cancers. We report the case of bilateral Psoas muscle metastases in a patient with advanced gastric cancer.

Case Presentation:

A 70 year old female with stage IV advanced Gastric adenocarcinoma diagnosed about a year ago, was admitted with complaints of bilateral hip and thigh pain, and weakness of both legs. On examination, she had decreased hip flexion and extension, and edema of both legs. At the time of initial diagnosis, the Positron Emission Tomography-Computer Tomography (CT) showed posterior mediastinal, bulky retroperitoneal and left supraclavicular lymphadenopathy with left humeral and right femoral skeletal metastases, without other visceral metastases. After completing 9 cycles of docetaxel, cisplatin, and 5-FU palliative chemotherapy, she was noted to have worsening retroperitoneal lymphadenopathy on imaging. Plan for switching to second line Ramucirumab was made. A body CT scan done during this admission showed extensive tumor infiltration and enlargement of Psoas muscles bilaterally. Her pain was well managed with opioids. The patient elected to proceed with Hospice care due to the declining performance status.

Discussion:

The prevalence of skeletal muscle metastases is extremely low(0.03%) due to changes in the blood flow with turbulence and muscle contractions preventing settlement of tumor cells, acidic environment caused by lactate, and increased natural killer cells activity. The Skeletal muscle metastases are associated with carcinomas (64.6%-comprising pulmonary, gastrointestinal and genitourinary in the respective order), more often than leukemia (14.7%) or lymphomas (20.6%). The Diaphragm(67.6%) and Iliopsoas(29.4%) are most commonly involved. Unilateral metastatic involvement of the Psoas mimicking Psoas abscess and presenting as Malignant Psoas Syndrome characterized by proximal lumbosacral plexopathy, painful fixed flexion of the ipsilateral hip with radiological or pathological evidence of ipsilateral Psoas muscle malignant involvement has been described in the past. But we could not locate any study report on bilateral involvement. The several modes of Psoas metastasis are direct invasion from adjacent lymph nodes(55%), primary tumor(20%) or a local recurrence(8%), and direct extension from vertebral metastasis(12%). The involvement of the Psoas in Gastric adenocarcinoma is very rare, as it most commonly metastasizes to the liver, peritoneal surfaces, and distant lymph nodes, and less commonly to the ovaries, central nervous system, bone, pulmonary and soft tissue. Often, pain control may be challenging. Our patient had bilateral involvement of the Psoas due to the advanced Gastric cancer, likely from invasion from adjacent lymph nodes. Psoas involvement typically occurs in the very advanced stage of Gastric Adenocarcinoma, possibly signifying poor prognosis.

Conclusion:

Malignant Psoas Syndrome may be suspected in patients with Gastric adenocarcinoma presenting with pain and myopathy of a lower extremity, and very rarely it can be bilateral.

**Author: Cristian Papazoglu, MD, PhD**

Additional Authors: Sumaira Shafi, MD

Stanley Holstein, MD

Stephen Jesmajian, MD,FACP

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

**Title: Asymptomatic central pontine myelinolysis: an early manifestation of lymphoma?**

Central pontine myelinolysis (CPM), now known as osmotic demyelination syndrome, is an acquired demyelinating lesion of the pons that typically occurs after rapid correction of hyponatremia. CPM is most often found in patients with chronic alcoholism, malnutrition, hyponatremia, liver disease, liver transplants and infections. Patients develop symptoms ranging from asymptomatic to extremely severe, possibly lethal. Typical symptoms include behavioral changes, confusion, mutism, dysarthria, dysphagia, bulbar and pseudobulbar paresis, hyperreflexia, quadriplegia and seizures.

Our patient is a 58 year old male with history of alcohol abuse, who presented with progressive dyspnea on exertion for 2 months. He also complained of fever, night sweats, anorexia and malaise for approximately 1 year. He underwent extensive workup for infections and malignancy but everything to date was negative. Physical exam was unremarkable. Labs showed anemia of mixed etiology (due to low vitamin B12 and folate levels and chronic inflammation) and hyponatremia (Na=127). During the 2nd day of hospitalization patient complained of neck pain. MRI of cervical spine showed an abnormal signaling in the pons and brain MRI was recommended. MRI of the brain revealed a hyperintense lesion in the pons on T2WI with diffusion restriction on DWI, consistent with CPM. Sodium level remained low (Na=130) and patient's neurological exam was negative. Further workup led to diagnosis of diffuse large B-cell lymphoma. Systemic chemotherapy led to resolution of CPM.

The patient had multiple risk factors for developing CPM: history of chronic alcohol abuse, hypovitaminosis and moderate hyponatremia (never aggressively corrected). He was asymptomatic and denied having any symptoms suggestive of CPM prior to admission, despite significant changes on brain MRI. There are only a few reports in the literature describing asymptomatic CPM. Interestingly, a case reported in 2012 by Shah et al, described a patient with history of alcohol abuse and lymphoma (although a different histologic type) that had asymptomatic CPM. Yamamoto et al described neuroradiologic findings in the form of hyperintense lesions located in central pons, in 5 of 11 patients with intravascular large B-cell lymphoma. These radiologic findings were similar to those seen in pontine osmotic demyelination syndrome. Remarkably, in 4 of the 5 patients, treatment of lymphoma led to a decrease in pontine lesions seen on MRI. These findings and our case, strongly suggest that there could be a correlation between asymptomatic CPM and at least some histologic subtypes of lymphoma.

Could these asymptomatic pontine lesions be an early manifestation of lymphoma? More studies are needed to fully elucidate if this connection occurs predominantly with specific lymphoma subtypes and also to understand the pathogenesis, underlying mechanisms and treatment implications of this association.

## Resident/ Fellow Clinical Vignette

**Author: Nirav Parikh, MD**

Additional Authors: Harris Kalatoudis, MD, Allen Weiss, MS, Doris Zavaro, MD, Adib Alhaddad, MD, Spiro Demetis, MD NYU Lutheran Medical Center  
Institution: NYU Lutheran Medical Center

### **Title: HYPERCALCEMIA INDUCED PANCREATITIS: AN UNUSUAL ETIOLOGY**

#### Introduction

Pancreatitis is an inflammation of the pancreas that can be attributed to various etiologies including gallstones and alcohol. Acute pancreatitis is a time-sensitive medical emergency. We present an atypical evolution of acute pancreatitis from ingestion of large amounts of TUMS® (calcium carbonate).

#### Case Presentation

A 47-year-old man with a past medical history of type 2 diabetes, hyperlipidemia, and asthma presented with severe abdominal pain for two days prior to admission after ingesting half a bottle of TUMS® for abdominal pain. The patient was confused and displayed altered mental status. His pain was 10/10 in intensity, non-radiating, exacerbated with movement, and associated with nausea, loss of appetite, three episodes of non-bilious, non-bloody vomiting, and non-bloody diarrhea. He denied any fever, weight loss, chest pain, or shortness of breath. The patient also denied any history of pancreatitis or ever having abdominal pain of this intensity. Labs were significant for the following: amylase level, 1859 U/L; lipase level, 4210 U/L; leukocytosis, 14.1 k/uL; calcium, 15.4 mg/dL; glucose, 243mg/dL; potassium, 5.2 mmol/L; triglycerides, 257 mg/dL; LDH, 173 IU/L; and AST, 36 u/L. Ranson's criteria point count at admission was 1. CT without contrast was remarkable for extensive pancreatic and peripancreatic edema with intra-abdominal fluid consistent with acute pancreatitis. Patient developed ARDS while in the ICU and was transferred to another facility for Extracorporeal Membrane Oxygenation (ECMO); however, treatment failed and the patient expired shortly afterwards.

#### Discussion

Acute pancreatitis is an inflammatory disease of the pancreas with a myriad of etiologies. Gallstones and chronic alcohol abuse account for 75% of the cases in the United States. Other causes include mechanical obstruction of the pancreatic duct, hyperlipidemia, hypercalcemia, infection, direct trauma, congenital, ischemia, vasculitis, and genetic.

The mechanism of acute pancreatitis begins with the intra-acinar activation of proteolytic enzymes that start a cascade of pancreatic autodigestion. Further release of enzymes due to ruptured cells causes swift spread throughout the gland and into the tissues surrounding the pancreas. Then, these enzymes also damage the vascular endothelium of the acinar cells, propagating pancreatic ischemia and increased permeability, leading to edematous changes surrounding the organ. Accumulation of inflammatory cells, cytokines, and free radicals further promotes ischemia and edema of the pancreas, and eventually necrosis. In this patient, the large amount of TUMS® (calcium carbonate) created an acute hypercalcemic crisis that caused acute pancreatitis. Physicians and medical personnel should be aware that ingestion of large quantities of TUMS® or similar supplements may lead to dangerously increased serum calcium levels.

**Author: Eshan Patel, MD**

Additional Authors: Galyna Ivashchuk MD, Richard Zuniga MD  
Institution: New York Methodist Hospital

### **Title: Osteolytic Bone Lesions in Metastatic Prostate Cancer**

#### INTRODUCTION

Prostate cancer is the second leading cancer-related death in men in United States and the fifth most common cancer worldwide. Mortality and morbidity are related to advanced disease, and metastasis to lymph nodes, bones and lungs. Bone lesions are characteristically osteoblastic on radiologic studies, and commonly involve vertebrae, sternum, pelvic bones, ribs and femurs.

#### CASE REPORT

A 67 year old male with history of DMII, HTN, HLD and prostate cancer was admitted after 3 weeks of progressive and incapacitating hip and back pain. Patient was diagnosed with prostate cancer in 2009 (unknown Gleason score, non-metastatic at time of admission). After initial diagnosis, patient received external beam radiation therapy. Patient was not eligible for radiation seeds due to a 'cardiac condition' and subsequently was treated with bicalutamide alone. According to the patient, his PSA improved and was stable for a number of years. Four months ago, the PSA began to rise and he was started on leuprolide. Upon admission, patient denied weight loss, hematuria, bowel or urinary incontinence; saddle numbness, focal motor or sensory deficits. On physical examination patient had lumbar spinal tenderness to percussion. His PSA level was 750 ng/ml, testosterone level was 61.45 ng/dl. Magnetic resonance imaging showed diffuse osteolytic lesions involving his lumbar, thoracic and sacral spine and an osteolytic soft tissue mass in the skull base. Imaging revealed diffuse retroperitoneal lymphadenopathy, which prompted work-up for multiple myeloma and lymphoma that turned out negative. Retroperitoneal lymph node biopsy was consistent with metastatic prostate adenocarcinoma. Patient was planned for palliative radiation therapy to the spine to be followed by docetaxel as outpatient.

#### DISCUSSION

Osteolytic bone metastasis in prostate cancer is extremely rare and only case reports exist in the literature. Because prostate metastasis is typically osteoblastic in nature, the presence of osteolytic lesions can cause delay in diagnosis, and work up for other causes of bone involvement, namely multiple myeloma, as in our case. PSA levels greater than 20 ng/ml and ALP greater than 90IU/L are predictors for presence of bone metastasis in patients with prostate cancer. Up to ninety percent of patients with advanced disease have bone involvement, which contributes to morbidity, with pain, immobility, pathological fractures, hypercalcemia, hematological disorders and spinal cord compression.

During metastasis to the bones osteoblasts and osteoclasts become activated. PSA is thought to promote proliferation of osteoblasts and apoptosis of osteoclasts, which is a reason why osteoblastic metastatic lesions predominate in prostate cancer. It has been theorized that in patients with predominantly osteolytic bone lesions, nuclear factor kappa-B ligand (RANKL) and osteoprotegerin (OPG) balance is altered. RANKL promotes osteoclastic activity, while OPG protects the skeleton from excessive bone resorption by binding RANKL and preventing it from binding its receptor.

<p><b>Author: Ronakkumar Patel, MD</b>                  Additional Authors: Acharya, Gyanendra MD, Moe, Khaing MD                  Institution: Wyckoff Heights Medical Center</p> <p><b>Title: Autoimmune Hepatitis: Diagnostic Dilemma when it is disguised in Iron Overload Syndrome</b></p> <p><b>INTRODUCTION:</b>                  Elevated serum ferritin level is a common finding in iron overload syndrome, autoimmune hepatitis (AIH), viral hepatitis, alcoholic and nonalcoholic fatty liver disease. Elevated transferrin saturation is not a common finding in above diseases except for iron overload syndrome. Very high transferrin saturation (~91%) is almost pathognomonic for iron overload syndrome. We encountered a challenging case of autoimmune hepatitis disguised in simulated iron overload syndrome and it required extensive work ups to arrive at final diagnosis.</p> <p><b>CASE PRESENTATION:</b>                  73 years old female with past medical history of hypertension and hypothyroidism presented with chief complaint of dark color urine for few weeks associated with dull abdominal pain, yellowish discoloration of skin and fatigability. She denied fever, recent travel, use of herbal medication, smoking, alcohol or illicit drug, blood transfusion and family history of liver disease.</p> <p>On examination, she was well-oriented obese (BMI 35) lady with stable vital signs. Moderate icterus was present. Systemic examinations were unremarkable except for mild tenderness in right upper quadrant of abdomen. Initial labs reported mild anemia; elevated ESR (44mm/hr), direct bilirubin 7.8 mg/dl (total bilirubin 10.9 mg/dl), AST (909 IU/L), ALT (826 IU/L), ALP (289 IU/L) and total serum protein (8.7gm/dl). Iron profile showed elevated transferrin saturation (~91%), which is considered to be pathognomonic to primary iron overload syndrome. However, Magnetic Resonance Imaging (MRI) of Liver was negative for iron overload. In the view of negative family history of hemochromatosis and unremarkable MRI liver, alternative causes of hepatic dysfunction were looked for. Drug, alcohol and viral hepatitis were essentially ruled out from history and laboratory investigations. Abdominal ultrasound and CT reported normal CBD. Serum copper level was normal. Anti-mitochondrial, anti-smooth muscle and kidney-liver antibodies were negative. Serum anti-nuclear antibody (ANA) with titer 1:160 and history of hypothyroidism (Hashimoto's) directed us to liver biopsy. Liver biopsy report was consistent with autoimmune hepatitis. So, autoimmune hepatitis was diagnosed based on AIH diagnostic criteria with positive liver biopsy, ANA and hypergammaglobulinemia IgG. Patient responded well on prednisone. She has been following up at clinic.</p> <p><b>DISCUSSION:</b>                  The diagnosis of AIH is based on histological finding of interface hepatitis with portal plasma cell infiltration, hypergammaglobulinemia and autoantibodies (ANA, SMA, and anti LKM). Viral hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, Wilson's disease, hemochromatosis and drug-induced hepatitis must be considered as differentials. Elevated transferrin saturation in AIH (as in our case) is most likely secondary to increased serum iron (through hepatocellular necrosis) and decreased transferrin synthesis (through liver failure). Markedly elevated transferrin saturation can simulate iron overload syndrome but liver biopsy can guide physicians to navigate the diagnosis. Prognosis of AIH is good if recognized and treated early.</p>	<p><b>Author: Kinner Patel, MD</b>                  Additional Authors: Ryann Quinn (Medical Student ACP member), Kriti Devkota, MB and Brain Changlai, MD.                  Institution: SUNY Upstate Medical University</p> <p><b>Title: A REVIEW OF PCP PNEUMONIA IN THE SETTING OF CHRONIC STEROIDS AND B-CELL TARGETED IMMUNE SUPPRESSANT THERAPY</b></p> <p><b>Introduction:?</b>                  Pneumocystis jirovecii is an opportunistic organism which almost exclusively infects immune compromised individuals causing pneumonia and acute respiratory failure. Although reports of patients on non-specific and T-cell mediated immune suppressant therapy developing Pneumocystis pneumonia (PCP) are present; reports of patients on steroids and B-cell targeted therapy complicated with PCP are recent. Here we present and discuss a case of sero-negative HIV female status-post Rituximab therapy and on prednisone for 10 weeks developing PCP.??</p> <p><b>Case:?</b>                  76-year-old sero-negative HIV female with a history of immune thrombocytopenic purpura (ITP) on prednisone therapy was admitted for diverticulitis and diverticular bleeding. She was diagnosed with ITP 2 months prior to admission and was on prednisone 80 mg daily for 6 weeks, followed by a prednisone taper for 4 weeks. At the time of admission she was on 15 mg prednisone daily. She had also received 4 weekly doses of Rituximab, last dose being 1 month prior to admission. Her diverticulitis was treated with piperacillin-tazobactam and her diverticular bleed resolved spontaneously without intervention. At the time of admission she was noted to have acute hypoxemic respiratory failure, with arterial pO<sub>2</sub> of 66 mmHg at FiO<sub>2</sub> of 30%. Her hypoxemia did not improve with resolution of gastrointestinal bleeding and sepsis and persistently required 28-32% FiO<sub>2</sub>. Although chest x-ray showed no significant pulmonary edema, she had rales on exam and was diuresed without significant improvement. She underwent echocardiogram, lower extremity doppler, V/Q scan and repeated chest x-rays which were unremarkable. Chronic steroid therapy and Rituximab therapy prompted a chest CT and sputum culture for PCP. Chest CT showed diffuse scattered ground glass opacities and sputum induction was negative for PCP. She underwent bronchoscopy with bronchoalveolar lavage which was positive for PCP and HSV-1. Patient was then started on trimethoprim-sulfamethoxazole 450 mg IV TID and prednisone 40 mg BID. Her antibiotic dose was subsequently changed to oral trimethoprim-sulfamethoxazole 160-800 mg for a total of 21 days with steroid taper after 5 days and Valtrex for 14 days for HSV-1. It was recommended that if she continues to be on steroids, she will start prophylactic trimethoprim-sulfamethoxazole.</p> <p><b>Discussion:?</b>                  T-cell immunity is classically related to defense against PCP however, recent data indicates the involvement of B-cell immune suppression to be associated with Pneumocystis. The above case illustrates PCP infection in B-cell immune suppression therapy along with chronic steroid therapy and supports the use of primary prophylaxis in patients with chronic steroids who have received Rituximab therapy.</p>
--	--

## Resident/ Fellow Clinical Vignette

**Author: ROSHNI Radhakrishna, MBBS**

Additional Authors: HAYAS HASEER KOYA MD, DONA VARGHESE MD, RAJEEV YALAMANCHILI MD  
Institution: SUNY UPSTATE MEDICAL UNIVERSITY

**Title: I SEE HORSES IN THE ROOM- AN INTRIGUING CASE OF NEURO DEGENERATIVE DISEASE IN AN ELDERLY MALE**

When an elderly patient presents with dementia and ataxia, there are a multitude of differentials one must think about. We present a case of a rare neurodegenerative disease presenting with predominant visual symptoms. An 80 year old man with a history of early Parkinson's disease (not on any medications) was admitted to our institution because of episodic confusion, visual hallucinations and intermittent diplopia of 2 weeks duration. Prior to this admission, family reported that except for some lapses in short term memory (which was attributed to normal aging), he was normal. His visual hallucinations were seeing horses in the room and in the walls around him. Ophthalmological evaluation was unremarkable. Neurological examination revealed myoclonic jerks of the left arm, cogwheel rigidity and ataxic gait. MRI brain done was unremarkable. An EEG revealed slowing and disorganization of waking background activities, polymorphic, slowing in right hemisphere and intermittent presence of periodic lateralized epileptiform discharges(PLED) localized to the right central parietal region. The patient's clinical condition deteriorated rapidly with worsening myoclonus. A repeat EEG revealed status epilepticus and antiepileptic therapy was initiated. Continuous EEG monitoring revealed myoclonic activity. Lumbar puncture and CSF analysis did not reveal any evidence of meningitis but revealed presence of Anti Tau antibody(6120 pg/ml) and protein 14-3-3(1150 pg/ml).A diagnosis of CJD was made , but because of the rapid clinical deterioration, the family opted to pursue comfort care measures and the patient eventually succumbed to his illness.

CJD is a form of sub acute spongiform encephalopathy caused by prions and may be sporadic, familial or acquired. 80%-90% of the cases are sporadic and affected patients are usually between 50 and 75 years of age. Visual symptoms are common in sporadic CJD and have been reported in at least 20% of patients in early stage. The diagnosis of CJD according to WHO criteria require 1) progressive dementia and two or more of: myoclonus, visual or cerebellar dysfunction, pyramidal/extra pyramidal signs or akinetic mutism 2) typical EEG and/or a positive 14-3-3 CSF assay and a clinical duration of less than 2 years before death and 3) exclusion of alternative diagnoses with routine investigations. For a definite diagnosis one of the following 2 criteria should be established in addition to the above: characteristic pathological changes in the brain or a positive Western blot to confirm the presence of prion protein Pr Psc. No effective treatment has been identified for CJD and is mainly supportive care. The disease is fatal within 1 year in 90% of cases. We reiterate the need for considering CJD as a differential diagnosis, although rare, in patients presenting with neurologic deterioration with associated visual symptoms, myoclonus and mental status changes.

**Author: ROSHNI Radhakrishna, MBBS**

Additional Authors: HAYAS HASEER KOYA MD, DONA VARGHESE MD, GUSTAVO DE LA ROZA MD, BRIAN CHANGLAI MD  
Institution: SUNY UPSTATE MEDICAL UNIVERSITY

**Title: YOUR EYES WILL NOT SEE WHAT YOUR MIND DOES NOT KNOW- UNRAVELLING THE MYSTERY OF A RARE SKIN LESION**

Differential diagnosis of skin lesions is vast and ranges from nutritional deficiencies to infectious causes. We present a curious case of skin lesions in an immigrant patient. A 56 year old male who migrated to the United States from Nepal presented with diffuse papular skin lesions of 10 months duration. He had no associated constitutional symptoms or history of medication allergies and was hemodynamically stable. Pertinent findings on physical examination included multiple diffuse pruritic lesions- papular pigmented flesh colored with surrounding erythematous raised lesions (figures 1-2) , spread across his face, extremities, chest and abdomen with associated tingling and numbness of these regions. A detailed initial serologic work up for infectious and vasculitic etiologies was negative. A skin biopsy was subsequently done which revealed chronic inflammatory infiltrates composed of lymphocytes and histiocytes with innumerable acid fast bacilli on fite stain within histiocytes and cutaneous nerves (figures 3-4) thus establishing a diagnosis of lepromatous leprosy. He was started on multidrug therapy (MDT) with rifampin, minocycline and dapson and discharged home eventually with an Infectious Disease follow up.

Leprosy is a chronic, slowly progressing infectious disease caused by Mycobacterium leprae mostly prevalent in developing countries of the world. It mainly involves peripheral nerves and skin with varied presentation based on a patient's immune response. Spectrum of disease ranges from tuberculoid to lepromatous leprosy (LL). Lepromatous leprosy presents in patients with no immunologic resistance to the bacilli, hence with diffuse lesions and high disease burden. Disease transmission is thought to occur through respiratory route via close contact. Diagnosis is established by skin biopsy and special staining which shows large numbers of Mycobacteriae in LL. Differential diagnosis includes non-tuberculous mycobacterial lesions, SLE, cutaneous Leishmaniasis, annular psoriasis or keloid. Treatment is by using MDT with rifampin, dapson, clofazimine or minocycline. If left untreated leprosy is debilitating and causes long term sequelae including neuropathies and deformities. In the face of a changing demographic in United States, diseases like leprosy are being increasingly reported since this nation has become the hub for influx of immigrants from different parts of the world especially the developing countries where leprosy is most prevalent. We reiterate the need for timely recognition of tropical diseases like leprosy, as delay in diagnosis and treatment can cause transmission of disease and permanent deformities/neuropathies.

## Resident/ Fellow Clinical Vignette

**Author: Shiv Raj, M.D.**

Additional Authors: Edward Chapnick, MD, FACP; Monica Ghitan, MD; Alexa Sasson, MD; Maxine Seales, MD; Lawrence Wolf, MD, FACP.

Institution: Maimonides Medical Center

**Title: PULMONARY MUCORMYCOSIS IN AN IMMUNOCOMPETENT HOST**

Introduction:

Pulmonary fungal infections may exhibit a broad spectrum of clinical manifestations and severity. Invasive aspergillosis and mucormycosis can be extensive and severe. These infections are generally categorized by their clinical presentation and site of anatomic involvement. We describe a patient who developed a pulmonary fungal infection in an area of lung parenchyma suspected to contain a Congenital Pulmonary Airway Malformation (CPAM), formerly known as Congenital Cystic Adenomatoid Malformation (CCAM).

Case Presentation:

A 48-year-old woman presented to the hospital with a 2-day history of shortness of breath and productive cough with blood-tinged sputum. Past medical history was significant for hypertension, diabetes mellitus, ventricular septal defect (VSD) repaired in childhood, and severe scoliosis. One year prior, she developed dyspnea and hemoptysis, and was treated for presumed culture negative tuberculosis with a 6-month course of rifampin, isoniazid, pyrazinamide, and ethambutol. The patient returned to the initial hospital for her recurring symptoms and underwent a broncho-alveolar lavage. Culture revealed aspergillus fumigatus. Voriconazole and prednisone were given for 6-months. Two weeks later, the symptoms recurred. Computed tomography (CT) of the chest showed a large thick-walled cavity in the right upper lobe accompanied by consolidation with air bronchograms in the anterior segment of the right upper and middle lobes, worse in comparison from prior imaging. A CT-guided needle biopsy was performed, with histopathologic examination revealing wide, thick-walled aseptate hyphae with branching at right angles, consistent with mucormycosis.

Discussion:

Treatment with voriconazole for presumed pulmonary aspergillosis may have predisposed the patient to the development of mucormycosis, as this has been reported in hematopoietic stem cell transplant recipients receiving voriconazole prophylaxis. Pulmonary mucormycosis has a very poor prognosis once there is spread to distant sites. Current guidelines recommend a combination of liposomal amphotericin B, along with surgical resection of the necrotic lung tissue.

In our patient, the infection predominantly involved a cystic area in the right upper lobe. The history of VSD and scoliosis, along with recurrent pulmonary infections within a walled cavity within the lung, suggested a diagnosis of CPAM. Surgery is recommended in older patients with CPAM suffering from recurrent infections. Our patient is a poor surgical candidate due to her refusal to accept blood transfusions, often needed in this a high risk procedure, and is currently being treated with liposomal amphotericin B and posaconazole.

Conclusion:

This case illustrates a rare presentation of pulmonary mucormycosis isolated to a focal region suspected to represent CPAM. Confirmation of the pathogen was essential in order to institute the appropriate therapy. Ideally, a multidisciplinary approach including medical and surgical intervention is warranted. Managing patients with mucormycosis is challenging, requiring a prompt diagnosis and aggressive treatment strategy as the cornerstone of therapy.

**Author: Poornima Ramadas, MBBS**

Additional Authors: Viveksandeep Thoguluva Chandrasekar, MBBS-SUNY Upstate Medical University, Syracuse, NY, Prathik Krishnan, MBBS, SUNY Upstate Medical University, Syracuse, NY, Simrit Walia, MD, SUNY Upstate Medical University, Syracuse  
Institution: Suny Upstate Medical University

**Title: SORE THROAT & SORE JOINT: JOINT TAP MAKES THE CONNECTION**

Introduction:

Streptococcus is an uncommon organism to cause arthritis in adults and presentation as polyarticular septic arthritis is highly unlikely in a previously healthy adult. Bacterial arthritis is often destructive to the joint and requires prompt diagnosis and intervention to prevent joint damage. This case describes a previously healthy female who presented with polyarticular septic arthritis with streptococcus.

Case Description:

57 year old female with no significant medical history, who presented with fatigue, fever with chills, along with swelling and redness of the left ankle, bilateral elbows, proximal and distal interphalangeal joints of hands. She reported a recent episode of upper respiratory tract infection with sore throat and rhinitis. Labs revealed a leukocyte count of  $10.9 \times 10^3/\mu\text{L}$  with neutrophilic predominance, platelets of  $96 \times 10^3/\mu\text{L}$ , mild transaminitis and hyperbilirubinemia. Urinalysis showed evidence of UTI. Suspicion of septic arthritis was low given the presentation of polyarthritis, immunological work up was sent and Rheumatology consult was done. ESR and CRP were elevated. Autoantibody panel including ANA was negative. Lyme, Parvovirus B19, Epstein Barr Virus, Mycoplasma, Ehrlichia titers, hepatitis panel, Gonorrhea & Chlamydia were negative. With the significant effusion in the left ankle, arthrocentesis was done which revealed evidence of septic arthritis. Blood cultures revealed gram positive cocci, further characterized as Group A beta hemolytic streptococci. Cultures from left ankle and urine cultures also revealed the same. She was initially started on intravenous vancomycin, pending final cultures. Orthopedics was consulted and she underwent wash out of left ankle and bilateral elbows. Left ankle and left elbow again grew the same organism in cultures. Culture from right elbow was sterile and effusions in the hands were not significant enough to be drained and improved with antibiotic therapy.

Transesophageal ECHO showed no evidence of endocarditis.

Antibiotic was switched to Ceftriaxone as per sensitivities and infectious diseases consult was done. Repeat blood cultures and urine cultures were negative. Immunoglobulin levels were done with the unusual presentation and showed low IgG, IgA levels and elevated IgM levels. She was later discharged on 6 weeks of intravenous Ceftriaxone with serial ESR and CRP monitoring. She is currently being investigated for possible immunodeficiency disorder.

Discussion:

Polyarthritis in an adult most often prompts investigation of rheumatologic diseases and viral infections. This case illustrates an unusual occurrence of polyarticular septic arthritis in a previously healthy adult and the importance of arthrocentesis for early diagnosis of septic arthritis. Delay in diagnosis can lead to joint destruction and loss of joint function.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Shanchiya Ravindradas, MD</b> Additional Authors: Thushara Paul, MD, Gbolahan Ogunbayo MD, Siddhant Datta, Odunayo, Olorunfemi, Nagesh Jadhav MD Institution: Rochester Regional Health</p> <p><b>Title: Paecilomyces: A rare Fungal Pneumonia in a Patient with Acute Myeloid Leukemia</b></p> <p>Introduction: Paecilomyces variotii is a ubiquitous saprophytic fungus that is found in compost, soils and food that can cause serious infection in immunocompromised individuals. Ocular, cutaneous and subcutaneous infections are most common, and, more rarely pneumonia has been reported. We present a case of Paecilomyces variotii pneumonia in a 68 year old with acute myelomonocytic leukemia.</p> <p>Case Presentation: A 68 year old gentleman with a five year history of myelodysplastic syndrome and chronic lung disease requiring home oxygen presented with three days of hemoptysis and cough productive of green sputum. He was afebrile and denied recent travel or sick contacts. On admission he was tachypneic with bibasilar crackles. Laboratory tests revealed leukocytosis of 30,000/L with &gt;30% blasts. Flow cytometry was consistent with acute myelomonocytic leukemia. He was started on 7+3 Cytarabine and Mitoxantrone. Neutropenia and fever developed and was treated with broad spectrum antibiotics for 10 days. Because of continued fever, CT chest imaging was obtained, revealing right lower lobe ground glass opacities. Sputum cultures grew Paecilomyces and he was started on Voriconazole. Repeat sputum cultures throughout his hospitalizations continued to grow Paecilomyces, which on average took 5-6 days for cultures to be positive. After a month in the hospital, his fever and neutropenia resolved and he was discharged home on Voriconazole therapy. Itraconazole was substituted for Voriconazole based on sensitivities. Repeat CT chest a month after discharge showed resolution of ground glass opacities. Several months later while on Itraconazole, he was admitted with a COPD exacerbation requiring intubation. When he failed to improve, he was made comfort care and expired.</p> <p>Discussion: There have been case reports of Paecilomyces pneumonia in the immunocompromised patients, especially those with hematological malignancies, stem cell, solid organ transplant patients and prolonged neutropenia. It is most likely due to suppression of cellular and humoral immunity with impairment of polymorph nuclear cells activity. In our patient the most likely reasons were prolonged neutropenia and impaired neutrophil function. Immunocompromised patients with respiratory symptoms and radiological changes revealing ground glass opacities or cavitations, suspicion should remain for fungal pneumonia. Cultures may not be revealing within twenty-four to forty-eight hours. Unlike Paecilomyces lilacinus, Paecilomyces variotii is susceptible to most antifungal therapy thus susceptibility testing is important for successful treatment.</p>	<p><b>Author: Hans Reyes, MD</b> Additional Authors: Suhein Galloza MD Sunggeun Lee MD Institution: New York Medical College - Metropolitan Hospital Center</p> <p><b>Title: Acute severe lithium intoxication without apparent lithium overdosing</b></p> <p>Introduction: Lithium is a commonly used medication for bipolar disorder. Levels of lithium in blood has to be monitored closely because lithium has a narrow therapeutic index to cause toxicity. Here, we describe a case of atypical acute lithium toxicity, which was successfully detected and treated.</p> <p>Case: A 53-year-old white female with medical history of bipolar disorder was admitted because of somnolence and slurred speech. She was on lithium 600 mg twice a day in the controlled medicating setting, caregiver denies any chances of lithium overdose. Patient reported diarrhea, denied fever, nausea, or vomiting. On physical exam, normal vital signs, dehydrated oral mucosa, disorientated. The EKG showed prolonged QT interval 500 ms, lithium level was 4.2 mEq/L, (normal values 0.6-1.2 mEq/L) with normal renal function. Urgent hemodialysis with high flux dialyzer was delivered for three hours. Serum lithium level became 1.39, 1.07, and 1.08 during hemodialysis, next morning it rose up to 1.52 mEq/L. After the second hemodialysis, lithium level stabilized to 0.89 mEq/L. Subsequently, mental status of patient improved and prolonged QT interval resolved. Additional work up revealed PTH level of 191 pg/ml, calcium of 10.5 mg/dl and phosphate of 1.9 mg/dl.</p> <p>Discussion: Treatment of choice of severe lithium toxicity is hemodialysis. Lithium is readily dialyzable due to its low molecular weight, poor protein binding and small volume of distribution. Indication for renal replacement therapy is lithium level more than 4 mEq/L or lithium level greater than 2.5mEq/L with renal insufficiency or symptoms. Usually additional hemodialysis is required because rebound increase in lithium levels occurs after the cessation of hemodialysis, as intracellular lithium diffuses into the extracellular space. Thus, checking level 6 hours after hemodialysis is recommended. There are certain conditions which lithium can slowly accumulates to the level which can cause toxicity, such as elderly population due to both a lower GFR and reduced volume of distribution, nephrotoxic drug interactions, or from dehydration. Interesting aspect about this case is also that she presented with lithium toxicity without any overdosing of lithium or suicidal attempt. Most likely dehydration from diarrhea was the precipitating factor for lithium toxicity. Physicians should monitor lithium level more frequently when patient is in vulnerable status to have lithium toxicity. Moreover, chronic lithium ingestion can lead to nephrogenic diabetes insipidus, when lithium accumulates in the principal cells of the collecting duct it can interfere with the ability of ADH to increase water permeability through aquaporin-2 water channels. Increased PTH level associated with hypercalcemia and hypophosphatemia is also an expected side effect of lithium as occurred in our patient. Clinicians should be able to anticipate possible complications from lithium toxicity and do proper work up for the conditions.</p>
---	---

## Resident/ Fellow Clinical Vignette

**Author: Hans Reyes, MD**

Additional Authors: Julie Islam MD

Savi Mushiyeve MD

Institution: New York Medical College - Metropolitan Hospital Center

**Title: Idiopathic large pericarditis presenting as hemorrhagic cardiac tamponade and bilateral pleural effusion**

Introduction:

Common causes of pericarditis can be infectious in etiology, autoimmune, or uremic but for considerable amount it may remain idiopathic. For hemorrhagic pericardial effusion, the culprit is limited to the few that also include myocardial infarction, trauma, malignancy or aortic dissection which can lead to cardiac tamponade, hemodynamic instability and compromise. Few cases thus far have been reported of pericarditis presenting as hemorrhagic cardiac tamponade. We are presenting a case that is also associated with bilateral pleural effusion.

Case:

A 31-year-old female immigrant from Mali presented to our hospital complaining of diffuse abdominal pain and one episode of vomiting. She had medical history of hypertension with poor medication adherence. Initial vital signs were remarkable for blood pressure of 161/109 mmHg and heart rate of 114 per minute. EKG showed normal sinus rhythm with normal voltage QRS complex. Work up showed no leukocytosis, iron deficiency anemia and hypoalbuminemia. Incidentally, abdominal CT revealed pericardial effusion along with bilateral pleural effusion. Few hours later, the patient developed orthopnea, shortness of breath and persisted to have tachycardia. Echocardiogram showed moderate size pericardial effusion with diastolic collapse of right atrium. IR-guided pericardiocentesis was performed: 150ml of exudative hemorrhagic fluid was drained. Cytology study returned negative for malignant cells. Laboratory work up for Tuberculosis, other infectious and autoimmune etiology was unyielding. Cancer markers: CEA, CA 19-9 was also negative as was HIV. Only erythrocyte sedimentation rate and C-reactive protein were elevated. Video-assisted thoracoscopic surgery and pericardial window were done where repeat pericardial and pleural fluid work-up reflected the previous results, including PCR for Tuberculosis. Biopsy was consistent with chronic pericarditis while pleural biopsy confirmed reactive pleuritis. Patient was started on colchicine 0.6 mg daily, remained asymptomatic and repeat echocardiogram one week later showed small pericardial effusion. Finally, she was discharged after an extensive negative work up for pericardial effusion etiology.

Discussion:

When a patient comes with hemorrhagic pericardial or pleural fluid, it is a challenge to determine the etiology. Our patient had recently emigrated from Mali three years ago, provoking a high suspicion of Tuberculosis. Her initial presentation was atypical, without evidence of Beck's triad on physical exam, suggesting a chronic process; which was identified in both biopsies. Idiopathic pericardial effusion is a diagnosis of exclusion, where pericardiostomy is most appropriate diagnostic test to guide management. Aside from this case's uniquely interesting clinical presentation of pericardial associated with bilateral pleural effusions, the response to colchicine has yet to be described in the literature. All practitioners should consider idiopathic cause when all other possibilities have been exhausted, even for large hemorrhagic effusion.

**Author: Yoandy Rodriguez Fuentes, MD**

Additional Authors: Guido Steegers, Maykelin Perez,

Carlos Lopez, Alexander Castillo, Samir Sarkar.

Institution: Jamaica Hospital Medical Center

**Title: Paradoxical Embolism: A Rare Life Threatening Condition.**

A paradoxical embolism is a rare condition. Three elements of the pathway have to be present for a diagnosis: thrombotic clot, intra-cardiac shunt and peripheral embolism events. Intra-cardiac thrombi or embolus in transit are associated with high mortality. Acutely elevated pulmonary arterial pressure due to pulmonary embolism or Valsalva maneuver allows a patent foramen ovale and promotes right to left migration of an intra-atrial clot. Surgical embolectomy, as in our case, has shown to have better outcomes in overall patient survival.

Our case involves a 53 years old female, with history of hypertension, who came to our Emergency Department after she was found minimally responsive, nonverbal with a right sided hemiparesis. Urgent Computed Tomography (CT) Scan of the head showed left middle cerebral Artery (MCA) ischemia. She was initially diagnosed with acute stroke, but no thrombolytic treatment was given due to the size of the ischemia area. Minutes later, patient started complaining of shortness of breath and an arterial blood gas revealed significant A-a gradient hypoxemia. Patient was intubated and CT scan of the chest was demonstrated bilateral pulmonary emboli (PE). Due to her high risk of bleeding, no thrombolytic agent was used to treat both conditions. Having high suspicion of paradoxical embolism, transthoracic echocardiogram demonstrated a patent foramen ovale (PFO). Right Ventricle was markedly enlarged with a right to left shunt. Deep venous thrombosis study positive for DVT in left lower extremity. Intravenous Cava Filter (IVC) was placed as an urgent procedure. Patient underwent successful surgical embolectomy along with closure of patent foramen ovale. Simultaneous pulmonary and systemic embolization is fairly uncommon: however the mortality of these two events together is extremely high. Initial treatment suggested by American Heart Association is heparinization or thrombolysis to decrease the risk of embolization. Although closure of a PFO using a percutaneous or surgical approach is recommended for patients who have sustained a cerebral embolism, a more aggressive approach is warranted when there is evidence of residual thrombus straddling a PFO, and perhaps even more so when there has been a simultaneous PE. Recognition and prompt treatment of intra-atrial thrombus by surgical intervention may be superior to other modalities in the treatment of this clinical condition.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Mouhamed Amr Sabouni, MD</b> Institution: UHS Wilson medical center</p> <p><b>Title: Automatic software interpretation of 24-hour impedance pH tracing: is it a mature technology?</b></p> <p>Automatic software interpretation of 24-hour impedance pH tracing: is it a mature technology? Background: 24-hour multichannel intraluminal impedance and pH (MII-pH) allows detection of both acid and non-acid reflux (NAR) episodes.? There is a concern in the community about complexity of interpretation of 24-hr MII-pH tracings, the amount of time a gastroenterologist may have to spend analyzing these tracings, as well as the reliability of the automatic analysis provided by the different softwares made available by the manufacturers.? Aim: ? To assess the reliability of 2 different types of 24-hr MII-pH analysis softwares compared to the interpretation provided by an expert ? Methods:? Review of 200 consecutive MII-pH studies on once or twice a day PPI therapy? 65% females, mean age 48.6yrs. ? 100 done using MMS equipment and recent software (MMS version V 8.19h). ? 100 were done using Sandhill equipment and recent software (Bioview analysis version 5.5.4.1).? All studies performed between 09/2009 and 9/2012. ? All tracings were interpreted by the same expert with an experience of having read more than 1500 MII-pH studies. ? For the purpose of this study, a trainee with no experience in interpreting these tracings collected the data from the expert analysis and then reset the tracings to their original status prior to modification by the expert, and applied automatic analysis using the newer versions of the softwares.? Results: ? The graph summarizes results of correlation between the interpretation of the expert and the automatic softwares, giving us the Pearson r for each pair.? Of note for all these data points, we had <math>p &lt; 0.0001</math>.? These results show a very strong correlation between both the expert interpretation and the automatic analysis. ? When looking at whether the overall interpretation resulted in either a normal or an abnormal study, the automatic software and the expert agreed 93% of the time for the Sandhill software and 95% of the time for the MMS software.? ?Conclusion: ? Our study shows a very strong correlation between the interpretation provided by an expert and the automatic software analysis for 24-hr.? However, there is not a total agreement all the time. These softwares are clearly very reliable at this time, but we would still recommend that the interpreting physician looks at the tracings before signing off the report to detect any possible problems such as probe malfunction.?</p>	<p><b>Author: Osman Saleem, MBBS</b> Additional Authors: Dr. Safi U. Khan Peter J. Osmond Dr. Joseph L. Izzo, Jr. Institution: University At Buffalo - Internal Medicine Catholic Health System</p> <p><b>Title: DIFFERENTIAL HEMODYNAMIC EFFECTS OF NEBIVOLOL AND VALSARTAN FROM 24-HOUR AMBULATORY PULSE WAVE ANALYSIS: IMPACT OF BLOOD PRESSURE CONTROL AND HEART RATE:</b></p> <p>Compared to an angiotensin receptor blocker (Valsartan, V), a beta-blocker (Nebivolol, N) or the combination of N+V reduces 24-hour ambulatory cardiac work and BP-heart-rate variability (Izzo, et al., JASH 2015, in press). This pre-specified secondary analysis compared additional hemodynamics effects of these drugs and the impact of BP control on 24-hour ambulatory hemodynamic variables. Subjects with hypertension (SBP &gt;140 or DBP &gt;90, n=26) were studied with a double-blinded, forced-titration, sequence-controlled, crossover design with 3 experimental periods: V 320, N 40, and N+V 320/40 mg daily. After 4 weeks of each drug, ambulatory pulse wave analysis (IEM MobilOGraph) was performed with readings every 20 min for 24 hours to determine heart rate, BP, cardiac output (CO), systemic vascular resistance (SVR), and stroke volume (SV). 24-hour brachial and central BP values were similar with all treatments but N and N+V resulted in lower heart rate and CO (<math>p &lt; 0.001</math> each) and higher SV and SVR (<math>p &lt; 0.002</math>) than V. SVR-CO isobars were similar for each treatment group but were shifted upward in those with uncontrolled hypertension. Heart rate was unrelated to BP but correlated strongly positively with CO and inversely with SV and SVR (<math>p &lt; 0.001</math>). When plotted as a function of heart rate, both SV and SVR were inappropriately high in uncontrolled hypertension (mean diastolic 100 vs 77 mmHg), in a ratio of about 2:1. We conclude that N and V have equivalent BP-lowering effects but different hemodynamic profiles: N lowers heart rate and CO, with compensatory increases in SV and SVR; no vasodilator effects were seen with N. SVR-CO isobars were very similar for the 3 treatments but effective BP control shifted the SVR-CO isobar downward and leftward. Heart rate does not directly affect BP but is a critical determinant of the hemodynamics of hypertension: inappropriately high flow and high resistance.</p>
--	--

## Resident/ Fellow Clinical Vignette

**Author: Mohammed Samannodi, MBBS**

Additional Authors: Danial Molloy, MD  
Shameem Beigh, MBBS  
Brandon Fyock, OMS III, Lake Erie College of  
Osteopathic Medicine  
Nicholas Palladino, MS III, University at Buffalo School  
of Medicine and Biomedical Sciences, Catholic Health  
System, South Buffalo Mercy Hospital  
Institution: Sisters of Charity Hospital/University at  
Buffalo

**Title: Persistent vomiting and headaches, don't forget  
the brain: A Case Report of Glioblastoma Mutliforme**

Background:

Some diseases have an insidious nature, often hiding until late in the disease process. Physicians must cast a wide net when initially evaluating a patient. In this case, the patient presented with non-specific symptoms, mainly nausea and vomiting. After further evaluation, a large brain lesion was discovered.

Glioblastoma Multiforme is an aggressive malignant cerebral neoplasm that often presents late in the disease process with an acute onset of symptoms. A majority of these symptoms are non-specific and are often dismissed as a less severe ailment.

Presentation:

In this case, a 27 year old Caucasian male presents in the Emergency Room with a three week history of occipitocervical headaches and two week history of nausea, vomiting, and abdominal pain. On exam; Vitals within normal limits, positive for horizontal nystagmus, positive Romberg test, loss of fine touch sensation on right side of the back. The patient had previously gone to the Emergency Room one week prior due to his persistent abdominal pain and nausea and vomiting. During that visit he had an unremarkable CT scan of the abdomen and was sent home with antiemetics without full neurological exam. Upon his return to the Emergency Room, a CT scan of the head w/o contrast was done. The CT showed a large amount of edema and a mass that invaded past the midline. To further evaluate the mass and confirm the CT head, an MRI Head with contrast was done. This showed a large mass in the right frontal lobe that invaded past the midline and had a large amount of cerebral edema. A neurosurgeon was consulted and the patient was started on IV dexamethasone to reduce the cerebral edema and levetiracetam for seizure prophylaxis. The patient was transferred to Roswell Cancer Institute for biopsy of the brain mass to confirm the diagnosis and proceed with further treatment options.

Discussion:

In this case, symptoms that physicians see every day progressed into a disease that is very uncommon. We suspect the diagnosis to be Glioblastoma Multifforme because the tumor was diffuse in nature, invaded across the cerebral midline, had a central area of necrosis and had a short onset of symptoms. Biopsy was done, confirmed Glioblastoma multifforme. Our advice for all physicians to do a complete physical exam for every patient

Conclusion:

A diagnosis of Glioblastoma Mutliforme is likely due to tumor invasion past the midline, a visible central area of necrosis, and the diffuse nature of the tumor. Prognosis for this patient is grim, with an average life expectancy post diagnosis of twelve months. Current chemotherapy/radiation treatments provide a modest added life expectancy of 2-3 months.

**Author: Uthpala Darshani Samaranayaka Munasinghe, M.B.B.S**

Additional Authors: Bijal Patel, M.B.B.S., Irina C. Delgado Varela, M.D., Harsh Patel, D.G.O, Maria T. Jose, M.B.B.S., Karen Beekman, M.D. Flushing Hospital Medical Center, Flushing, NY  
Institution: flushing hospital medical center

**Title: METASTATIC LIVER ABSCESS FROM PREVOTELLA (B.) LOESCHEII, ANAEROCOCCUS PREVOTII AND KLEBSIELLA PNEUMONIAE**

Pyogenic liver abscess with septic lung embolization in the absence of bacteremia is not a common phenomenon. Although Klebsiella Pneumoniae is commonly associated with liver abscesses; Prevotella (B.) loescheii and Anaerococcus prevotii are extremely rare.

A 55 year old Chinese female known hepatitis B carrier presented to the emergency department with fever, chills, night sweats and myalgia for 10 days. History was negative for sick contacts, recent travel, oral or dental infection, recent surgery and illicit drug use. Laboratory evaluation showed leukocytosis and mildly elevated alkaline phosphatase. Chest X-Ray demonstrated bilateral pulmonary nodules. CT scan of chest with contrast showed multiple bilateral pulmonary nodules (largest 15mm) bilateral pleural effusions and large complex mass in the liver involving the dome and anterior segment of right lobe. Alpha fetoprotein, serology for entamoeba, and HIV test was negative. On CT abdomen with and without contrast there was a large complex mass 9 x 7.7 x 6.7 cm in the right anterior segment and left medial segment of the liver with multiple thick enhancing internal septations. Intravenous ceftriaxone and metronidazole was begun. Ten ml of cloudy liquid was aspirated under CT guidance. Cytology and AFB was negative. Aspirate culture grew Klebsiella Pneumoniae, Anaerococcus Prevotii, and Prevotella (B.) loescheii. Blood cultures were negative. Quantiferon was positive and AFB smear of sputum was negative in 3 consecutive samples. Repeat chest CT scan after 1 week and ultrasound of liver after 2 weeks showed improvement in lung nodules and decreasing size of liver abscess. Patient was treated with 3 weeks of IV antibiotics and 3 weeks of oral antibiotics. On completion of antibiotic treatment repeat CT scan of abdomen and pelvis showed marked reduction in size of abscess.

Our patient had a primary liver abscess with Klebsiella Pneumoniae, Prevotella (B.) loescheii, and Anaerococcus prevotii. Pulmonary nodules were likely due to septic emboli as reduction in size was noted with antibiotic treatment. No periodontal, gastrointestinal or biliary tract infection was detected which might be a nidus for a liver abscess. In the absence of bacteremia, septic emboli to lungs from primary liver abscess are very rare. Very limited data available on Prevotella (B.) loescheii, Anaerococcus Prevotii which can potentially cause a metastatic liver abscess as shown in this case report. These organisms can be treated with metronidazole as shown in our case. Further studies need to be done to better understand characteristics, pathogenicity and virulence factors for Prevotella (B.) loescheii, Anaerococcus Prevotii as possible emerging pathogens.

## Resident/ Fellow Clinical Vignette

**Author: Yazan Samhour, MD**

Additional Authors: Ming Zhao MD

Mahesh Krishnamurthy MD

Institution: Rochester Regional Health System-Unity Hospital

### **Title: A BIZARRE CASE OF WATER INDUCED BLEEDING**

#### INTRODUCTION

Drug-induced immune thrombocytopenia (DIIT) is a relatively uncommon adverse reaction caused by drug-dependent antibodies (DDAbs). We are reporting an unusual case of immune thrombocytopenia caused by consumption of tonic water for leg cramps.

#### CASE DESCRIPTION

A 69-year-old man with medical history of Diabetes, Hypertension and varicose veins, presented to emergency room with hemoptysis, easy bruising and ecchymosis for one week. His daily medications included Amlodipine, Losartan, Insulin Lispro and Glargine. Patient denied any recent history of viral illness. On examination, he had diffuse ecchymosis on his abdomen, lower back and left thigh. Laboratory tests showed platelet count of 1000/ $\times 10^9$ /L, WBC 9700/ $\times 10^9$ /L and hemoglobin of 13.6 gm/dl with normal chemistries. HCV antibody and HIV screening were negative. Peripheral blood smear showed sparse platelets with normal WBCs and RBCs. The etiology of his thrombocytopenia was unclear on admission. However, upon further questioning, patient admitted that he had been drinking tonic water containing quinine, intermittently for the past 10 years for lower extremity cramps. He increased intake to 500 ml daily over the past 2 weeks. Patient was diagnosed as quinine induced thrombocytopenia, treated with Methylprednisolone 125 mg twice daily given his severe thrombocytopenia. He was given intravenous immune globulin (IVIG) on day 3 for persistent thrombocytopenia (less than 1000/ $\times 10^9$ /L). Patient was discharged on day 7 with Platelet count of 72,000/ $\times 10^9$ /L and no evidence of bleeding.

#### CASE DISCUSSION

The development of DIIT is a well-recognized side effect of many drugs. Quinine is a classic example of drugs that cause severe immune thrombocytopenia. Tonic water is popularly used for leg cramps. Quinine stimulates IgG antibodies causing destruction of platelets only when the drug is present. The diagnosis is made by documenting prompt resolution of thrombocytopenia after discontinuation of the suspected drug and excluding other causes. Bone marrow biopsy can be used to exclude blood dyscrasias if suspected. Laboratory testing for DDAbs is not required to make the diagnosis. Testing is technically demanding and not widely available, hence, not useful in the immediate care of a patient. Most patients with DIIT require no specific treatment, as their platelet counts will recover promptly following withdrawal of the offending agent. However, treatment in addition to drug withdrawal may be required when thrombocytopenia is severe or bleeding is present. Corticosteroids, IVIG and plasma exchange have been used successfully to improve platelet count, as in the present case, although the benefit of these treatments is uncertain.

#### CONCLUSION

DIIT should be suspected in any patient who presents with acute thrombocytopenia of unknown cause. A detailed, careful history of drug exposure is essential.

**Author: Harish Shanthanu Seethapathy, MD**

Additional Authors: Farrukh Abbas MD, Department of Internal Medicine, Rochester General Hospital, Rochester, NY

Mysore Seetharaman MD, Department of Internal Medicine, Rochester General Hospital, Rochester, NY  
Institution: Department of Internal Medicine, Rochester General Hospital

### **Title: SCURVY: A LESSON FROM HISTORY**

#### Introduction:

Scurvy, a disease of Vitamin C deficiency is presumed to be rare in developed countries. But, about 7% of the healthy population in the United States has been found to be Vitamin C deficient. It is not a commonly diagnosed disease in modern clinical medicine and calls for a high degree of clinical suspicion. Alcoholics, smokers, mentally impaired and people from a low socio-economic status are particularly at high risk. Here, we present a homeless alcoholic patient who was diagnosed and treated for scurvy.

#### Case description:

A 45 year old Caucasian homeless man was brought by emergency personnel for lethargy and melena. After fluid resuscitation, he became alert and responsive. He did not complain of abdominal pain or vomiting, and remained vague about his tarry stools. He did not have significant neurological, cardiac or respiratory symptoms. He had no regular health maintenance and admitted to heavy alcohol use and missing most of his meals on a daily basis. He denied using any drugs or medications. On physical examination, he was unkempt and emaciated. Old crusted blood filled the oral cavity and his dentition was poor with severe gingivitis. Multiple petechiae were evident over his lower extremities with perifollicular hemorrhages. Ecchymoses were seen over pressure points. Laboratory data was significant for anemia and low pre-albumin. Platelet count, vitamin B12 and coagulation profile were normal. MRI of the brain showed an acute or subacute infarct involving the right frontal lobe and an old right parietal lobe infarct. Transthoracic echocardiogram was normal. Carotid Doppler demonstrated 80-90% occlusion of the right internal carotid artery. Esophagogastroduodenoscopy showed signs of severe hemorrhagic gastritis. Scurvy was suspected based on the skin changes and Vitamin C level was less than 0.1 mg/dl (0.6-2.0 mg/dl). He was placed on 1000mg Vitamin C daily. Because of his severe carotid atherosclerosis and strokes, he underwent right carotid endarterectomy with patch angioplasty.

#### Discussion:

Contrary to popular opinion, scurvy is not an uncommon disease in the developed world, especially in high risk individuals such as alcoholics. The signs and symptoms of scurvy are not specific and can present quite a diagnostic challenge. Poor dentition, bleeding gums, poor wound healing, skin changes such as petechiae and perifollicular hemorrhages as well as anemia are the common clinical manifestations of scurvy. Atherosclerosis can be induced experimentally with vitamin C deficiency in animals, but no conclusions can be made regarding the contribution of vitamin deficiency to atherosclerosis in our patient. High index of clinical suspicion can help early diagnosis of this condition and avoid unnecessary and potentially harmful investigations.

## Resident/ Fellow Clinical Vignette

**Author: Ki Won Seo, M.D.**

Additional Authors: Sharmila Tilak, M.D.

Roxana Lazarescu, M.D.

Institution: New York Presbyterian Queens

### **Title: "Air"ing on the Side of Caution with Radiographic Findings of Portal Venous Gas**

Introduction: Hepatic portal venous gas (HPVG) is a rare radiographic finding that has been almost exclusively associated with life-threatening conditions; namely, bowel ischemia, which carries a mortality risk as high as 75%.<sup>1</sup> A clinician must maintain high suspicion for ischemic bowel disease in the presence of HPVG.

Case Presentation: 76 year-old Korean female with a past medical history of hypertension, hyperlipidemia, coronary artery disease, gastroesophageal reflux disease, appendicitis status post appendectomy 20 years prior presents with acute generalized abdominal pain described as dull and progressively worsening over the course of one day without relief from over-the-counter medications. Associated symptoms included nausea without any fevers, vomiting, constipation or diarrhea. The patient was afebrile with stable vital signs on presentation. She appeared to be in distress and was noted to have right lower quadrant tenderness with guarding and rigidity. Initial blood tests showed significant leukocytosis and lactic acidosis without transaminitis or lipase elevation. Plain radiograph of the abdomen was negative. CT abdomen and pelvis showed cecal and proximal descending colonic wall thickening suggestive of colitis as well as an air-filled tubular focus within the left hepatic lobe representing portal venous air. General surgery was urgently consulted for concerns of ischemic colitis with peritonitis. Emergent diagnostic laparoscopy was performed with conversion to exploratory laparotomy during which a gangrenous cecal wall was discovered. The patient underwent a partial hemicolectomy without complications. Pathology showed cecum and ascending colon full thickness necrosis, pseudomembrane formation and serositis.

Discussion: Although recent literature has shown association of HPVG with nonfatal conditions including inflammatory bowel disease, bowel obstruction, and certain intra-abdominal malignancies, the most common etiology of HPVG remains intestinal necrosis due to mesenteric thrombosis.<sup>1</sup> The exact mechanism of HPVG is yet to be determined; however, the postulated pathophysiology include microbe-derived air and absorbed intraluminal air as possible explanations. Focus on academic inquiry, however, should not come at the expense of delaying appropriate management in the setting of a positive HPVG finding on CT imaging.<sup>2</sup> For this particular patient who presented with abdominal pain and peritoneal signs on examination in the setting of lactic acidosis, the decision to immediately consult general surgery was appropriately made. In spite of recent literature showing a number of nonfatal associations with a positive HPVG finding, it is imperative that a clinician correlates radiographic results with clinical findings and maintain a high index of suspicion for bowel ischemia.

References:

1. Kevin McElvanna, Alastair Campbell, Tom Diamond. Hepatic portal venous gas – three non-fatal cases and review of the literature. *Ulster Medical Journal*. 2012 May; 81(2): 74-78.
2. Aaron L. Nelson, MD, PhD; et al, "Hepatic Portal Venous Gas: The ABCs of Management" *JAMA Surgery*. 2009 June 15;144(6):575-581. doi:10.1001/archsurg.2009.88.

**Author: Sumaira Shafi, MD**

Additional Authors: Prasanta Basak MD, Stephen

Jesmajian MD

Institution: Montefiore New Rochelle Hospital and

Albert Einstein College of Medicine.

### **Title: TUBERCULOUS LIVER ABSCESS IN AN IMMUNOCOMPETENT PATIENT**

Introduction:

Primary hepatic tuberculosis (TB) is rare, and is usually seen in immunocompromised patients. It can present as miliary TB of the liver with or without involvement of other organs; or as primary liver abscess or nodular lesions. We present a case of primary TB liver abscess in an immunocompetent patient.

Case Report:

A 74 year old female immigrant presented with right upper quadrant abdominal pain, for 10 days. On examination she was a frail lady in no acute distress, with stable vitals. Abdomen was soft, with RUQ tenderness. No lymph nodes or masses were palpable. Lab data revealed Hb 7.5g/ dl, WBC 14.9, and normal platelet count. Serum chemistries were within normal limits. AST and ALT were 29 and 22 respectively, alkaline phosphatase 133, albumin 2.9, total bilirubin 0.5 and INR 1.33. CEA and CA19-9 were within normal limits. CXR was unremarkable. CT scan of abdomen showed focally enhancing lesions in the region of the porta hepatitis. In addition, several large masses in the right lobe of the liver were seen. MRCP showed areas of signal abnormality in the right hepatic lobes, which were partly solid and partly cystic. Repeat CT scan of the abdomen showed multiple areas of heterogeneous fluid collection in the right lobe of the liver. A possible necrotic lymph node was seen at the porta hepatitis. A diagnosis of liver abscess was made and ceftriaxone and metronidazole started. CT guided abscess drainage was done and 5 ml of purulent thick fluid was aspirated. The pus was negative for Gram stain and AFB smears. A single multinucleated giant cell was identified.

After four weeks, repeat CT scan of the abdomen and pelvis showed subtle reduction the size of the lesions. Bacterial and fungal cultures on liver aspirate were negative. AFB cultures detected mycobacterium tuberculosis complex RNA by DNA probe. Quantiferon TB gold was indeterminate. Antibiotics were held and she was started on INH, ethambutol, pyrazinamide and rifampin. The abscesses were considerably smaller on CT scan after 6 weeks of therapy.

Discussion:

With an estimated 8 million new cases per year, TB is an ongoing global challenge. The clinical presentation of hepatic TB is non-specific. Severe liver dysfunction and hyperbilirubinemia are uncommon in primary TB abscess of the liver. Hypoproteinemia with reversed albumin and globulin ratio and disproportional elevation of the serum alkaline phosphatase level are characteristic biochemical features. Positive AFB stain on aspiration and biopsy samples in hepatic TB range from 7 to 59%. Mycobacterial culture, although specific, has a low positive yield of 10%. ELISA and PCR have emerged as useful tools. Anti TB drugs for at least 1 year, with percutaneous drainage is the preferred management for this uncommon condition.

## Resident/ Fellow Clinical Vignette

**Author: Yash Shrivah, MD**

Additional Authors: Roxana Elena Lazarescu, MD

Stanley Josue, MD

Institution: New York Presbyterian Queens

### **Title: Uropathy in Young Asian Women**

Introduction: Ketamine is a powerful anesthetic agent that acts by blocking the NMDA receptor and thereby causing sedative and dissociative effects<sup>1</sup>. With its increase in use, the side effects of ketamine have become more prevalent necessitating a more thorough understanding of its presentation and management by physicians. With its growing use, there is an increasing susceptibility of simultaneous urinary tract symptoms, prompting discussion on a new syndrome appearing with chronic ketamine abuse. Here we present 3 cases depicting the presentation, radiological and laboratory findings of patients who use ketamine.

Case Series Presentation: Three patients were admitted to our hospital between September 2014 and July 2015. They were all young immigrants of Chinese descent that came to the hospital for hematuria and severe lower urinary tract symptoms. Patient's symptoms included epigastric abdominal pain for several days with non-bloody/non-bilious vomiting, urinary frequency/urgency, nausea and subjective fevers. The pain was described as sharp and non-radiating with localization to the epigastric region. Among the three patients, 2 had urinalyses suggestive of a UTI while one only showed gross hematuria. Blood results were significant for transaminitis, and mild AKI. Ultrasound imaging was consistent among the three patients by showing hydronephrosis with no obvious signs of obstruction. CT scan with contrast of the abdomen showed nonspecific thickening of the bladder and ureteral walls with post-inflammatory strictures present. One of the three patients had strictures severe enough to require ureteral stent placement to alleviate the hydronephrosis. Of note, patients were heavy ketamine abusers, using once a day for 2-3 year span prior to presentation.

Discussion: The use of ketamine has led to a dramatic increase in the number of patients seen with ketamine related toxicities. In our hospital alone we have seen 3 in a span of 9 months. Many young patients in the Asian community also do not go to the doctor unless symptoms are severe enough to warrant a visit and therefore this number is likely a gross underestimation of the burden of uropathy secondary to ketamine use. Ketamine can affect both the upper and lower urinary tracts<sup>2,3</sup> as well as the common bile duct which was seen in our small sample size. This constellation of signs and symptoms highly suggest a syndrome which could be appearing with chronic ketamine abuse. The pathophysiology is likely secondary to ketamine's propensity to affect smooth muscle structures<sup>4</sup>. It is becoming increasingly important for physicians to recognize these constellations of symptoms as many patients will deny a history of ketamine abuse and recognition of this new syndrome could potentially save thousands of healthcare dollars in additional testing. In addition, patient safety is an important factor and physicians should be comfortable discussing ketamine and its toxic effects.

**Author: Sabah Siddiqui, MD**

Additional Authors: Dr. Patricia Park

Institution: Maimonides Medical Center

### **Title: Postpartum Lows: Hypoglycemia in Acute Fatty Liver of Pregnancy**

Introduction:

Acute fatty liver of pregnancy (AFLP) is a microvesicular fatty infiltration of hepatocytes that typically occurs in the third trimester. Patients with AFLP can have abnormal liver tests and extrahepatic complications such as increased white blood cell count and decreased platelet count with or without signs of disseminated intravascular coagulation. The main differential diagnosis include Hemolysis, elevated Liver enzymes and Low Platelets (HELLP) syndrome or Thrombotic Thrombocytopenic Purpura (TTP). Hypoglycemia in AFLP is not very well described in the literature. Here we describe a case of AFLP focusing on the hypoglycemia presentation, management and recovery time.

Case: We present the case of a Thirty one-year-old woman in her first pregnancy with twin gestation. She had preterm labor and premature rupture of membranes at thirty four weeks, with malrepresentation of twin A, and underwent successful C-section of two male infants. Postpartum she developed abnormal liver function tests, increased serum creatinine, and hypoglycemia. Despite improving kidney and liver function, she continued to have hypoglycemia requiring dextrose 10% infusion since delivery, and an Endocrine consult was called on postpartum day eight. The differential diagnosis for hypoglycemia at this time included hepatic dysfunction due to fatty liver of pregnancy, adrenal insufficiency, insulinoma, or mesenchymal tumor with excess Insulin-like growth factor (IGF-II) production. She was managed with supportive multidisciplinary care and was weaned off dextrose infusion by day ten postpartum. She had full clinical and biochemical recovery, with results indicating a diagnosis of AFLP.

Discussion:

AFLP is more common in primigravida women with multiple gestation pregnancy and male fetus. Clinically, the prominent organ system derangements are hepatic dysfunction, renal insufficiency, and impaired procoagulant synthesis. Hypoglycemia in this setting is multifactorial and includes depleted hepatic glycogen, decreased glucose output, enhanced peripheral glucose uptake, and impaired gluconeogenesis and insulin degradation. Occurrence of AFLP is also associated with inherited fetal defects in the mitochondrial beta-oxidation of fatty acids. We were unable to obtain genetic testing in this patient and her infants for long chain 3-hydroxyacyl-CoA dehydrogenase deficiency and the most common mutation G1528C.

The duration and severity of hypoglycemia in AFLP has not been well-described. The recovery from hypoglycemia after delivery and the return of normal liver function seems to be dependent on overall disease severity. Authors observed clinical recovery in most women within three to four days postpartum; however, normalization of laboratory studies lagged. Interestingly, our patient had prolonged hypoglycemia greater than one week postpartum despite earlier normalization of liver and renal function tests.

Hypoglycemia in AFLP can be life-threatening, and internists must be vigilant regarding this diagnosis in the postpartum patient. Strict blood glucose monitoring is indicated, and prolonged supportive treatment with 10% or higher dextrose intravenous infusion may be required.

## Resident/ Fellow Clinical Vignette

**Author: Kevin Singh, MD**

Additional Authors: Ahmad Jamal Nadeem DO, Sunny Patel MD

Institution: NYU-Woodhull Medical and Mental Health Center

**Title: A Rare Case of Atypical Hemolytic Uremic Syndrome with Preceding Acute Pancreatitis**

Introduction:

Hemolytic Uremic Syndrome is a triad of hemolytic anemia, renal failure and thrombocytopenia. We describe a case of a 61-year-old female with previous Adenocarcinoma of the Lung and alcohol abuse, who was managed for acute on chronic pancreatitis but was later found to have atypical HUS as a complication.

Case:

A 61-year-old female with a past medical history of Lung Adenocarcinoma with resection and Alcohol Abuse, initially presented with epigastric pain after she was binge drinking approximately 1.5 L of vodka for 6 days due to social stressors. Further questioning on review of systems was negative. Physical examination was only remarkable for tenderness on palpation in the epigastric region and right upper quadrant. Initial labs were significant only for BUN 33 mg/dL and Creatinine 1.9 mg/dL, Hb 12.2 g/dL, AST 112 U/L, ALT 37 U/L with lipase >1200 U/L. The patient was admitted for management of acute on chronic pancreatitis. She was monitored with CIWA protocol for alcohol withdrawal, hydrated with intravenous fluids, kept NPO with diet eventually advanced as tolerated with pain management. On the third day, there was no evidence of alcohol withdrawal or symptoms of acute pancreatitis but an abrupt reduction in the platelet count was noted, from 276k to 76k. Also, there was a rise in serum creatinine to 3.1 mg/dL, LDH to 1,565 U/L and Hb dropping to 9.3 g/dL. Unfractionated heparin for DVT prophylaxis was replaced with intermittent pneumatic compressive devices. However on the following day, thrombocytopenia worsened to 25k. Peripheral smear showed schistocytes. TTP-HUS was suspected, so plasmapheresis was initiated promptly. Assay for ADAMTS13 activity was 55%. She underwent 10 plasmapheresis sessions with serum creatinine improving to 1.6 mg/dL and a reduction in LDH to 241 U/L. She followed up in the Hematology/Oncology clinic and her serum creatinine improved further without evidence of coagulopathy or anemia.

Discussion

Atypical HUS is unrelated to Shiga toxin associated dysentery and accounts for 10% of HUS cases. This form of HUS often recurs and usually follows an aggressive course. Rarely, acute pancreatitis can precipitate atypical HUS. It is thought that inflammatory cytokines from acute pancreatitis including IL-1, IL-6, IL-8 and TNF- $\alpha$  mediate the initiation of HUS in these cases. Without prompt treatment, End Stage Renal Failure is a significant cause of mortality with a rate as high as 25%. Plasmapheresis remains to be the mainstay of treatment and introduces complement proteins that may have been depleted from hemolysis in addition to removing cytokines that promote endothelial damage, preventing further platelet aggregation and hemolysis. Acute pancreatitis can lead to many complications and HUS should be considered in patients with worsening anemia, renal function and thrombocytopenia given the high mortality without timely plasmapheresis.

**Author: Dipekka Soni, MD**

Additional Authors: Harish S Seethapathy MD

Yousuf Kyeso MD

Farhan Imran MD

Institution: Rochester General Hospital

**Title: A Diagnostic Dilemma: Beware the Pac Man of the Immune System.**

Introduction:

Hemophagocytic lymphohistiocytosis (HLH) is a disorder of immune activation that can occur in settings which disturb immune homeostasis. While a rare phenomenon, its clinical implications can be tremendous, with diagnostic delay translating into life-threatening consequences. The following case demonstrates a unique scenario of this immunological catastrophe.

Case:

A 57 year old male with Crohn's disease on Remicade developed agranulocytosis. Initial work up, including a bone marrow biopsy was negative. After treatment with neupogen, low grade fevers developed, lasting three weeks. Physical exam was notable for oral thrush, diffuse expiratory wheeze and lower extremity livedo reticularis. An extremely high ferritin (108,416), pancytopenia (Hb 7.3, WBC 0.8, Plt 24) and abnormal coagulation profile (PT 18.2, INR 1.7, aPTT 67.7, FSP 5-20, Fibrinogen 43) was strongly suggestive of HLH and a confirmatory bone marrow biopsy showed hemophagocytosis with histiocytes containing platelets and red cells. Parvovirus antibody was also detected in bone marrow but peripheral PCR was negative. After treatment with Etoposide, dexamethasone and multiple supportive blood transfusions, his liver function tests slowly improved with a reduction in ferritin and transfusion requirements. Unfortunately, he eventually died from gastrointestinal hemorrhage.

Discussion:

While HLH is predominantly a disease of the pediatric population, it can also arise in any condition which can compromise the immune system. Our patient had Crohn's disease, and was being treated with immunosuppressive agents. It is unclear if Parvovirus was another contributing factor. Similar elevations in inflammatory markers (i.e. high ferritin, coagulopathy) can be seen several conditions, most commonly sepsis, but it is vital to keep HLH in mind, because early, appropriate therapy can mean the difference between life and death.

## Resident/ Fellow Clinical Vignette

**Author: Aung Tun, MBBS**

Additional Authors: Nay Min Tun, MD; Yu Yu Thar, MD;  
Maxim Shulimovich, MD  
Institution: The Brooklyn Hospital Center

**Title: AN ELDERLY MAN WITH UNEXPLAINED LIVER FAILURE AND CONCURRENT PANCYTOPENIA: THE IMPORTANCE OF EARLY DETECTION OF HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS**

**INTRODUCTION:** Hemophagocytic lymphohistiocytosis (HLH) is a rare, life-threatening disorder caused by abnormal and excessive activation of immune system secondary to excessive cytokine dysregulation with subsequent multi-organ failure. It can be primary (familial) or secondary (sporadic). Familial HLH, an autosomal recessive disorder, has an incidence of about 1:50,000 live births and occur in infancy or early childhood with a median survival of less than 2 months for untreated familial HLH. Secondary HLH may be caused by infections such as viral, bacterial, fungal and parasitic infections, rheumatologic disorders, and malignancies. Early detection and prompt initiation of treatment of HLH are important to prevent morbidity and mortality.

**CASE:** An 83-year-old African American man presented with worsening shortness of breath, productive cough with whitish sputum and subjective fever for 2 weeks. He had no sick contacts or recent travel. He had a past medical history of asthma and a remote history of prostate cancer status post brachytherapy. Physical examination was remarkable for temperature of 100&deg;F, conjunctival pallor, scleral icterus, and hepatomegaly of 3cm from the right costal margin. Laboratory studies showed a hemoglobin concentration of 8.6g/dL, white blood cell count  $1.4 \times 10^3/\text{L}$  and platelet count  $73 \times 10^3/\text{L}$ ; the peripheral blood smear examination was unremarkable. Presumed asthma exacerbation responded well to antibiotics, steroids, and bronchodilator, however, the patient was noted to have fever spikes ranging from 101&deg;F to 103&deg;F despite negative septic workup. Subsequent laboratory studies were obtained which revealed increased level of direct bilirubin, significantly elevated transaminases, ferritin of >40,000 &#181;g/L, hypofibrinogenemia, and hypertriglyceridemia. Autoimmune hemolytic anemia was ruled out, and viral assays for HIV, CMV, EBV and hepatitis were not significant. A bone marrow examination, done due to suspected HLH, revealed occasional hemophagocytosis. All these findings point towards a diagnosis of HLH. Subsequently, computed tomography of the chest, abdomen and pelvis was performed which did not reveal malignancy or lymphadenopathy, but it showed splenomegaly of 14.5 cm. Patient is currently being treated with standard HLH-94 protocol with resultant resolution of fever, recovery of cytopenias, and significant reduction of in the levels of ferritin, d-dimer and liver enzymes.

**DISCUSSION:** According to 2004 Revised Diagnostic Guideline from the Histiocyte Society, five out of eight criteria (fever, splenomegaly, cytopenias, hyperferritinemia greater than 500 &#181;g/L, hypertriglyceridemia = 265mg/dL and/or hypofibrinogenemia = 150 mg/dl, hemophagocytosis in the bone marrow, spleen or lymph nodes, low or absent NK-cell activity, and high level of soluble IL-2 receptor) must be present to diagnose HLH unless patient has a molecular diagnosis of HLH. Currently, HLH-94 protocol is accepted as a standard treatment with an estimated 5-year survival of 50%. High index of suspicion and prompt institution of treatment of HLH are crucial in order to increase survival.

**Author: Srija Vangala, MD**

Additional Authors: Umut Gomceli MD ; Masooma Niazi MD ; Zeana Cosmina MD  
Institution: Bronx Lebanon Hospital Center

**Title: Kikuchi- Fujimoto Disease with Exophytic Soft tissue Mass and Erosion of Mandible: A Case Report and Review**

**Abstract**

**Introduction:** Kikuchi-Fujimoto disease or histiocytic necrotizing lymphadenitis is a rare, benign and generally self-limiting condition of unknown etiology, which is often underdiagnosed. This is the first case report of Kikuchi presenting with exophytic soft tissue mass.

**Case description:** We present a case of 34 years old African-American woman with chronic hepatitis B and microcytic anemia who presented with a 2 week history of high-grade fever, nonproductive cough, malaise and right neck swelling. At admission she was noticed to have neutropenia, anemia, abnormal liver function tests and renal function. Cultures, serologies for viral infections and work-up for connective tissue diseases were negative. CT scan revealed cervical lymphadenopathy and erosive mandibular mass displacing mandibular teeth. Excisional biopsy of cervical lymph node revealed patchy areas of necrosis, eosinophilic fibrinoid deposits, aggregates of histiocytes with cellular debris, karyorrhexis and foam cells, suggesting acute necrotizing lymphadenitis, consistent with Kikuchi-Fujimoto lymphadenitis. She was initially started on broad spectrum antibiotics which were later discontinued in light of negative septic work up and diagnosis of Kikuchi-Fujimoto disease (KFD) on biopsy. Patient was given supportive treatment, with improvement of neutropenia, renal functions and reduction in size of neck mass.

**Discussion:** Kikuchi-Fujimoto Disease is a rare disease initially reported in Southeast Asia. In the US 63% of the cases were described in Caucasians with only 5% in Afro-Caribbean. It typically presents in young adults and has no clear sexual predilection. Even though typical findings of KFD like cervical lymphadenopathy and fever are present in our case, exophytic soft tissue mass with mandibular erosion and loose teeth was a unique presentation. Other symptoms like weight loss, nausea, vomiting, sore throat, and night sweats are less common. Definitive diagnosis is by excisional biopsy and histopathology. Treatment is usually symptomatic with analgesics, antipyretics and rest. Clinical course is usually self limited and resolves within one to four months. Corticosteroids appear to improve the patient's condition rapidly in extranodal disease and neurological involvement. Kikuchi-Fujimoto disease may precede Systemic Lupus Erythematosus (SLE) or may occur simultaneously or several weeks to several years after the initial diagnosis of SLE. Therefore follow up is important in these cases. Although it is uncommon in the Western countries, it is important to consider it among differential diagnoses, as its treatment drastically differs from that of lymphoproliferative diseases, infectious diseases, tuberculosis, or SLE. Our case illustrates the importance of early suspicion and prompt tissue sampling to prevent unnecessary investigations and potentially harmful treatments.

## Resident/ Fellow Clinical Vignette

**Author: Kadambari Vijaykumar, MD**

Additional Authors: Laura J. von Doenhoff, Krishna M. Rao

Institution: ROCHESTER GENERAL HOSPITAL

**Title: Heart bleed: Spontaneous Left Atrial Cystic Hematoma**

Background: Left atrial intramural hematomas are uncommon entities, conventionally developing post cardiac surgery or interventional procedures, and less commonly secondary to cardiac amyloidosis or blunt trauma. Cystic or cavitated intracardiac masses are also rare, with predominantly infective or neoplastic etiology. We report a case of spontaneous left atrial intramural hematoma mimicking an echinococcal hydatid cyst.

Case: A 62 year old woman, an avid traveler with no prior cardiac history, was admitted with new-onset pleuritic chest discomfort, difficulty swallowing, and fatigue. She denied fever, chills, palpitations, rash, nasal congestion, headache, cough, abnormal bladder or bowel movements. She denied recent exposure to sick contacts or new pets. Her past history was significant only for hypothyroidism. Physical exam was unremarkable except for tachycardia.

Electrocardiogram showed sinus rhythm with intermittent junctional tachycardia. Chest CT revealed a 7 cm mass along the posterior wall of the left atrium. Transthoracic and transesophageal echocardiography showed a septate cystic left atrial mass. Ventricular function was normal. Coronary arteries were normal on angiography. Cardiac magnetic resonance imaging confirmed a large (4.2 x 7.0 x 4.8 cm), smoothly encapsulated mass in the left atrium, not extending beyond the left atrial wall. Differential diagnoses at this point were hydatid cyst, left atrial myxoma, teratoma, or sarcoma. She underwent open heart surgery for removal of the mass. Tissue culture was negative for leucocytes or organisms and echinococcal IgG antibody was negative. On histopathological examination, the mass consisted of fibrin, thrombus, and reactive fibroblastic proliferation which confirmed the diagnosis of an intramural hematoma with cystic degeneration.

Conclusion: Intramural hematomas can mimic cystic or lobular masses or tumors. Spontaneous atrial intramural hematomas are extremely rare, but need to be considered in the differential diagnosis of left atrial masses.

**Author: Deepa Vincent, MBBS**

Additional Authors: Member; Radhika Voleti MD, Member; Shravan Likki MBBS; and Regina Tarkovsky MD

Institution: MAIMONIDES MEDICAL CENTER

**Title: DISSEMINATED GONOCOCCAL INFECTION PRESENTING AS STOMATITIS AND GLOSSITIS**

Introduction

Disseminated gonococcal infection (DGI) is an uncommon complication of gonorrhea caused by the hematogenous spread of the bacterium *N. gonorrhoeae*. The clinical presentation of DGI is diverse, but generally manifests as one of two forms. The first form is a triad of skin lesions, arthralgias and tenosynovitis. The second form is purulent arthritis without skin involvement. Oropharyngeal lesions may also be seen, with the pharynx being the most commonly affected site. Involvement of the oral cavity is very rarely reported in literature. We describe an uncommon presentation of stomatitis and glossitis in a patient with classic manifestations of DGI.

Case Presentation

A 25-year-old female with history of asthma presented to our emergency department with complaints of fever, rash, and soreness in her mouth. She initially developed a pustular lesion on her right thigh ten days prior to admission that spread to her arms and chest. She subsequently developed pain in her throat, which progressed to painful ulcers on her tongue and lips. She developed fever and right eye redness on the day prior to admission. Physical exam revealed healing pustular skin lesions about 0.5cm in diameter the right thigh, arms and dorsal surface of feet. Conjunctival injection and purulent discharge of the right eye were noted. Examination of oral cavity revealed numerous ulcerations over the tongue, buccal mucosa and lips. The tongue was inflamed and covered with a white membrane that could not be scraped off with a tongue depressor. No vaginal discharge or genital lesions were noted. The patient stated that she was in a monogamous relationship with a male sexual partner for the past year. Throat culture grew normal pharyngeal flora. Nucleic acid amplification test of the urine was positive for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. The patient was treated for DGI with intravenous ceftriaxone and concurrent chlamydia infection with doxycycline. She developed polyarthralgia and tenosynovitis during her hospital stay that responded to indomethacin. The oral and skin lesions resolved after initiating antibiotic treatment.

Discussion

DGI may be difficult to identify because of its rarity, variability in presentation, and the common absence of urogenital symptoms. Disseminated infection is a complication that is only seen in 0.4-3% of individuals with gonorrhea. Although this condition generally occurs in two forms, a wide variety of presentations have been reported, including abscesses, perihepatitis, endocarditis and meningitis. DGI is a serious complication of gonorrhea that is easily treatable once identified. We suggest that a high index of suspicion for DGI should be maintained in sexually active individuals with systemic symptoms and oral lesions.

<p><b>Author: Radhika Voleti, MD</b>                  Additional Authors: Deepa Vincent MD; Arun Kumar Arumugam Raajasekar, MD; and Salwa Gerges MD, Maimonides Medical Center, Brooklyn NY.                  Institution: Maimonides Medical Center</p> <p><b>Title: NEUROFIBROMATOSIS ASSOCIATED WITH GIST, CARCINOID TUMOR AND PRIMARY HYPERPARATHYROIDISM</b></p> <p>Background                  Neurofibromatosis type 1 (NF-1) is an autosomal dominant genetic disorder that is characterized by café-au-lait spots, lisch nodules, musculoskeletal abnormalities, and neurofibromas. Individuals with neurofibromatosis are at increased risk of benign and malignant neoplasms unrelated to neurofibromas. Gastrointestinal stromal tumors are known to occur at greater frequency in those with NF-1, with the majority of them occurring in the small intestine. Neuroendocrine tumors are also associated with NF-1, with predilection for the periampullary region. There are several documented cases of primary hyperparathyroidism in patients with NF-1. This case report describes the concomitant occurrence of gastrointestinal stromal tumor, ampullary carcinoid tumor and primary hyperparathyroidism in a patient with NF-1.</p> <p>Case Presentation                  A 46-year-old female with history of neurofibromatosis type 1 was admitted for nausea and cramping postprandial abdominal pain that had worsened over the course of several months. Computed tomography scan revealed multiple masses in the abdomen involving the duodenum and ampulla of Vater. She underwent a pylorus-sparing pancreaticoduodenectomy (Whipple procedure). Histopathologic examination demonstrated a low-grade carcinoid tumor in the Ampulla of Vater and gastrointestinal stromal tumor in the duodenum. In addition, multiple neurofibromas were found in the gallbladder. The patient's postoperative course was complicated by severe nausea and vomiting requiring readmissions. Her symptoms eventually resolved after administering promotility agents. She was subsequently admitted 4 months after her surgery for confusion. Physical exam revealed dry oral mucosa and cutaneous neurofibromas on the arms, back and legs. She was alert and awake but was oriented only to place and person. The remainder of neurologic exam was normal. Psychiatric exam was significant for hallucinations and delusions. CT scan of the head was negative for acute intracranial pathology. Laboratory studies were significant for hypercalcemia, low 25-hydroxyvitamin D, normal 24-hour urine calcium, normal parathyroid hormone related-protein and high-normal parathyroid hormone level. The laboratory findings are consistent with primary hyperparathyroidism. She is scheduled to undergo parathyroidectomy as an outpatient.</p> <p>Discussion                  The incidence of GIST, neuroendocrine tumors and primary hyperparathyroidism in patients with neurofibromatosis type 1 is well documented. However, to our knowledge, the coexistence of all three conditions in a single patient has not previously been reported. Individuals with NF-1 have a significantly higher risk than the general population of developing GIST and neuroendocrine tumors. The incidence of GIST in individuals with NF-1 is estimated to be 5-25%. Abdominal imaging should be strongly considered in NF-1 patients with persistent GI symptoms, even if mild. It has been suggested to screen all NF-1 patients for hypercalcemia, as early detection can prevent the long-term sequelae of hyperparathyroidism.</p>	<p><b>Author: Radhika Voleti, MD</b>                  Additional Authors: Oscar B. Lahoud, MD; Abhinav Chandra MD; William B. Solomon, MD, Maimonides Medical Center, Brooklyn, NY                  Institution: Maimonides Medical Center</p> <p><b>Title: SECONDARY HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN A PATIENT WITH SYNCHRONOUS COLON AND BREAST CANCERS</b></p> <p>Introduction                  Hemophagocytic lymphohistiocytosis (HLH) is a rare disorder of immune system hyper activation resulting in severe systemic inflammation. This potentially fatal condition is characterized by inappropriate activation of normal macrophages and lymphocytes, cytokine storm and multi-organ damage. HLH clinically manifests as fever, rash, hepatosplenomegaly, and neurologic symptoms. Primary HLH is caused by gene mutations and generally presents in childhood. The secondary form is triggered by conditions that activate the immune system, such as infection, immune deficiency, and malignancy. Secondary HLH is most commonly associated with hematologic malignancies, and its occurrence with solid tumors is rare. We describe a case of secondary HLH in a patient with synchronous primary colon and breast cancers.</p> <p>Case Description                  A 59-year-old female with newly diagnosed stage III right breast invasive ductal carcinoma presented to our emergency department with severe abdominal pain and nausea. Computed tomography scan of the abdomen showed an obstructing apple core lesion in the right colon. She underwent right hemicolectomy with diverting ileostomy and was diagnosed with stage I colon cancer. Shortly after discharge, she returned to the hospital for generalized weakness and persistent fevers. Physical exam was significant for tachycardia and dry mucus membranes. She was started on broad-spectrum antibiotics and underwent extensive infectious and rheumatologic workup. No infectious or autoimmune etiology could be identified, and fevers persisted. Laboratory studies were significant for anemia with hemoglobin of 8.2g/dL and hyperferritinemia (&gt;1500ng/ml). Bone marrow biopsy was performed, which revealed hemophagocytosis and increased reticuloendothelial iron. Serum soluble CD25 (Interleukin-2 Receptor) was elevated at 627,389pg/mL, consistent with HLH. She was initiated on treatment with steroids and received neoadjuvant chemotherapy for breast cancer followed by mastectomy. Following treatment, her symptoms resolved and red blood cell count recovered to normal levels.</p> <p>Discussion                  HLH is defined by the presence of at least five out of the following eight findings: prolonged fever, splenomegaly, evidence of hemophagocytosis on bone marrow biopsy, cytopenia involving two or more cell lines, hyperferritinemia, hypertriglyceridemia or hypofibrinogenemia, elevated soluble CD25, and low or absent NK cell function. HLH secondary to solid tumors is rare. To our knowledge, this is the first documented case of HLH associated with two synchronous primary solid malignancies. HLH can be challenging to diagnose because the disorder is rarely seen and the symptoms are nonspecific. HLH has a high mortality rate; therefore, treatment should be promptly initiated when clinical suspicion is high. In our patient, the HLH syndrome resolved after treatment with steroids in conjunction with surgery and chemotherapy directed at her colon and breast cancers.</p>
---	---

<p><b>Author: Alexandra Walczyszyn, MD</b>                  Additional Authors: Craig Grossman, MD. Steven Mandel, MD. Nicky Bhatia, MD.                  Institution: Lenox Hill Hospital</p> <p><b>Title: AN UNUSUAL CASE OF DESCENDING PARALYSIS CAUSED BY WEST NILE VIRUS</b></p> <p>West Nile Virus (WNV) is a mosquito-borne flavivirus first identified in Africa, now endemic to the US. WNV infection is usually asymptomatic, but may present with West Nile Fever, which is indistinguishable from other viral syndromes and mostly self-limiting. However, approximately 1% of those infected with WNV develop serious neurologic manifestations, including meningitis, encephalitis and acute flaccid paralysis. We present a case of acute descending flaccid paralysis caused by WNV, underscoring the importance of keeping neuroinvasive WNV on the differential when working up acute flaccid paralysis. A 51 year old female presented to the ER with fever, dizziness, blurred vision, and headaches. She was febrile on admission, but the remainder of her exam was unremarkable. Three days after admission, the patient developed fevers to 106.1F and tachycardia. Search for infectious etiology was negative, and fever of unknown origin work-up was begun. There was no suggestion of any recent infection, though diagnostic studies were positive for H. pylori, and she recalled having sustained a bug bite several weeks prior. Also on hospital day three, she developed dysarthria. MRI brain showed suspicion for cerebellar stroke, but no other abnormalities. Supportive care and broad-spectrum antibiotics were initiated. She then developed lethargy and right upper extremity weakness over the following two days, which progressed to bilateral upper extremity weakness, and then quadriplegia. Eventually she became severely lethargic with respiratory distress and hypotension, requiring intubation and ICU transfer. At the nadir of her clinical course, she did not exhibit response to noxious stimuli, and had no eye tracking, off sedation. Initially, GQ1B IgG was borderline positive, suggesting a Guillain-Barre-like syndrome. She was started on IVIG for presumed Guillain-Barre vs Bickerstaff encephalitis, pending results of further serology. The patient underwent three courses of IVIG. Lumbar puncture was preliminarily negative and EEG showed generalized slowing but no epileptiform activity. Repeat MRI revealed abnormal T2 and flair hyperintensity in the right thalamus, which was enlarged. There was also a smaller patchy area of T2 and flair hyperintensity in the left thalamus, and hyperintensities in the bilateral basal ganglia, thalami, substantia nigra/cortical spinal tracts, pons and medulla. EMG studies were consistent with a severe, acute, symmetric motor axonopathy affecting all extremities. Eventually, serum and CSF antibodies came back positive for WNV. The patient made gradual improvements in motor functioning, allowing for discharge to a rehabilitation facility after a 64-day hospital course. Currently, she is able to speak and walk with assistance. She continues to progress with intensive physical therapy. This case illustrates the importance of having WNV on the differential when working up a patient with neurologic symptoms, including acute flaccid paralysis. Supportive therapy and IVIG may minimize the neurologic sequelae and start patients on their road to recovery.</p>	<p><b>Author: Mike Wei, BS</b>                  Additional Authors: Scott Ely, David Posnett                  Institution: Weill Cornell Medical College</p> <p><b>Title: Spontaneous Regression in Angiosarcoma: Possible Abscopal Effect</b></p> <p>The authors report a case of spontaneous regression of epithelioid angiosarcoma in a 76-year-old Asian male, initially identified upon amputation of 2 necrosing toes after a suitcase dropped on his left foot. A second prophylactic below-the-knee amputation was performed. Nine months later following injury from a poor-fitting prosthesis, the patient had recurrence of angiosarcoma at the stump. In spite of local radiotherapy to the stump, the masses enlarged with ulceration and exposure of the underlying bone, followed by pathologic fracture of the femur treated with an above-the-knee amputation. Residual thigh masses and subcutaneous embolic tumors were documented by MRI (Figure 1A/1B) and pathology. The patient requested comfort care and took Ganoderma lucidum spores daily. Over 6 months there was clinical and radiologic improvement of bone metastases in the femur and deep thigh tissues to complete clinical remission (Figure 1C/1D) followed by recurrence at 45 months following the injury, with progressive lymphedema and death 4 months later. This case report illustrates two poorly understood phenomena. First, the onset of this cancer occurred in the setting of tissue injury and regenerative wound healing with angiogenesis apparently gone awry. Second, the malignancy underwent a 2 year long spontaneous regression, possibly due to an abscopal response (anti-tumor immune response) suggested by an immune infiltrate of the tumor (Figure 2, 3). Full exome sequencing of the tumor obtained from several tissue samples over time, and compared to normal patient saliva RNA, is presented. This case represents 1 of 8 published cases of spontaneous regression of angiosarcoma. These cases are reviewed herein (Table 1).</p>
--	--

## Resident/ Fellow Clinical Vignette

<p><b>Author: De-Ann Williams, M.B.B.S</b> Additional Authors: Andrew Chang MD Institution: SUNY Downstate Medical Center</p> <p><b>Title: NOT YOUR GARDEN VARIETY DRUG REACTION : A CASE REPORT</b></p> <p>Leukocytoclastic vasculitis (LCV) is a small vessel vasculitis with multiple etiologies. One such could be an adverse reaction to a drug and the attendant immune complex reaction. A careful history will sometimes reveal this to be the case and we highlight this with the following case. A 65 year old man presented with a one day history of pruritic red rash that started on the feet and then progressed to his trunk and upper extremities. He denied fever, joint, urinary and gastrointestinal complaints. Examination of the skin revealed a petechial and occasionally palpable purpuric rash in the distribution of his lower and upper extremities, palms, soles and flank. There was no lymphadenopathy or hepatosplenomegaly. The oropharynx, conjunctiva and gingivae were unremarkable. Laboratory data showed a normal white blood cell and platelet count. Chemistry findings were chronic and consistent with his past history of stage 3 chronic kidney disease. Urinalysis showed 5-10 white blood cells per high power field with no red blood cells, protein, granular or hyaline casts. Given the morphology of the rash, a diagnosis of vasculitis was entertained and confirmed by a skin biopsy which showed early leukocytoclastic vasculitis. As part of the workup to identify the etiology, investigations were pursued to isolate a possible infectious agent; studies obtained to this end included an Anti-Streptolysin O (ASO), Anti-DNAse, hepatitis A, hepatitis C, human immunodeficiency virus and rapid plasma reagin (RPR) which were negative. He had hepatitis B immunity. On reviewing the drug history it was noted that he had completed a course of levofloxacin eight days ago for an acute bacterial sinusitis. Given the possibility of an adverse drug reaction as the etiology he was again interviewed and had a Naranjo score of 5 making it a probable cause. He was treated conservatively in the absence of systemic involvement and there was gradual regression of the rash. Fluoroquinolones were subsequently added to his list of drug allergies.</p> <p>LCV can masquerade as a Type 111 hypersensitivity reaction. It is characterized clinically by palpable purpura because of circulating immune complexes are formed in the post capillary venules. When considering the probability of an adverse drug reaction, the Naranjo Adverse Drug Reaction Probability Scale can be used. The Naranjo scale is a weighted questionnaire. As clinician, it is important to inquire about a patient's past medication exposure, as this may be responsible for the clinical presentation. Early recognition of an adverse drug reaction allows for avoidance of the medication and prevents life-threatening medical problems if re-exposure to the drug were to occur.</p>	<p><b>Author: Ivan Wong, M.D.</b> Additional Authors: Ashwad Afzal, M.D. Mikhail Yakubov, M.D. Berhane Worku, M.D. Iosif Gulkarov, M.D. Institution: New York Methodist Hospital</p> <p><b>Title: Recurrent Pneumothoraces: Making the link to Catamenial Pneumothorax</b></p> <p>Introduction: Catamenial pneumothorax (CP) is a rare cause of recurrent pneumothorax. It is the most common form of thoracic endometriosis syndrome, which includes catamenial hemoptysis, catamenial hemothorax, catamenial hemopneumothorax and endometriosis lung nodules. CP is defined as spontaneous recurrent pneumothorax occurring in women of reproductive age, within 24 hours before or 72 hours after the onset of menstruation. We present a case of a young female with catamenial pneumothorax requiring surgical intervention.</p> <p>Case report: A 44 year old woman with a history of endometriosis on ovulatory suppressant therapy and right-sided spontaneous pneumothorax treated with tube thoracostomy two months prior presented with dyspnea and pleuritic chest pain. Physical exam was remarkable for decreased breath sounds and right hemithorax hyperresonance. Her labs were unremarkable. Chest radiography revealed a right pneumothorax. Her prior pneumothorax occurred within 2-3 days following the onset of menstruation. Prior computed tomography scanning demonstrated no blebs or bullous disease. She underwent video-assisted thoracoscopic surgery (VATS) pleurodesis. Extensive diaphragmatic attenuation was noted such that all that remained of the central tendon was a serosal lining through which the abdominal contents was visible. No obvious fenestrations were noted. Due to these findings, an aggressive approach was taken with a combination of pleurectomy, mechanical and talc pleurodesis. She recovered well post-operatively and discharged on a progestin. Pleural biopsy pathology was negative for endometrial tissue. She underwent elective diagnostic laparoscopy with fulguration of pelvic endometrial tissue three months later. On follow up she feels well and chest radiography demonstrates resolution of CP.</p> <p>Discussion: We describe a case of non-thoracic-endometriosis related CP requiring surgical intervention. Our patient had recurrent spontaneous pneumothoraces occurring within 24 hours before and 72 hours after the onset of menses with the absence of thoracic endometrial tissue. The majority of CP cases involve the right side (87.5-100%). Several hypotheses exist regarding the cause of CP, from alveolar rupture caused by vasoconstriction and bronchospasm due to high prostaglandin F2 levels during menses to retrograde menstruation resulting in subdiaphragmatic endometriosis. The prevalence of this rare entity is unclear. However, in a retrospective study of 156 premenopausal women who were surgically treated for spontaneous pneumothorax, 31.4% could be classified as having catamenial and/or thoracic endometriosis related pneumothoraces with 8.3% of the 156 woman were classified as non-thoracic-endometriosis related CP.</p> <p>Conclusion: CP and TE is still under diagnosed and should be considered in premenopausal women with recurrent spontaneous pneumothoraces. Better knowledge about this disease among internists' may help reduce the delay in diagnosis and prevent its recurrence.</p>
--	--

## Resident/ Fellow Clinical Vignette

**Author: Ivan Wong, M.D.**

Additional Authors: Ashwad Afzal

Aleksandr Korniyenko

Alex Ivanov

Iosif Gulkarov

Institution: New York Methodist Hospital

### **Title: Superior Vena Cava Syndrome from an Invasive Thymoma with transcaval invasion to the right atrium**

Introduction: Superior Vena Cava (SVC) syndrome is a rare presentation of invasive thymomas. Invasive thymomas can be diagnosed incidentally but presentation can vary significantly based on the extent of tumor invasion and associated paraneoplastic syndromes. We present a case of SVC syndrome emanating from a type B1, stage IVa Masaoka invasive thymoma that was invading multiple mediastinal structures including the superior vena cava, right atrium, pericardium, and right upper lung lobe.

Case Report: A 74-year-old female with a 40-pack-year smoking history presented with facial and upper extremity swelling and dyspnea on exertion over the last month. Physical exam was remarkable for mild facial swelling, non-pitting edema of the upper extremities, and distention of the superficial veins of the anterior chest and jugular vein. Labs were unremarkable. An echocardiogram showed moderate right atrial dilation with a mobile mass in the atrial cavity prolapsing through the tricuspid valve, bowing of atrial septum from right to left, moderate tricuspid regurgitation. Cardiovascular Magnetic Resonance (CMR) imaging revealed a 9.9 x 4.3 centimeter heterogeneous mass admixed with thrombus in the anterior mediastinum compressing the SVC and endovenously extending into the right atrium. Anticoagulation with heparin drip was initiated. A CT guided biopsy revealed a tan colored mass with CD 5 lymphocyte predominance, inconspicuous epithelial cells positive for P63 expression and cytokeratin AE1/AE3 that was morphologically consistent with thymoma type B1.

An open thymectomy was performed. A large tan-colored mediastinal mass was found infiltrating the right upper lobe of the lung, pericardium, right phrenic nerve, brachiocephalic vein, right innominate vein, SVC and the right atrium. The mediastinal mass was resected followed by wedge resection of the right upper lobe. The mass was classified as a stage IVa invasive thymoma using the Masaoka classification due to pleural and pericardial invasion. There were no postoperative complications. The swelling of the face and upper extremity improved. She is undergoing outpatient treatment with radiation therapy to reduce risk of recurrence.

Discussion: Thymoma is a rare neoplasm primarily arising within the anterior mediastinum. Invasive thymoma invades surrounding thoracic structures such as the heart, lungs and vessels causing compressive symptoms. Most thymomas with cardiac involvement are limited to the pericardium and very few cases of transcaval extension with intracardiac involvement have been reported. SVC syndrome typically arises from extrinsic compression and less commonly from transcaval thymoma infiltration. Based on studies, the invasiveness of a type B1 thymoma is between 70%-75% and the potential to invade the greater vessels is less than 10%. Relapse in 5 years ranges between 2-10% after complete resection followed by either radiation therapy, chemotherapy or a combination of both.

Conclusion: SVC syndrome from a stage IV B1 thymoma should be treated with surgery, radiotherapy and or chemotherapy.

**Author: Jing Xu, MD**

Additional Authors: Arash Velayati, Ming Liu, Sucharita

Cheedella, Vladimir Gotlieb

Institution: Brookdale University Hospital Medical Center

### **Title: Leiomyosarcoma of the Inferior Vena Cava in HIV-infected Patient: A Case Report and Review of the Literature**

Leiomyosarcoma is the most common primary inferior vena cava (IVC) sarcoma consisting about 10% of retroperitoneal sarcomas. This tumor is very rare and there are only about 300 cases reported worldwide in the English literature so far. Presentation is usually nonspecific and diagnosis is often delayed until tumors reach a large size, which leads to a dismal prognosis. Epidemiologically, leiomyosarcoma were frequently reported in HIV-infected people and organ transplant recipients, supporting an increased risk of this sarcoma in immunosuppressed people. The majority of HIV-infected individuals reported with leiomyosarcoma had AIDS, and CD4 cell counts were typically low. Moreover, it occurs disproportionately with peaks in two age groups, in children aged 0-9 years and young adults aged 30-39 years.

Here we report a very unusual presentation of leiomyosarcoma in a 46 year old HIV-infected female on HARTT with CD4 count of 934 who presented with 10-day history of constant back pain radiating to both lower limbs. Patient was found to have an IVC mass on lumbar spine MRI. Abdomen CT scan showed an enhancing thrombus in the mid inferior vena cava and a non-enhancing thrombus in the distal inferior vena cava. 1st CT-guided IVC mass biopsy was not diagnostic. Patient received IVC filter and was then discharged home while biopsy result was pending. 20 days later, patient returned to ER with acute onset 9/10 sharp non-radiating right flank pain. MRA showed a 3.5 x 2.8 x 4 cm irregularly shaped retroperitoneal enhancing mass between the distal abdominal aorta and IVC which invaded into IVC wall. Repeat biopsy showed spindle cells with smooth muscle features with positive biomarkers of vimentin, desmin and smooth-muscle a-actin. A moderately differentiated leiomyosarcoma was diagnosed. Patient was later on transferred to another institute for surgical resection, chemotherapy and radiation therapy. To date, patient survived more than two years with this very aggressive cancer and is still alive. Based on our knowledge this is the first report of primary IVC leiomyosarcoma in HIV-infected adult population.

In summary, we report a case of IVC leiomyosarcoma in a 45-50 year age group HIV-infected female with relatively high CD4 count. The permissiveness and tumorigenesis associated with immunodeficiency stage still remains unclear. Since leiomyosarcoma of IVC is extremely rare and has dismal prognosis, recognition of its clinical, radiologic, surgical and pathologic findings in routine work may have obvious clinical significance.

## Resident/ Fellow Clinical Vignette

**Author: Abraham Yacoub, MD**

Additional Authors: Regina Frants, M.D., F.C.C.P., Leslie Bank, M.D., F.A.C.G., Jagmohan S. Sidhu, M.D., F.A.C.P., Peter Nicholson, M.D

Institution: United Health Services Wilson Medical Center Internal Medicine Residency Program

**Title: An Unusual Presentation of Esophageal Cancer: A Case Report and Review of Literature.****Introduction**

There are two major types of esophageal cancer: esophageal adenocarcinoma (EAC) and esophageal squamous cell cancer (ESCC). EAC arises from the distal third of the esophagus and is commonly found in Caucasian men. ESCC arises from the proximal two thirds of the esophagus and is commonly found in African Americans and Caucasian females.

**Case Presentation**

A 49-year-old white female with a past medical history of hypothyroidism and generalized anxiety disorder was referred to our gastroenterology clinic by the pulmonologist for an abnormal finding on the CT scan of the chest. The patient was evaluated by her pulmonologist for a chronic cough of one year duration. Despite 3 courses of antibiotics, the cough continued to worsen. She denied any medical history of GERD, esophagitis, or aspiration pneumonitis. She denied any tobacco smoking or second hand smoking exposure. She consumed 2 drinks of alcohol on the weekend. She works as a pharmaceutical representative and was on medical leave due to the severe cough during the nights.

She denied any fever, chills or night sweats. She denied any heartburn, nausea, vomiting, dysphagia, odynophagia, and weight loss. The patient's vital signs were unremarkable.

CT of the chest revealed abnormal thickening of the thoracic esophagus. EGD revealed diffuse multiple masses in the esophagus. Histopathology of the masses revealed adenocarcinoma.

**Discussion**

This case is unique due to a number of atypical features. Firstly, the patient had respiratory symptoms rather than gastroenterology symptoms. The most common symptoms for esophageal cancer are dysphagia, odynophagia, and weight loss. Our patient presented with a chronic cough. Esophageal cancer can present with respiratory symptoms of cough and lung infection. This mechanism is commonly due to the presence of an acquired tracheoesophageal fistula (TEF), which the patient did not have. In the literature, there was a case of an esophageal cancer in a patient presenting with respiratory symptoms rather the chronic, without a history of respiratory disease or acquired TEF.

Secondly, EAC arises from the distal third of the esophagus. The unusual location and distribution of the tumor in our case was very rare. The first friable mass was located at 22 cm from the incisors, which is part of the proximal two-thirds. The lesions were diffuse and extending down 35 cm from the incisors. Thirdly, common risk factors for EAC are Barrett's esophagus caused by chronic GERD, low socioeconomic status, obesity, and male gender. Higher alcohol consumption was not associated with increased risk of EAC. Our patient did not have any risk factors to suspect esophageal cancer.

**Conclusion:** We present a case of an atypical feature of esophageal cancer. It is important for clinicians to keep esophageal cancer in the differential diagnosis in patients presenting with the symptoms previously mentioned.

**Author: Srikanth Yandrapalli, MD**

Additional Authors: Pratik Mondal MD 1, Bella Mehta MD 1, Pallavi Khatkar MD 2, George Kleinman MD 2, Sachin Sule MD 1, Randy Goldberg MD 1.

1 Department of Medicine, New York Medical College, Valhalla, NY. 2 Department of Pathology, New York Medical College, Valhalla, NY.

Institution: Westchester Medical Center - New York Medical College

**Title: PAPILLARY FIBROELASTOMA: THE NEED FOR TIMELY DIAGNOSIS**

Cardiac papillary fibroelastomas (CPF's) represent a rare entity in clinical medicine and literature regarding their management is limited. This is a report of an experience with a case of CPF. A 64 year old man presents with worsening dyspnea and orthopnea. His medical history was significant for congestive heart failure and NSTEMI. Transthoracic Echocardiography (TTE) was suggestive of a reduced bi-ventricular systolic function and a diagnosis of acute decompensated heart failure was made. Prior coronary angiography was significant for non-obstructive coronary artery disease (CAD). A few hours after admission he developed severe hypotension which rapidly progressed to cardiac arrest. NSTEMI was diagnosed based on elevated troponin-I and an unchanged ECG. Autopsy revealed an acute myocardial infarct secondary to papillary giant fibroelastoma of aortic valve completely occluding the right coronary ostium. Coronary atherosclerosis with non-obstructive CAD was evident. This case illustrates a fatal outcome associated with an undiagnosed CPF. CPF's, the most common cardiac valvular tumors, are benign endocardial papillomas predominantly affecting the cardiac valves, with highest prevalence in the 6th to 8th decade of life. Although most were incidental autopsy findings, the past two decades have witnessed reported cases with cardiac and neurological symptoms, including transient ischemic attack, angina, syncope, stroke, myocardial infarction and sudden death. In a large single center review of highly-selected referral population, CPF was more prevalent (0.089% of all echocardiograms) than cardiac myxoma, generally thought to be the most common primary intracardiac tumor based on autopsy studies. The advent of higher-resolution imaging especially transesophageal echocardiography (TEE) facilitated rapid ante-mortem diagnosis. Currently, depending on symptoms and tumor mobility either surgical resection or anticoagulation is offered to patients. Literature suggests that successful complete resection of CPF is curative with an excellent long-term prognosis and a lower stroke risk. Given the potential to cause fatal outcomes and the availability of successful therapies, CPF should be a diagnostic consideration in elderly patients with myocardial infarction with normal coronaries or non-obstructive CAD after other potential causes of myocardial injury and other confounding diagnoses have been ruled out. Diagnostic evaluation begins with a TTE, but it is advisable to further evaluate with TEE when clinical suspicion for CPF is high, as TTE can miss a third of CPF's evident on TEE. Randomized studies are needed to make valid recommendations as most of the available studies are single center experiences.

## Resident/ Fellow Clinical Vignette

<p><b>Author: Jill Yeager, DO</b> Additional Authors: Wajihuddin Syed, MBBS Amit Dhamoon, MD Anup Shah, MSIV Institution: SUNY Upstate Medical University</p> <p><b>Title: LYME'S DISEASE MANIFESTING AS SIADH</b></p> <p>Introduction: The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a disorder caused by the inability to suppress the secretion of ADH, usually caused by CNS disorders, ectopic production or drugs. We report a case of SIADH caused by Lyme's disease.</p> <p>Case: An 86 year old female presented to hospital with altered mental status and left-sided facial droop. The duration of symptoms was unclear, as family had not seen her for one week. Concerned for an acute CVA, CTA head/neck were ordered which did not show any evidence of vascular disease or stroke. She was suspected to have Bell's palsy and a Lyme antibody was sent which returned positive. The patient was also noted to have a sodium level of 120 with serum osmolality of 268. An extensive work up of hyponatremia ruled out heart failure, cirrhosis, renal failure, adrenal insufficiency or hypothyroidism as the cause of hyponatremia. She was not on any medications at home and had no evidence of volume loss (like bleeding, diarrhea or vomiting). Urine electrolytes revealed a urine sodium of 63 and Urine osmolality of 352 which suggested the patient had SIADH. Further work-up revealed no evidence of pulmonary disease or chest malignancy that could cause her SIADH. She was started on steroids for her Bell's palsy and doxycycline for the Lyme's disease. She showed remarkable improvement in her mental status and her sodium corrected with supportive treatment. The rapid response of sodium to treatment of Lyme's disease and no other explanation for the SIADH, makes Lyme Neuroborreliosis the likely cause of this patient's SIADH.</p> <p>Discussion: SIADH is a disorder caused by the inability to suppress ADH, which results in impaired water excretion, leading to hyponatremia secondary to excess water. It is therefore intuitive that the main treatment is to restrict water intake in order to raise serum sodium. However, the underlying cause of SIADH is important to recognize in order to effectively treat the patient.</p> <p>The most common causes of SIADH are malignancy, medications, CVA, trauma, and recent surgeries. CNS infections are a rare but established cause of SIADH, including herpes, bacterial, and tuberculous infections. CNS involvement in Lyme's disease can manifest in early to late disseminated disease as a lymphocytic meningioencephalitis associated with cranial neuropathy. Lyme's disease causing SIADH is reported in two other cases of disseminated disease, and could have precipitated SIADH in this patient.</p> <p>This case illustrates that although CNS infection is a rare cause of SIADH, it is vital to recognize encephalitis in the setting of SIADH, as treatment of the underlying disease can effectively treat the electrolyte disturbance and prevent fatal complications from the primary cause itself.</p>	<p><b>Author: Mazen Zaarour, MD</b> Additional Authors: Mazen Zaarour1*, Samer Hassan2, Nishitha Thumallapally1, Qun Dai2 1Department of Medicine, Staten Island University Hospital, North Shore-LIJ Health Care System, Staten Island, New York 2Department of Medicine, Division of Hematology and Oncology, Stat Institution: Staten Island University Hospital</p> <p><b>Title: Rivaroxaban-Induced Spontaneous Spinal Subdural Hematoma: An Uncommon, Yet Devastating Complication</b></p> <p>Spinal hematoma is an uncommon, yet devastating neurosurgical entity. Spontaneous or non-traumatic spinal subdural hematomas (SSDH) are extremely rare, and are associated with numerous risk factors, such as iatrogenic causes, bleeding disorders, spinal tumors, arteriovenous malformations, and oral anticoagulants (OACs). The incidence of SSDH in the setting of rivaroxaban is unknown, and is limited to case reports. To the best of our knowledge, to date, only three cases described rivaroxaban-induced spontaneous spinal hemorrhages, including only one case of rivaroxaban-induced spontaneous SSDH.</p> <p>We report the case of a 58-year-old male that presented to our institution with acute inter-scapular back pain. In the emergency department, the patient developed rapidly progressing ascending bilateral lower extremity weakness. He denied any history of trauma. The patient's prior medical history was remarkable for diabetes mellitus, hypertension, atrial fibrillation treated with rivaroxaban, and a recent left hip arthroplasty that required spinal anesthesia.</p> <p>Physical examination revealed an alert and oriented patient in no acute distress. There were no neurological deficits in the cranial nerves or upper limbs. The motor strength in both lower limbs was of 0/5. A sensory level was evident at the level of T2, with positive bilateral babinski reflexes. The rest of the exam was unremarkable. Initial laboratory assays included a hemoglobin concentration of 11 g/dL, a platelet count of 176,109/L, an aPTT of 36.6 second, a PT of 18.4 second and an INR of 1.6. Kidney function and liver enzymes were normal. A non-contrast CT of the brain was negative for bleed.</p> <p>The clinical presentation combined with findings on the neurological exam pointed towards a spinal pathology. Moreover, a spinal bleed was strongly suspected given the history of recent spinal anesthesia in addition to the rivaroxaban use. An emergent cervical and thoracic MRI demonstrated an acute 6.3 x 0.6 x 1.6 cm intradural hematoma from C7 to T2, with spinal cord edema. Given the high risk of rivaroxaban-induced bleeding associated with surgery, the patient was treated conservatively with intravenous dexamethasone and aminocaproic acid. Surgical intervention and evacuation of the hematoma was done on hospital day 5, after which the patient began slowly regaining motor strength. He was discharged a few days later for intensive rehabilitation.</p> <p>Recently, the indications for OACs, including rivaroxaban, have expanded, and thus physicians should be aware of the increase in the incidence of spinal hematomas in patients receiving anticoagulant therapy. Available clinical trials showed lower incidence of rivaroxaban-associated major bleeding events, however, even a single serious bleeding event secondary to OAC therapy is a devastating complication for the caregiver and for the patient. Therefore, establishment of a treatment algorithm or discovery of an antidote is of utmost importance.</p>
--	---

**Author: Fnu Zarlisht, MBBS**

Additional Authors: Thomas Frohwein, MD; Katherine Lin, MD; Nisarg Patel, MD  
Institution: Lincoln Medical and Mental Health Center

## **Title: RHEUMATOID ARTHRITIS COMPLICATED BY HYPERVISCOSITY SYNDROME**

**INTRODUCTION:** Rheumatoid arthritis is an immunological condition that primarily affects the joints, but may include symptoms like serositis and anemia. Hyperviscosity syndrome is a rare complication [1, 2], associated with markedly elevated titers of rheumatoid factor and elevated immunoglobulin levels. We present a case report of a patient with rheumatoid arthritis and hyperviscosity syndrome, presenting with chronic intermittent nasal bleeding. This is a unique presentation of rheumatoid arthritis and will increase physician awareness.

**KEY WORDS:** rheumatoid arthritis, rheumatoid factor, hyper viscosity syndrome.

**CASE PRESENTATION:** A 50-year-old female with history of rheumatoid arthritis, splenomegaly, 3-5 % plasma cells, and pancytopenia came for follow up to the hematology/oncology clinic. She complained of intermittent nosebleeds, as well as dizziness and dry mouth. Workup revealed significant serum hyper viscosity of 11.9 (relative to water, reference range 1.5-1.9), evidence of hypergammaglobulinemia with elevated IgG (5209) and IgM (1113) concentrations. Bone marrow biopsy was negative for malignancy, showing polyclonal plasmacytosis 3-5%. Other results were CCP strong positive at 250, positive cryoglobulins, pancytopenia (WBC 4.1, Hgb 7.3, Hct 22.6, Plt 96), elevated erythrocyte sedimentation rate of 135 mm/h, positive anti-nuclear antigen (ANA) at 1:160 with homogenous pattern, elevated rheumatoid factor at 26,900 (reference range <15). Imaging revealed splenomegaly, known from before, without lymphadenopathy. Ophthalmological evaluation was negative for acute hyper viscosity retinopathy. The bone marrow biopsy revealed reactive, polyclonal plasmacytosis. SSA/SSB antibodies were negative. Several blood samples were rejected by the lab due to specimen clotting. The patient was diagnosed with seropositive rheumatoid arthritis, complicated by hyper viscosity syndrome.

**DISCUSSION:** This 50yo woman was asymptomatic for known rheumatoid arthritis when she developed recurrent nosebleeds and was diagnosed with hyperviscosity syndrome. This is likely due to increased, non-directed activation of the immune system, as evidenced by strongly positive rheumatoid factor and hypergammaglobulinemia. Hyperviscosity syndrome is a potentially serious condition that can lead to retinopathy, stroke, bleeding, and other end-organ damage related to "clumping" of the blood cells. Clinicians should be aware of this potential complication and recognize the signs and symptoms of hyperviscosity syndrome in patients with underlying rheumatoid arthritis. This patient was diagnosed before the development of further complications and treated with plasmapheresis, followed by immunosuppressive therapy.

### **REFERENCES:**

1. Silberman S, Holmes EW, Miller BJ, Messmore HL Jr, Barr WG. A case of rheumatoid hyperviscosity syndrome with characterization of the serum immune complexes. *Ann Clin Lab Sci* 1986;16:26-33.
2. Scofield RH, Tardibono G. Rheumatoid hyperviscosity: analysis of a patient with intermediate complexes that block other antibodies and a review of the literature. *Semin Arthritis Rheum* 1998;27:382-391.

**Author: Adam Zayac, MD**

Additional Authors: Rushikesh Shah  
Nidhi Bansal  
Institution: Upstate Medical University Hospital

## **Title: Thyrotoxic Hypokalemic Periodic Paralysis-The Importance of History**

The diagnosis of Thyrotoxic hypokalemic periodic paralysis can be missed due to its rarity. The disease is an easily treatable entity but, if missed, can be debilitating and life threatening. Awareness among clinicians about this rare but potentially treatable condition is vital. We present a case where a young healthy male was admitted to our Critical Care Unit with suspicion of stroke, was diagnosed and treated for hypokalemic periodic paralysis, and had complete recovery. A 27-year-old Asian male came into the emergency room with significant weakness in his arms and legs following an episode of nausea and vomiting. He denied any other past medical or surgical history. He was adopted as a child by Caucasian parents and is an engineering student. He denied any history of smoking and use of illicit drugs, with his last consumption of alcohol about four months ago. He denied taking any prescription or over-the-counter medications. On admission, his vitals were within normal limits. Upon exam, he had zero out of five strength in his hips, two out of five strength in his knees and shoulders, normal strength distally, and bicipital and patellar hyporeflexia. CT scan of head was within normal limits. On laboratory findings, his potassium was 1.7. Further labs revealed TSH less than 0.030, T3 391.1, Free T3 17.42, and Free T4 4.24. Thyroglobulin antibodies were 7.2. Thyroid peroxidase Ab was 0.9. Ultrasound of his neck revealed a heterogenous, enlarged thyroid gland without masses and slightly asymmetric, with increased blood flow to the right thyroid lobe. Nuclear thyroid uptake scan was consistent with Graves' disease. He was diagnosed with Thyrotoxic hypokalemic periodic paralysis. Aggressive potassium replacement protocol was started in the ICU. The patient was started on metoprolol and methimazole for thyrotoxicosis. His symptoms resolved completely on day three and returned to his baseline – able to perform all ADLs by himself. He was discharged from hospital on potassium supplements, metoprolol, and methimazole and was instructed to follow-up in Endocrine clinic. At time of follow-up, his symptoms had not recurred, TSH was undetectable, Free T4 was 0.98, T3 131.7 and potassium was 4.4; radioactive iodine ablation was also discussed.

HPP can be hereditary or acquired, related to thyrotoxicosis. Thyrotoxic hypokalemic periodic paralysis is a rarely seen condition in the US, but is a well-known complication of uncontrolled hyperthyroidism in eastern Asian populations. TPP affects males more than females. These episodes can be triggered by large, carbohydrate-laden meals or following heavy exercise. Definitive management of hyperthyroidism is the mainstay of therapy; however, non-selective beta-blockers and avoidance of high-salt and high carbohydrate meals, and strenuous exertion until thyrotoxicosis is adequately managed are essential in acute management.

**Author: Zhihong Zhang, MD**

Additional Authors: Aaron Goldberg, MD, PhD, Raajit Rampal, MD, PhD Address: Memorial Sloan Kettering Cancer Center, New York, NY 10065  
Institution: SUNY Downstate Medical Center

**Title: INDOLENT SYSTEMIC MASTOCYTOSIS PRESENTING AS ALCOHOL INTOLERANCE**

**Introduction**

Mastocytosis refers to a group of clonal hematologic diseases involving abnormal growth and accumulation of mast cells. Typically, cutaneous mastocytosis (CM) is limited to the skin, while systemic mastocytosis (SM) involves infiltration of extracutaneous organs, with or without skin involvement. Here we report a case of indolent SM presenting as alcohol intolerance.

**Case**

A 49-year old man with a 20-year history of pruritus and rash after showering presented with significant reactions to alcohol for 1.5 year. Ten minutes or less after drinking, he would experience severe flushing and a very upset stomach with nausea that prevented him from further drinking and eating. He also reported 2 prior episodes of severe chest discomfort and rash with unclear trigger; cardiac workup was unrevealing. The patient denied a history of anaphylaxis, angioedema, syncope, aspirin hypersensitivity, or peptic ulcer symptoms. Physical examination revealed minimal splenomegaly, with spleen palpable on deep palpation. No hepatomegaly or lymphadenopathy was noted. Erythematous macules over knees were noted. Darier's sign was negative. Splenomegaly was confirmed by images of abdominal ultrasound measuring 16cm in sagittal plane and CT of abdomen and pelvis. The CT also showed multiple slightly enlarged retroperitoneal lymph nodes (1.6x1.2cm), as well as multiple mildly prominent mesenteric lymph nodes. CT chest showed very small pericardial lymph nodes. Bone marrow biopsy was consistent with SM, with a phenotypically abnormal CD2+/CD25+ mast cell population and multifocal dense infiltrates of mast cells. There was a high percentage (25-30%) of mast cells in these infiltrates, but the overall percentage of mast cells remained low. Molecular studies were negative for KIT D816 mutation. RT-PCR was negative for BCR-ABL. Tryptase was elevated at 93 ng/dl (normal: 2-10 ng/dl). Symptomatic improvement in terms of pruritus was observed by admission of famotidine and cetirizine.

**Discussion**

Symptoms of SM are various, including flushing, nausea, diarrhea, pruritus, hypotension, syncope, anaphylaxis and pain. The patient's overall presentation is consistent with indolent SM without skin involvement, which is diagnostic challenging. Early recognition of this disease is important for timely therapy to improve quality of life. An important aspect of treatment is avoidance of known symptom triggers, including complete abstinence from alcohol. Most SM patients require one or more medications, such as H1 and H2 antihistamines, cromolyn sodium, antileukotriene agents, and proton pump inhibitors, to counteract the symptoms. Since the patient is already on antihistamine medications with some symptomatic improvement, the medications should continue. The patient should wear a medical alert bracelet and carry epinephrine in a self-injectable form at all times for treatment of possible anaphylaxis. The patient would need multidisciplinary planning prior to any surgery. Close coordination between the hematologist or allergist and members of the surgical and anesthesia teams is important.

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

**Resident/ Fellow**  
**Quality, Patient Safety and Outcomes**

<p><b>Author: Arjun Bhansali, MD</b>                  Additional Authors: Jerry Jomi, MS4                  Justin Alexander, MS4                  Bisrat Haile, MD                  Kalpana Panagrahi, MD                  Rajat Mukherji, MD                  Institution: Kingsbrook Jewish Medical Center</p> <p><b>Title: An Analysis of Head CT Scans in Syncope: Is the Benefit Worth the Cost?</b></p> <p>Syncope is a sudden and transient loss of consciousness and postural tone. It is a common clinical problem, accounting for up to 3% of Emergency Department visits and 6% of hospital admissions every year. Head CT scans, though routinely ordered, have an unclear benefit in the work up of syncope. This was a retrospective study performed to assess the percentage of patients who benefited from a non-contrast head CT procedure, and to estimate the total cost of those CT scans where no benefit was achieved. The population of study included patients presenting to the Emergency Department of Kingsbrook Jewish Medical Center (KJMC) with a working diagnosis of syncope from June 2011 to January of 2014, and all CT scans were completed within 24 hours of patient arrival. Patients in whom another major diagnosis (e.g. CHF exacerbation) as a cause of syncope were not included. Of 477 CT scans of the head, 15 patients had head CT scans which demonstrated evidence of scalp injuries or hematomas. 25 patients showed other unexpected but clinically significant findings. There were 17 space occupying lesions, including 12 previously undiagnosed meningiomas and 5 patients with radiological evidence of normal pressure hydrocephalus, and we also discovered vascular pathology in 8 patients of whom seven had evidence of acute cerebral infarction. At \$696 per CT scan (including interpretation fee), the total cost over the 44 month study period was \$331,992. A total of 437 patients (91.6%) had no significant findings on head CT scan. This totaled \$304,152 spent on scans wherein no benefit was achieved. The results of our study clearly show that indiscriminate use of head CT scans in patients admitted with the diagnosis of syncope is an unproductive exercise. Although in our study 25 patients (5.2%) of patients did have unexpected pathology, the actual benefit is questionable. More stringent use of head CT's can ultimately lead to better appropriation of hospital funds and resources.</p>	<p><b>Author: Lediya Cheru, M.D.</b>                  Additional Authors: Shreyas Patel M.D., Tarek Ibrahim M.D., Jingping Hao M.D., Nouman Syed M.D., Josephine Lee M.D.                  Institution: Albany Medical College, Albany Medical Center</p> <p><b>Title: Using the Electronic Health Record (EHR) to Enhance Continuity of Care in Patient-Centered Medical Home</b></p> <p>Purpose of the Study:                  The objective of our study was to improve patient empanelment in a resident primary care clinic by linking patients with specific resident physicians through the EHR.</p> <p>Methods:                  Prior to the initiation of the study, patients at the Albany Medical Center Internal Medicine Group resident clinic were linked to a supervising attending through an "identification banner" in AllscriptsTouchworks. This led to inconsistencies with patient follow-up and the fragmentation of patient care provided by residents. To improve patient empanelment, eight medical residents rotating through the outpatient clinic identified 2,922 patients seen by 24 residents over the previous 12 months. Pre-established criteria were created to link each patient to a specific resident primary care physician (PCP). The "PCP" fields in the EHR were updated to reflect the appropriate resident PCP. "Monday Morning Huddles" (MMHs) were also instituted to review upcoming schedules, identify incorrectly assigned patients, and assess the accuracy of the banners. Residents used a tasking message system within AllscriptsTouchworks to notify scheduling staff of the appropriate follow-up PCP. One year following these changes, we surveyed 15 residents, 5 attendings, and 4 scheduling staff members to evaluate the impact on continuity of care.</p> <p>Results                  Of the 2,922 patients, 1,368 were identified as duplicates or assigned to attendings. Of the remaining 1,554 patients, each was linked to a specific medical resident. Information Technology (IT) department, subsequently changed the banners to reflect the assigned residents. In retrospective surveys, attendings and residents reported improvement in appropriately scheduled resident appointments from 25% to 65% and 35% to 72%, respectively. Attendings attributed 42% of this improvement to banner changes, 52% to the "3+1" block system instituted one year prior to the study, and 6% to other factors. Residents credited 55% to banner changes, 34% to the new "3+1" block system, and 11% to other factors. Attendings and residents reported that banner changes have had a significant impact on resident-patient relationships and continuity of care. Implementation of MMHs decreased inaccuracies in mislabeled PCP banners from 50% to 28%. Qualitative data obtained from scheduling staff reflected improvement in identification of the appropriate resident for follow-up. Residents also noted improvement in patient attendance and personal satisfaction.</p> <p>Conclusions:                  An interdisciplinary team of medical residents, administrative staff, and the IT department was able to utilize the EHR to improve empanelment of patients in the primary care resident clinic. Based on surveys of attendings, residents and administrative staff, the addition of the banner changes played a major role in establishing consistent resident patient panels. MMHs continued to be valuable to monitor the accuracy of the banners and continuity of care.</p>
---	---

## Resident / Fellow Quality, Patient Safety and Outcomes

<p><b>Author: Christine Garcia, MD, MPH</b>                  Additional Authors: Sahar Ahmad, MD                  Institution: Stony Brook University Department of Internal Medicine</p> <p><b>Title: Success, adverse event rates, and time to procedure for resident and fellow-performed ultrasound-guided procedures compared to a specialty procedure service- driven paradigm</b></p> <p>Introduction:                  Ultrasound (US) guidance for procedures including paracentesis and thoracentesis is widely accepted as best practice at many academic institutions. US guided paracentesis is associated with fewer adverse events and lower hospitalization costs than procedures where US is not used. US guidance reduces risk of pneumothorax after thoracentesis and bleeding complications after paracentesis, resulting in a measureable reduction in hospitalization costs and LOS. US guidance also increases the yield of thoracentesis.                  Internal medicine residents and fellows trained in US currently perform bedside procedures, including vascular access, paracentesis, thoracentesis and abscess drainage by US guidance at our institution. Stony Brook house staff report varying levels of confidence in performing US procedures which correlated to number of times performed. This survey showed a need for more equipment, 61.70% reported to have witnessed the unavailability of portable US resulting in a delay in patient care.</p> <p>Methods:                  A retrospective chart review of thoracentesis and paracentesis procedures was completed to compare procedures performed at the bedside with US and those performed by Interventional Radiology (IR), a procedure specialty service at our institution. The following parameters were assessed: Resource utilization, time delay between request for and performance of paracentesis and thoracentesis, success rates and adverse events.</p> <p>Results:                  Review of 39 patients showed that mean time to bedside US-paracentesis was 3 hours and 40 minutes (SD 0.09) compared to IR-paracentesis was 14 hours and 23 minutes. (SD 0.33). Mean delay time for bedside US-thoracentesis was 4 hours and 32 minutes (SD 0.08) compared to IR- thoracentesis was 8 hours and 49 minutes (SD 0.32). Bedside US-guided paracentesis was completed on average 11 hours faster than IR procedures and US-guided thoracentesis was completed 4 hours faster. All bedside-US procedures were successful without adverse events and performed by residents or fellows supervised by an attending. IR procedures also were successful without any adverse events, however resource utilization was markedly higher. All IR procedures were performed by attendings and required additional ancillary staff including transport and nursing support, with some requiring portable oxygen and respiratory support.</p> <p>Conclusions: Resident and fellow-performed US-guided paracentesis and thoracentesis were associated with 100% success rates no adverse events, equal to that of specialty service-driven results. US-guided procedures carry the benefit of reduction in time delay and resource utilization. Additional US units are needed to enable residents and fellows to continue performing these procedures which are anticipated to produce improved work flow and quality of care.</p>	<p><b>Author: Deepthi Kunduru, MD</b>                  Additional Authors: Deepika Gupta MD, Bharat Bajantri MD, Franchin Giovanni MD, Sridhar Chilimuri MD                  Institution: BRONX LEBANON HOSPITAL CENTER</p> <p><b>Title: EFFICACY OF MONITORING FOR AMIODARONE TOXICITY</b></p> <p>Title : Efficacy of Monitoring for Amiodarone toxicity                  Presenting Author: Deepthi Kunduru MD, Bronx-Lebanon Hospital Center</p> <p>Objective : Aim of the study is to evaluate the efficacy of monitoring of Amiodarone toxicity in our inner city hospital system.</p> <p>Background: Amiodarone is increasingly being used for refractory arrhythmias in spite of its potential toxicity. The North American Society of Electrophysiology and Pacing recommends baseline complete physical including eye examination, EKG, Chest X ray (CXR), Liver, Thyroid and Pulmonary function testing (LFT, TFT and PFT). Follow-up annual EKG and CXR and half-yearly liver and thyroid function testing are also recommended. In symptomatic patients, follow up eye examination and PFTs are recommended. Experts also recommend a quick taper to the minimum effective dose .</p> <p>Quality Assessment Methods: We did a retrospective study including patients who were discharged from our hospital on Amiodarone between January 2013 to June 2014. Charts were reviewed to assess for adherence to follow-up, tapering of the dose and monitoring for toxicity as per guidelines . A total of 58 patients were discharged on Amiodarone from our hospital during this period . Maximum duration of follow-up was 18 months.</p> <p>Results: All 58 patients had baseline chest X-ray and EKG. 57 patients (98%) had baseline LFTs and 53 patients (91%) had documented baseline TFTs. On follow-up visits, 39 patients (67%) had follow-up EKG and 14 patients (41%) had follow-up CXR at the end of one year. 24 patients (57%) had follow-up LFTs and 18 patients (45%) had follow-up TFTs at the end of 6 months. One patient had baseline PFT with no follow-up. We observed the following complications; 5 patients (9%) had EKG changes/bradycardia precluding continuation of drug ; 4 patients (7%) had abnormal TFTs ,which required dose adjustment and thyroid replacement therapy ; 2 patients (4%) had abnormal LFTs prompting discontinuation of drug ; One patient (2%) was noted to have pulmonary fibrotic changes and one patient (2%) had Cornea verticillata.</p> <p>Conclusion: In our study population, Amiodarone toxicity was not uncommon and monitoring was inadequate .Hospitals need to develop protocols for proper follow-up of patients on Amiodarone to minimize toxicity. We have developed a new order set in our Electronic Medical Records which includes baseline and follow-up monitoring tools including alerts for physicians .</p>
--	--

## Resident / Fellow Quality, Patient Safety and Outcomes

**Author: Narasa Madam, MBBS**

Additional Authors: Abhijana Karunakaran, MD ; Kothai Divya Guruswamy Sangameswaran, MBBS; Roberto Diaz Del Carpio, MD:

Institution: UNIVERSITY AT BUFFALO

**Title: IMPROVEMENT IN HYPERLIPIDEMIA MANAGEMENT.**

Purpose: To increase the number of patients aged 40-75 years at the Hertel clinic appropriately managed for hyperlipidemia by 15%, by care providers based on ATP4(Adult Treatment Panel 4) cholesterol management guidelines.

Methods: Root-cause analysis was done to identify limitations in hyperlipidemia management at various levels of care. Initial hypothesis was that the greatest limitations were professional factors such as providers not risk stratifying due to forgetfulness, inconvenient access of risk calculators, and knowledge of current guidelines. Two interventions were conducted via PDSA (Plan Do Study Act) format targeting the identified limitations. Ten patients aged 40-75years per week were randomly selected. Outcomes were compared between a baseline group (40 patients prior to any intervention) and a cumulative 100 patients post interventions.

Results:The first intervention targeting memory limitation of providers by placing reminders around the clinic failed to achieve the significant rise in use of 10year ASCVD(Atherosclerotic Cardiovascular disease) risk calculator(from 11.5% to 13.63%). Root-cause analysis restudied and second intervention was conducted to educate providers on current guidelines and availability of an easily accessible calculator in the Electronic Medical Record (EMR) system. This intervention increased use of risk calculator (from 11.5% to 25%) and patients appropriately screened (from 61.6% to 88.8%) and correctly managed (from 35.8% to 50%). Results were analyzed using run charts.

Conclusion:Interventions placed to improve hyperlipidemia management targeting professional limitations through reminders, education of guidelines and accessibility of tools for 10 year ASCVD risk calculation resulted in rise of patients appropriately screened and correctly managed. To sustain this rise it is planned to continue this education to providers.

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

**Resident/ Fellow**  
**Research**

## Resident / Fellow Research

<p><b>Author: Eliane Abou-Jaoude, MD</b>                  Additional Authors: Anju Chana, Rukaya Khan, Amardeep Sapple, Jose Luis Aranez, Nitu Kataria, Catherine O'Neill                  Institution: University at Buffalo</p> <p><b>Title: Acceptance and Perceptions of Influenza Vaccination in a Single Ambulatory Primary Care Center</b></p> <p>Background:                  Influenza can lead to a significant disease burden with serious morbidity and mortality. Since the 2009 H1N1 outbreak, several studies have attempted to estimate the impact of influenza and found that disease related burden might be under-detected. An estimate from 2010-2011 noted that only 40% of adults received the influenza vaccination. Several studies have therefore identified barriers to vaccination such as fear of side effects, perception of low risk status, ineffectiveness, and necessity. Our aim was therefore to evaluate our patient population's acceptance of the influenza vaccine and to investigate factors that could be associated with perceptions about the vaccine.</p> <p>Methods:                  An observational study involving 146 subjects was conducted at our ambulatory center between September 2014-December 2014. We provided a single paged questionnaire while our patients were in the waiting room. We collected demographic data on age, sex, race, education, perceived health status, prior vaccination. We requested the patients to score perceptions regarding the influenza vaccine (effectiveness, efficacy, and belief contracting influenza) on a 4 point scale.</p> <p>Results:                  The majority of patients was in the age 41-50 group (n = 71; 48.6%), female (n = 82; 56.2%), black (n = 75; 51.4%), high school education (n = 55; 37.7%) and had a perceived health status of fair (n = 82; 56.2%). There were 96 subjects (65.8%) who refused the influenza vaccine and the most common reasons were "side effects" (n = 20; 20.8%), "does not work" (n = 13; 13.5%), "never get the flu" (n = 13; 13.5%), and "do not like shots" (n = 11; 11.5%). Our population included 20 (13.7%) never vaccinated compared to 103 (70.5%) prior vaccinated. There was no statistical significance in the perceived effectiveness of the vaccine between the groups (p = 0.133). The prior vaccinated group had perceived a "somewhat high" chance of contracting the flu without the vaccination (p = 0.009) and also perceived a belief of "not" contracting the flu from the vaccination (p = 0.004).</p> <p>Discussion:                  Our study showed that only 30% of our population agreed to receive the influenza vaccine. Previous vaccination status appears to be associated with better tolerance. Further efforts need to be developed in educating our patients regarding the benefits of the influenza vaccination.</p>	<p><b>Author: Andrea Alejo, MD</b>                  Additional Authors: Silvano Rodriguez MD, Waina Cheng MD, Misbahuddin Khaja MD                  Institution: Lincoln Medical Center</p> <p><b>Title: An Unusual Presentation of Plasmablastic Lymphoma</b></p> <p>INTRODUCTION Plasmablastic Lymphoma (PBL) was first described as an aggressive B-cell lymphoma occurring in the oral cavity arising in the context of HIV infection (1), also frequently associated with EBV infection. PBL is a rare entity, thought to account for approximately 2.6% of all AIDS-related lymphomas. It is characterized by a monomorphic proliferation of round to oval shaped cells with plasmacytoid features. (2) The hallmark immunohistochemical staining pattern of PBL is that of terminally differentiated B lymphocytes (2). An important aspect of the initial treatment of PBL is the use of chemotherapy. Given the high proliferation index of PBL and its aggressive blastic appearance, more intensive regimens like CODOX-M/IVAC (cyclophosphamide, vincristine, doxorubicin, methotrexate, ifosfamide, etoposide, cytarabine )were used in some series (3). In patients with HIV infection and PBL, the use of HAART is recommended. Based on the principle that PBL is a rare entity, we present a case of an HIV-positive man with Plasmablastic Lymphoma, arising from a mediastinal mass, who's presentation and natural history of the disease lacks the typical characteristics presented in the literature available.</p> <p>CASE SUMMARY This is a case of a 59 year old man with a history of HIV on antiretroviral therapy with CD4 count of 349 cells/mm<sup>3</sup>; was brought into the hospital with complaints of one month history of constitutional symptoms. Initial assessment consisted of severe hypercalcemia, acute kidney injury and lactic acidosis. Brain CT was obtained that revealed innumerable lytic lesions throughout the calvarium and the skull base. Chest CT showed multiple cystic masses within the anterior mediastinum and a 2 cm soft tissue nodule just inferior to the left thyroid lobe, retrospectively a chest CT from 2 years prior to the diagnosis revealed an anterior mediastinal mass. A bone marrow biopsy was obtained that was negative for lymphoproliferative disease and finally a mediastinal biopsy of the mass showed PBL with EBV scattered Neoplastic Cells, CD 138+, CD 20- and EBER+. Soon after diagnosis patient was started on aggressive chemotherapy and expired less than a year later.</p> <p>DISCUSSION This case represents a patient that has characteristics that are typically associated with the presence of PBL such as HIV, positive EBV infection. Yet also has atypical manifestations of the disease, such as the location, clinical presentation and his CD 4 count of &gt; 300, differing from the usual HIV positive patient that develops PBL.</p> <p>CONCLUSION PBL continues to be a rare and difficult to treat disease. The prognosis remains poor and ,it seems as though even the most aggressive chemotherapies do not increase survival (4). Throughout the years cases have been published with rare disease presentation, such as the case presented, making it even more arduous to diagnose and manage.</p>
---	---

## Resident / Fellow Research

<p><b>Author: Hassan Alkhawam, M.D.</b>                  Additional Authors: Raef Madanieh M.D., Robert Sogomonian M.D., Feras Zaiem M.D., Mohammed El-hunjul M.D., Joseph J Lieber M.D., Timothy J Vittorio M.D.                  Institution: Icahn School of Medicine at Mount Sinai (Elmhurst) program</p> <p><b>Title: RISK FACTORS FOR CORONARY ARTERY DISEASE AND ACUTE CORONARY SYNDROME IN YOUNG ADULDS</b></p> <p><b>Objective</b>                  To assess the risk factor profile for premature coronary artery disease (CAD) and Acute Coronary Syndrome (ACS) presentation in younger adults.</p> <p><b>Methods</b>                  Retrospective chart analysis of 393 patients =40 years old admitted from 2005 to 2014 for chest pain and underwent coronary angiography. The implication of modifiable risk factors and non-modifiable risk factors were evaluated in those with obstructive CAD (LM stenosis of =50% or stenosis of =70% in a major epicardial vessel), non-obstructive CAD (=1 stenosis =20% but no stenosis =70%) and normal coronaries (no stenosis &gt;20%). Additionally we evaluated the impact of the same risk factors on ACS presentation (NSTEMI vs STEMI) and the extent of CAD (single-vessel/multi vessel).</p> <p><b>Results</b>                  Of 393 patients, 212 had CAD (153 obstructive vs 59 non-obstructive) while 185 had normal coronaries. 52 patients presented with STEMI while 140 presented with NSTEMI. Of 212 patients with CAD, 96 patients (45.3%) had single vessel disease vs 116 (44.7%) multiple vessel disease. When compared to patients with normal coronaries patients with CAD were more likely to be smokers (RR 1.7 p &lt;0.0001), dyslipidemic (RR 1.5 p &lt;0.0001), Diabetic (RR 1.4 p 0.0002) cocaine users (RR 1.2 p 0.4) have a family history of premature CHD (RR 1.5 p &lt;0.0001) and be males (RR 1.3 p &lt;0.0001). Smokers were 5 times more likely to present with STEMI (p &lt;0.0001) and 1.7 with NSTEMI (p 0.0003) compared to the control group. When compared head to head, smokers were 2.2 times more likely to present with STEMI compared to NSTEMI (p&lt;0.001). Smoking also, alone and with another risk factor increased the risk of obstructive vs no obstructive CAD (for both groups RR: 1.2, p 0.02). No significant difference was noted in the single vessel vs multi vessel CAD subgroups.</p> <p><b>Conclusion</b>                  In our population of young adults, smoking as a single risk factor was the most prevalent for earlier CAD. It was also associated with more STEMIs and obstructive CAD. Healthcare intervention in the general population through screening, counseling and education regarding smoking cessation is warranted to reduce premature coronary artery disease.</p>	<p><b>Author: Hassan Alkhawam, M.D.</b>                  Additional Authors: Raef Madanieh, Mariya Fabisevich, Robert Sogomonian, Mohammed El-Hunjul, Sumair Ahmad, Timothy J Vittorio                  Institution: Icahn School of Medicine at Mount Sinai (Elmhurst)</p> <p><b>Title: The Role of Alcohol Abuse and Tobacco Use in The Incidence of Early Acute Coronary Syndrome</b></p> <p><b>Objective/Purpose:</b> To investigate the synergistic role of alcohol abuse/dependence and tobacco use in the early incidence of Acute Coronary Syndrome (ACS).</p> <p><b>Methods:</b> A retrospective chart analyses of 8076 patients diagnosed with ACS between 2000 to 2014, defined by ICD-9 codes for acute MI, alcohol abuse/dependence and tobacco use. Average age of ACS was calculated for the general population. Patients were then divided into 4 subgroups based on alcohol abuse/dependence and tobacco use status as follows: non-alcoholic non-smokers, non-alcoholic smokers, alcoholic non-smokers and alcoholic smokers.</p> <p><b>Results:</b> The mean age of our 8076 ACS patients population was ~59.5 (95% CI 59.2-59.8). Alcoholic abuse/dependence patients developed ACS at age 55.1 (n=172, 95% CI: 52-58) compared to 63.8 year old (n=7,904, 95% CI: 63.6-63.9) in non-alcoholic abuse group (P value &lt;0.001). When tobacco use is incorporated as a risk factor, those with both alcohol abuse/dependence and tobacco use seemed to develop ACS at age 51.1 years old (n=51, 95% CI: 48-54.2) compared to 56.3 (n=909, 95% CI:55-57.7) year old in Smoker Non-alcoholic abuse groups (P value 0.002). Furthermore, Alcoholic abuse/dependence and tobacco use developed ACS ~20 years earlier (Incidence age 51.1 years, n=51, 95% CI: 48-54.2) when compared to those with neither alcohol abuse/dependence nor tobacco use (Incidence age 71.3 years, n=6995, 95% CI: 71-71.6) (P value &lt;0.001). Finally, Alcoholic abuse/dependence and Non-smoker patients developed ACS at age 56.1 (n=121, 95% CI: 54.6-57.6) vs 71.3 years old (n=6995, 95% CI: 71-71.6) in Non-alcoholic Non-smoker group (P value &lt;0.001).</p> <p>The mortality rate of ACS in alcoholic abuse/dependence group was 9.1% vs 5.7% in non-alcoholic abuse patients (OR: 1.7, P value 0.1). 30-days readmission in the alcoholic abuse/Dependence group was 18.6% vs 11.24% in non-alcoholics-abuse (OR: 1.8, P value 0.03). Length of hospitalization was higher in ACS alcoholics patients'~9.3 days vs ~5.2 days in non-alcoholics patients (P value &lt;0.001).</p> <p>Alcoholic abuse/dependence patients with hypertension showed a higher rate of ACS (85.12%) compared to hypertension without alcoholic abuse (64.4%) (P &lt;0.001) while patients with dyslipidemia and DM didn't show any statistical significant (p values 0.4 and 0.9 respectively)</p> <p><b>Conclusions:</b> Alcohol abuse/dependence appears to be a risk factor for earlier ACS. In our population, the average age of ACS incidence in alcoholic patients was significantly earlier than non-alcoholic patients. Furthermore, alcoholic patients who also used tobacco developed ACS at an even younger age when compared to those who had history of either alcohol abuse/dependence or tobacco use alone, suggesting a possible synergistic effect of these two risk factors in developing early ACS. 30-days readmission rate and length of hospitalization were significantly higher in Alcoholic abuse/dependence group. Healthcare intervention in this population through screening, counseling and education regarding alcohol abuse/dependence and smoking cession is warranted to reduce early ACS.</p>
---	---

## Resident / Fellow Research

**Author: Lu Chen, MS**

Additional Authors: Uta Guo BS, Lijo C Illippambil MS, Crystal Ang BS, Juan Paolo Prudente MSS, Parag Mehta MD

Institution: New York Methodist Hospital

**Title: Caring For the Computerized Patient - Internal Medicine Residents Time Consumed by Clinical Documentation**

Introduction: For the past 20 years, increasing amounts of time were spent on clinical documentation. The implementation of Electronic Health Records (EHR) resulted in both physicians and patients complaining that physicians spent more time on computerized patient records than at the bedside. This study provides objective and longitudinal analysis of the time spent using EHR by Internal Medicine (IM) residents over the span of an academic year.

Methods: Active EHR usage data was collected from the EHR audit logs for IM residents from July of 2014 thru June of 2015. Active EHR use was defined as more than 3 mouse clicks, 1,700 mousemiles or 15 keystrokes per minute. EHR usage activities were divided into four sub-categories: Chart Review, Placing Orders, Documentation, and Other Activities. Electronic patient encounter (EPE) was defined as the total active EHR usage time on the same patient record within a single day. A total of 109 residents (41 PGY1, 31 PGY2, and 37 PGY3) were identified and included in the study.

Results: During the academic year, 109 residents accumulated 112,705 hours of active EHR use on 165,293 EPEs. An average resident spent most time per EPE in July and least amount of time in June (47 min vs. 33 min,  $p < 0.001$ ). Statistically significant reductions in time were also noted in three sub-categories of EPE: Chart Review (17 min vs. 12 min), Placing Orders (9 min vs. 6 min), and Other Activities (10 min vs. 6 min, all  $p < 0.001$ ). In addition, a modest reduction was seen in Documentation (11 min vs. 9 min,  $p = 0.2$ ). In July, each resident spent an average of 26 hours on clinical documentation in an 80-hour workweek. In June 2015, this amount was effectively reduced to 18 hours per workweek (a 31% reduction). Conclusion: Resident physicians spent a significant amount of their duty hours actively using EHR. Although reducing clinical documentation time due to increased proficiency and familiarity with EHR is observed. Further curtailing of time spent documenting on a computer is needed in order to optimize a physician's presence at the bedside.

**Author: Farshid Daneshvar, M.D**

Additional Authors: Farshid Daneshvar, M.D (ACP resident/fellow member, Staten Island University Hospital, Staten Island, NY); James Gabriel, M.D (North Shore University Hospital, Manhasset, NY); Danial Daneshvar, M.D (Staten Island University Hospital, Staten Island, NY); Michael Weinreich, M.D (North Shore University Hospital, Manhasset, NY); Michael Sperling, M.D (Staten Island University Hospital, Staten Island, NY); Chadi Salmane, M.D (ACP resident/fellow member, Staten Island University Hospital, Staten Island, NY); Harout Yacoub, M.D (ACP resident/fellow member, Staten Island University Hospital, Staten Island, NY); Marianne Smith, M.D (ACP associate member, Staten Island University Hospital, Staten Island, NY); Thomas McGinn, M.D (North Shore University Hospital, Manhasset, NY); Institution: Staten Island University Hospital

**Title: CARDIORESPIRATORY FITNESS IN INTERNAL MEDICINE RESIDENTS: ARE OUR FUTURE PHYSICIANS BECOMING DECONDITIONED?****Purpose:**

Maintaining Cardiorespiratory Fitness during residency training is a considerable challenge. Previous studies have shown a falloff in physical activity from medical school to residency. The implications of poor cardiorespiratory fitness may result in stress, increased resident burnout and ultimately cardiovascular disease and increased all-cause mortality. Physicians with poor exercise habits are also less likely to counsel their patients to exercise. Our study was conducted at the North Shore Long Island Jewish Health System Internal Medicine Residency programs at Staten Island University Hospital (SIUH), North Shore University Hospital and Long Island Jewish Medical Center to assess Internal Medicine Residents in these separate training programs for their change in exercise habits as well as their Cardiorespiratory Fitness age.

**Methods:**

Data regarding physical fitness levels and exercise habits along with height, weight, waist circumference and resting heart rate was collected in an anonymous cross-sectional survey. Cardiopulmonary fitness age was determined using a non-exercise regression model developed using data from the Norwegian Nord-Trøndelag Health Study (HUNT) cohort study. Differences between groups for continuous variables were evaluated with an independent-sample t test. For categorical data the Chi-square test or Fisher's exact test was used. Comparability of exercise habits before and during residency was evaluated using Bowker's test for table symmetry.

**Results:**

We found a significant reduction in the activity level of young doctors as they progress from medical school to residency: 9.1% of the residents responded that they never exercised prior to residency while, this number increased to 36.8% after starting residency ( $P < 0.001$ ). Additionally, 34.7% reported exercising every day prior to starting residency while only 4.8% reported exercising every day during residency ( $P < 0.001$ ). 79.1% of participants reported that residency obligations were their main barrier to regular exercise. We also found our residents' predicted fitness age to be higher than their chronological age. The residents' mean fitness age was 5.6 years older than their mean chronological age ( $P < 0.001$ ).

**Conclusion:**

A significant drop in physical activity and fitness was self reported as study participants progressed from medical school to residency training. We suggest that this change is likely related to the rigorous training and significant time constraints of a resident's schedule. Previous reports have found that physicians who are more dedicated to their own personal fitness are more likely to counsel their patients on the health benefits of regular exercise. We believe that our study adds to the current body of evidence that our residents are at risk, and intervention is necessary to improve the physical fitness of our future doctors and the patients they care for.

## Resident / Fellow Research

**Author: Yasaman Eslaamizaad, MD**

Additional Authors: Haris Kalatoudis, MD  
Roman Bernstein, MD  
Ravi Gupta, MD  
Institution: NYU Lutheran Medical Center

**Title: IS D-DIMER EFFECTIVELY USED TO DETERMINE THE NEED FOR CT WHEN EVALUATING PATIENTS FOR PULMONARY EMBOLISM?**

### Introduction

The D-dimer assay has been utilized for evaluating low risk patients for the appropriateness of imaging studies in diagnosing a possible pulmonary embolism (PE). The high sensitivity of the D-dimer assay makes the test an indispensable tool in the Emergency Department (ED) for aiding in the risk stratification of a possible PE. However, its low specificity, particularly in patient populations with multiple medical comorbidities, can make positive results difficult to interpret. The purpose of our study was to investigate whether D-dimer screening is being effectively used to determine the need for further diagnostic testing in ED patients.

### Methods

We performed a retrospective review of all patients who underwent D-dimer testing in the ED from July 2013 to December 2014. Vitals and modified Wells inclusion criteria were collected for each patient. A D-dimer value of >0.5 mg/L was considered positive and diagnosis of PE was made on the basis of CT-angiography (CTA). Clinical algorithms for diagnosing PE mandate that patients with a low clinical suspicion undergo D-dimer testing, followed by CTA if positive. For patients with high clinical suspicion for PE, CTA should be performed without D-dimer testing.

### Results

We evaluated 68 patients with D-dimer testing ordered, 38 of whom had positive D-dimer tests. Five patients with a Wells score =4 had an inappropriate D-dimer ordered and each of them had a confirmed PE on CTA. 63 (93%) patients with Wells scores of 0-3 had D-dimer tests appropriately ordered. Of the 33 patients who received appropriate D-dimer testing and had positive D-dimers, 23 patients (70%) did not undergo CTA and 30 patients (91%) did not receive a venous Doppler study.

### Conclusion

The accuracy of identifying PE improves when clinical probability is estimated before the use of diagnostic testing. Within our institution, our data suggest that 93% of D-dimers were correctly ordered based on Wells criteria, indicating that ED is appropriately ordering D-dimer assay. However, we found lack of follow-up by CTA for 70% of positive D-dimers for suspected PE. This indicates a potential discrepancy in ED utilization of D-dimer testing as a screening tool in the diagnosis of PE. Our study suggests that despite stringent evidence-based protocols for evaluating patients with suspected PE, these algorithms are poorly implemented in our ED setting. Future studies, with an increased sample size, should investigate the causes of lack of CTA follow-up for positive D-dimer tests in the ED setting.

**Author: Zerelda Esquer, M.D.**

Additional Authors: Debroy Paula M.D., Quintero Orlando M.D., Katsa, Ioanna M.D., Julie Hoffman M.D.  
Institution: Jacobi Medical Center

**Title: High Late HIV presentation in the Honduran Garifuna community in the Bronx: a retrospective analysis**

### Background:

The Garifuna area Honduran ethnic minority of African-Caribbean origin, of which an estimated 200,000 live in New York City (NYC). Prior studies reported a high seroprevalence among the Garifuna living in Honduras (5%), though little is known among those living in NYC. We evaluated the characteristics of HIV infection in this understudied and underserved population at an urban medical center in the Bronx.

### Methods:

We retrospectively identified 27 Honduran Garifuna patients attending Jacobi Medical Center's HIV Clinic (JMC), between 1991-2014. Demographic and clinical data were extracted from their charts. Late HIV presentation (LHP) was defined as patients presenting for care with CD4 count below 350 cells/mm<sup>3</sup> or AIDS-defining event regardless of the CD4 cell count.

### Results:

Median age was 48 (30-66), 15 were men, 12 women. Presumed HIV acquisition was primarily heterosexual sex 90%, whereas MSM and blood transfusion accounted for 2 and 1 cases, respectively.

Median CD4 at presentation to JMC was 207 cells/mm<sup>3</sup> (14-981) in 13 patients with prior antiretroviral treatment (ART) and 55 cells/mm<sup>3</sup> (6-834) in 14 patients naïve to ART. LHP was observed in 90% of the patients (24), 10 of them by CD4 cell count alone, whereas 14 had AIDS-defining event including: TB(4), PCP (4), esophageal candidiasis (3), disseminated MAC (1), toxoplasmosis (1) and recurrent pneumonia (1). Viral load suppression was achieved in 78% of patients, only after an average of 5 years in care. STDs were common: syphilis (10), herpes simplex (10), gonorrhea (2), chlamydia (1) and trichomoniasis (3). Among parasitic infections, strongyloidiasis was the most frequent observed 8/27. Two patients were linked to care by a traditional healer.

### Conclusions:

Garifuna patients in the Bronx present for care with LHP and a high burden of conditions of public health importance. Delayed diagnoses and viral load suppression lead to higher mortality, morbidity, costs and greater HIV transmission within the community. New approaches to testing and linkage are needed to incorporate culturally sensitive resources, such as traditional healers, in order to expand testing acceptance, and link seropositive members to care earlier, leading to substantial improvements in community health.

## Resident / Fellow Research

<p><b>Author: Carlos Gongora, MD</b> Additional Authors: Carlos A. Gongora 1, Abel Casso Dominguez 1, Chirag Bavishi 1, Jacobo Pena 2, Patricia Chavez 1, Shilpkumar Arora 1, Hafeez Ul Hassan1, Pedro Moreno 2, Jacqueline Tamis-Holland 1. 1 Mount Sinai Roosevelt and Mount Sinai St. Luke's. New York, NY, 2 Mount Sinai Hospital. New York, NY Institution: Mount Sinai Roosevelt and Mount Sinai St. Luke's</p> <p><b>Title: The Long Term Clinical Outcomes with Drug Eluting Stent vs Coronary Artery Bypass Surgery for the Treatment of Left Main Coronary Artery Disease</b></p> <p>Background Percutaneous coronary intervention (PCI) with drug eluting stenting (DES) is an appropriate alternative to coronary artery bypass graft (CABG) for the treatment of left main coronary artery disease (LMCAD). Previous meta-analysis have shown similar short term outcomes with DES vs CABG. There is less information regarding the long term follow-up of these patients. Recent clinical studies have reported on the long term results with DES and CABG. We hypothesized that the long term use of DES would yield similar outcomes to CABG for the treatment of patients with LMCAD.</p> <p>Methods: We performed a meta-analysis to summarize the up-to-date evidence on this subject. We included studies comparing clinical outcomes for DES vs LMCAD with results at 3 to 7 years. We examined the following endpoints: Mortality, myocardial infarction (MI), target-vessel revascularization (TVR), and stroke. In addition we examined the rates of major adverse cardiac events (MACE). MACE was defined according to the definition used by each of the included studies. Pooled risk ratios (RR) and their 95% confidence intervals (CI) were calculated for all the clinical outcomes using a random-effect model.</p> <p>Results A total of 11 studies met our search criteria and were included in the analysis. Among these studies 4520 patients were treated with DES and 3375 were treated with CABG. There was no significant difference in MI or Stroke in the group of patients treated with DES as compared with those treated with CABG, although there was a trend toward a lower long term mortality with DES. TVR was lower with CABG. (Figure 1). In addition, there was no significant difference in MACE rates (RR: 1.12; 95% CI: 0.86 to 1.46; p 0.39).</p> <p>Conclusion Our findings suggest that DES is a safe alternative to CABG in patient treated with LMCAD with acceptable long term outcomes.</p>	<p><b>Author: Carlos Gongora, MD</b> Additional Authors: Carlos A. Gongora 1, Abel Casso Dominguez 1, Chirag Bavishi 1, Jacobo Pena 2, Patricia Chavez 1, Shilpkumar Arora 1, Pedro Moreno 2, Jacqueline Tamis-Holland 1. 1 Mount Sinai Roosevelt and Mount Sinai St. Luke's. New York, NY, 2 Mount Sinai Hospital. New York, NY Institution: Mount Sinai Roosevelt and Mount Sinai St. Luke's</p> <p><b>Title: Clinical Outcomes of Patients Treated With First Generation Vs. New Generation Drug-Eluting Stent for Left Main Coronary Artery Disease: A Meta-Analysis.</b></p> <p>Background The second generation drug-eluting stents Everolimus and/or Zotarolimus (S-DES) have been proven to be superior to the first generation drug-eluting stents Sirolimus and/or Paclitaxel (F-DES) when treating non-left main coronary artery lesions. Previous studies have come to inconsistent results regarding the efficacy of these two stent types in patients treated for unprotected left main disease (ULMD). We hypothesized that the use of S-DES would results in better outcomes than F-DES in patients with ULMD.</p> <p>Methods We performed a meta-analysis to summarize the up-to-date evidence on this subject. We included studies comparing clinical outcomes for S-DES vs F-DES in patients with ULMD. We examined the following endpoints: Mortality, myocardial infarction (MI), and target-vessel revascularization (TVR). In addition we examined the rates of stent thrombosis and major adverse cardiac events (MACE). MACE was defined according to the definition used by each of the included studies. Pooled risk ratios (RR) and their 95% confidence intervals (CI) were calculated for all the clinical outcomes using a random-effect model.</p> <p>Results A total of 4 studies met our search criteria and were included in the analysis. Among these studies 701 patients were treated with F-DES and 753 patients were treated with S-DES. There was no significant difference in all-cause mortality or MI in the group of patients treated with S-DES as compared with those treated with F-DES. TVR was lower with S-DES. (Figure 1). In addition, S-DES was associated with lower MACE rates (RR: 0.63; 95% CI: 0.48 to 0.82; p &lt;0.05) and there was a trend toward a lower rate of stent thrombosis (RR: 0.40; 95% CI: 0.13 to 1.20; p 0.10) with S-DES.</p> <p>Conclusion The use of S-DES for the treatment of LMCAD is associated with improved clinical outcomes as compared with F-DES. S-DES should be the stents of choice when treating patients with ULMD.</p>
--	---

## Resident / Fellow Research

<p><b>Author: Sonam Kiwalkar, MD</b> Additional Authors: Suzana John, MD; Tina Brar, MD; Michael Henderson, MD Institution: Rochester General Hospital</p> <p><b>Title: IPODS TRIAL: INTERVENTION TO PROMOTE OSTEOPOROSIS SCREENING WITH DXA SCAN</b></p> <p>Purpose: Osteoporosis is the most common metabolic bone disease in the US affecting more than 54 million adults over the age of 50 years. Despite its adverse effects on general health, it is often overlooked in large part because it is often clinically silent before manifesting in the form of fracture. The aim of this trial was to improve provider awareness and screening for osteoporosis in postmenopausal women above the age of 65 years, using DXA scanning in inner-city practices of Rochester, NY.</p> <p>Methods: This was a cohort, retrospective and prospective study assessing pre and post intervention data on ordering of DXA scans in all females above the age 65 years, between November 2013 to March 2015. The intervention included a one-time educational lecture aimed towards primary care providers on diagnosis and treatment of osteoporosis. This was supplemented by staff message reminders sent by investigators every 5 weeks. The study was based in outpatient clinics of Rochester General Hospital, Rochester NY. Primary analyses compared rates of ordering DXA scan before and after implementation of educational session. Rate of completion of DXA scan in the population where it was ordered was computed and patient non-compliance was also explored. Secondary analyses looked at number of patients receiving appropriate treatment as per DXA scan results, as well as calcium and vitamin D supplementation. Barriers to ordering a screening DXA by providers were also computed by provider based survey.</p> <p>Results: The primary analyses included rate of screening in eligible women before the intervention, which was 15.6%. After the intervention, it increased to 49.8%, which was statistically significant (p-value: 0.0001). The rate of DXA completion before intervention was 11%, and after intervention was 23.6% (p-value: 0.0001). Patient non-compliance was 29.7% vs. 44.4% (p=0.01) before and after the intervention respectively.</p> <p>The use of Vitamin D/calcium supplements in post-menopausal women above the age of 65 years, prior to the intervention was 41.7% and after the intervention went up to 70.5% (p-value: 0.0001).</p> <p>Per the survey, most providers felt that time constrain was an important barrier in limiting ordering of DXA scans.</p> <p>Conclusions: The IPODS trial intervention of education and periodic reminders resulted in a significant increase in DXA scan ordering. Further studies are required to assess the lasting effect of the intervention and to analyze barriers faced by non-complaint patients. This is being addressed in IPODS phase 2 trial.</p>	<p><b>Author: Sateesh Kotturi, MD</b> Additional Authors: Shikha Singh, MD Arjun Ohri( MS IV) Shashank Agarwal, MD Vijaypal Arya, MD, FACP, FACG, AGAF Institution: Wyckoff Heights Medical Center</p> <p><b>Title: The Prevalence and Risk Factors for HCV Infection in 'Baby Boomers'- A Retrospective Analysis.</b></p> <p>Introduction: In the United States, hepatitis C virus (HCV) is the second most common cause of chronic liver disease after non alcoholic fatty liver disease. The majority of patients diagnosed with HCV infection have some identifiable risk factor that increases the likelihood of exposure to the virus. However, current CDC recommendations are one-time screening for "asymptomatic" adults in the US born between 1945 and 1965. Apart from resistance from some patients, this raises ethical issues for the physicians, adds to the psychological stress in the asymptomatic patient population along with adding to the financial burden on the health system. We hypothesized that universal screening for Hepatitis C in baby boomers without any risk factors for HCV, may be an unnecessary burden and hereby presenting the initial remarkable findings of our ongoing project.</p> <p>Materials and Methods: The study was approved by Institutional Review Board at Wyckoff Hospital Medical Center, Brooklyn, NY. We retrospectively reviewed the charts of all baby boomers that visited our facility in NY in between June 1, 2013- June 30, 2015 and were offered testing for anti HCV antibody/ HCV RNA. Baby boomers routinely tested for complete hepatic profile in the clinic simultaneously were reviewed as well. Information collected included the date of birth, complete hepatitis panel, hepatitis C RNA viral load, genotype and HCV risk factors. The prevalence and risk factors stratification was performed and data was analyzed to particularly assess the prevalence of HCV with respect to their risk factors.</p> <p>Results: A total of 411 baby boomers were offered complete hepatic panel. Only 280 patients had HCV testing done. The overall prevalence of anti HCV antibody in the study subjects was 28.9% (81/280). Out of the 71.07 % (199/280) subjects that were seronegative for anti HCV Ab 85.9% (171/199) had no risk factors for HCV infection after detailed medical history interview. Only 0.02%(2/81) patients had no risk factor for HCV infection but were seropositive for HCV infection whereas 92.5 % of seropositive baby boomers did report exposure to risk factors in the past.</p> <p>Conclusion: Baby boomers with no risk factors associated with HCV have much lower prevalence of HCV infection and the baby boomers having risk factors have a markedly higher prevalence of HCV infection. The necessity of HCV testing in baby boomers in relation with meticulous history taking for risk factors has been less explored. This study highlights the need for meticulous screening for risk factors during patient encounters so as to reduce the psychological burden and efficient utilization of the health care resources. It also reinforces the imperative need of further studies to assess the relevance of testing all baby boomers for HCV in the absence of the risk behaviors.</p>
--	--

## Resident / Fellow Research

<p><b>Author: Hong Seok Lee, MD</b>                  Additional Authors: Amrut Savadkar, M.D., Khatuna Kadeishvili M.D. Theodore Lenox M.D., Alexander Sy M.D.                  Institution: Metropolitan Hospital center, new york medical college</p> <p><b>Title: PREVALENCE OF HYPERTENSION AND DIFFERENCE OF BLOOD PRESSURE CONTROL BETWEEN METABOLICALLY OBESE NORMAL BODY WEIGHT (MONW) AND METABOLICALLY HEALTHY OBESE(MHO) IN HIV POPULATION</b></p> <p>Objective: The differences between metabolically obese obesity and metabolically healthy obese persons in terms of prevalence and factors related to hypertension control have not been investigated thoroughly. So, in this study, prevalence and related factors were analyzed.</p> <p>Background: Metabolic abnormalities which are usually associated with obesity do not affect all obese people. The subset of obesity comprised of metabolically obese, normal weight individuals and metabolically healthy but obese. Metabolic abnormalities could be caused by both HIV infection itself and antiretroviral therapy. MONW in HIV population could be prevalent, therefore, healthier lifestyles and strategies specifically addressed to diminish cardiovascular complication will be needed for preserving the overall health in aging HIV-infected persons.</p> <p>Method: 468 HIV patients from 2012 to current at Metropolitan Hospital Center were analyzed regarding demographic factors, chemistry test results. This is a cross-sectional survey. Metabolic syndrome was defined following National Cholesterol Education Program's Adult Treatment Panel (NCEP's ATP) III definition. We applied the JNC 8 Hypertension Guidelines for blood pressure goal. Demographic, anthropometric factors and chemistry laboratory results were collected from Electronic medical record and used. Institutional review board and New York Medical College approved this study. There is no disclosure. The data were analyzed by SAS Ver. 9.4.</p> <p>Results: Interestingly, uncontrolled blood pressure was significantly associated with MONW compared to MHO. In addition, female and high LDL was also associated with uncontrolled blood pressure.</p> <p>Conclusion: Even if HIV patients were not obese, however, metabolically obese normal body weight patients may need more intensive blood pressure treatment and monitoring. Furthermore, it may suggest obesity paradox in HIV population. By the JNC 8 guidelines 8, HIV population with normal body weight metabolically obesity requires close and more frequent check-up for blood pressure.</p> <p>Key words: MONW, MHO, HIV, Obesity , Blood pressure</p>	<p><b>Author: Hong Seok Lee, MD</b>                  Additional Authors: Belen Nunez, M.D., Ferdinand Visco, M.D., Savi Mushiyevev, M.D., Gerald Pekler, M.D.                  Institution: Metropolitan hospital center, HHC</p> <p><b>Title: EVALUATING EJECTION FRACTION AND OBESITY IN A HEART FAILURE PROGRAM PREDOMINANTLY COMPOSED OF A BLACK AND HISPANIC POPULATION</b></p> <p>Objective: To relate the obesity paradox to ejection fraction and obesity</p> <p>Background: The obesity paradox remains controversial in the literatures. Obesity has detrimental effects on heart failure, but has been found to be paradoxically associated with improved survival.</p> <p>Method: This is a cross-sectional study. We analyzed 732 patients who were enrolled in our heart failure program and excluded those who did not follow up or patients discharged from the cardiology clinic. 688 patients who have been followed since 2013 were included. Using ACC/AHA guidelines, heart failure is classified as a reduced ejection fraction (HFrEF, EF &lt;40), preserved ejection fraction (HFpEF, EF &gt;50) and heart failure with an improved ejection fraction (HFpEF(i), EF = 40). BMI was classified according to NCEP-ATP III. Basic biochemical data, and biophysical data were collected from electronic medical record. Institutional review board and New York Medical College approved this study. All variables were analyzed by SAS Ver. 9.4.</p> <p>Results: The number of normal weight (BMI &lt;25kg/m<sup>2</sup>), overweight (30 kg/m<sup>2</sup> &gt; BMI = 25kg/m<sup>2</sup>) and obesity (BMI = 30kg/m<sup>2</sup>) were 250(35.7%), 242(35.1%) and 196(29.1%) respectively. The number of patients in our selected populations of HFrEF, HFpEF and HFpEF(i) were 456(67.9%), 136(20.2%) and 80(11.9%) respectively. A preserved EF had a significant P-value significantly associated with the overweight group compared to our normal weight group. In addition, the absence of diabetes mellitus, an ICD, no prior cardiac catheterization and age over 65 were associated with a preserved EF.</p> <p>Conclusion: The obesity paradox applied to our study group. The overweight group had a higher percentage of patients with a preserved ejection fraction compared to the normal weight group. Factors favoring a preserved EF were different among our three BMI groups. Targeted management of related factors in heart failure could lead to different approaches in the future treatment of heart failure.</p>
---	---

<p><b>Author: Evan Siau, MD</b> Additional Authors: Gilbert Brovar, MD Institution: St Barnabas Hospital, Internal Medicine</p> <p><b>Title: Prevalence of Abnormal Hemoglobin A1c and HOMA Insulin Resistance Index in the Adult U.S Population From 2000 to 2012</b></p> <p>Purpose for study To study the trend of glycemic control status in adult U.S. population from 2000 to 2012.</p> <p>Methods Prevalence estimates of abnormal glycohemoglobin (A1c) level and Homeostatic Model Assessment (HOMA) index were estimated in adults(=20 years of age) from the National Health and Nutrition Examination Survey (NHANES) 2000-2012, a survey that examines nationally representative samples of the non-institutionalized, civilian U.S. population. HOMA index, the approximating score of insulin resistance, were derived from fasting insulin and glucose levels. The thresholds used for A1c were 5.7% and 6.5%, and for HOMA Insulin Resistance index were 3 and 6. The prevalence rates were age-adjusted using U.S. Census 2000 data. Statistical analyses were performed using SAS 9.4.</p> <p>Results The age-adjusted prevalence of abnormal A1c (=6.5%) from 2000 and 2004 to 2009 and 2012 increased from 8.01% (95% Confidence interval [CI]: 7.34%-8.67%) to 11.67% (95% CI: 10.86%-12.49%). During this period the prevalence of pre-diabetic A1c (5.7%-6.4%) increased from 15.25% (95% CI: 14.43%-16.06%) to 27.48% (95% CI: 26.43%-38.53%). The prevalence of increased HOMA index also increased during this period, prevalence of HOMA =6 increased from 12.98% (95% CI: 11.70%-14.25%) to 20.37% (95% CI: 18.74%-22.01%) and prevalence of HOMA 3-5.9 increased from 24.73% (95% CI: 23.11%-26.36%) to 31.80% (95% CI: 29.99%-33.62%). Similar findings of increased prevalence rates were observed from 2000 and 2004 to 2005 and 2008. These trends varied by sex and race/ethnicity.</p> <p>Conclusions The increasing trend of abnormal A1c and HOMA index indicate increasing prevalence of diabetes and insulin resistance in the U.S. population and support earlier predictions on the nature of diabetes epidemic.</p>	
--	--