

2015 ACP Colorado Chapter Meeting- February 5, 2015 thru February 7, 2015  
Broadmoor Hotel, Colorado Springs, Colorado



# RESIDENT ABSTRACTS

**PRESENTED:  
FEBRUARY 6, 2015**

**Name:** Max Bastow, MD

**Presentation Type:** Oral

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Aaron Calderon, MD, FACP

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**Abstract Title: Rare Colitis in a HIV Positive Patient on HAART**

**Abstract Information:**

**Case Presentation:** A 50-year old HIV positive male was admitted with a 10 day history of constipation and abdominal pain. Prior to this he was in his normal state of health with no GI complaints. His pain was so severe that he self-induced vomiting after eating to relieve his pain and distention. Abdominal CT scan revealed diffuse colonic thickening especially affecting the sigmoid colon where there was also concern for an intramural abscess. The initial differential diagnosis was broad and included malignancy, IBD, and opportunistic infections. His CD4 count on admission was 371 cells/uL and he was actively being treated with highly active antiretroviral therapy (HAART). Further workup including sigmoidoscopy and EGD revealed a sigmoid stricture with biopsy showing inflammatory changes and no cytomegalovirus (CMV). His symptoms continued despite bowel rest and NG tube decompression, and he continued to complain of constipation. Repeat CT one week after admission demonstrated similar colonic thickening, but a new 5cm abscess. The abscess was not drained due to a perforation risk, so he was treated with levofloxacin and metronidazole. He failed to respond to medical management and ultimately required a diverting loop colostomy. Subsequently, he underwent repeat endoscopy through the loop colostomy which revealed deep ulcerations proximal to his obstruction that were concerning for Crohns disease. Biopsies of these lesions were positive for CMV. Consequently, he was treated with IV gancyclovir for 2 weeks followed by oral valgancyclovir for 4 weeks. Repeat biopsy after treatment showed resolution of CMV; however his stricture persisted and eventually had to be surgically removed.

**Discussion:**

CMV colitis is an opportunistic infection in HIV positive patients usually affecting those with CD4 counts <50 cells/uL. CMV rarely causes colitis in HIV patients with higher CD4 counts, however this usually occurs in the setting of a new diagnosis of HIV or in patients who have yet to be treated with HAART. While it has also been reported in patients on HAART, typically CD4 counts are severely depressed to < 50 cells/uL. Since the advent of HAART, the rate of this once common disease has greatly decreased. It has been shown that the use of HAART results in reconstitution of the immune response against CMV, independent of the CD4 count. Our patient is unusual because he developed CMV colitis while on HAART with a reasonably preserved CD4 count. Furthermore, he presented with constipation instead of diarrhea.

This case highlights that CMV colitis can present with protean manifestations and cause significant disease even in a patient on HAART with a relatively preserved CD4 count. CMV colitis needs to be considered in the differential diagnosis of any HIV positive patient with GI complaints regardless of the use of HAART and irrespective of the CD4 count.

**Name:** Sarah Dill, MD

**Presentation Type:** Oral

**Residency Program:** University of Colorado Health Science Center

**Additional Authors:** Sarah Stella, MD

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**Abstract Title: A Grave Case of Weakness**

**Abstract Information:**

Acute onset weakness may have a variety of different etiologies, requiring a broad differential with consideration of neurologic, infectious, rheumatologic, metabolic and even endocrine derangements. A 41-year-old Hispanic gentleman with a history of seizures and alcohol abuse, presented with acute onset lower extremity weakness. On the morning of admission, he reported a fall while attempting to get out of bed and was subsequently unable to stand for several minutes. He had a second episode later that morning after walking to a bus stop, in which his legs became acutely weak, precipitating a fall and prompting subsequent evaluation in the Emergency Department. The weakness was associated with pain and numbness in both legs. He also noted several weeks of palpitations, diarrhea, unintentional weight loss, and feelings of anxiety. Physical examination demonstrated an anxious gentleman who appeared younger than his stated age as a result of his smooth complexion. He was tachycardic and had a 1-2 Hz resting tremor. The remainder of his neurologic exam was unremarkable. Thyroid was normal in size without appreciable bruit. Initial labs revealed a potassium of 2.0mEq/L, and magnesium of 1.2mg/dL. TSH was notably low at <0.01mIU/L, with free T4 of 5.07pmol/L. An electrocardiogram showed sinus tachycardia with first degree AV block. A presumptive diagnosis of thyrotoxic hypokalemic periodic paralysis (THPP) was made, with suspected underlying Graves' disease, (later confirmed with positive thyroglobulin stimulating antibodies). Initial treatment involved potassium repletion; although as the problem results from an intracellular potassium shift, the process of repletion requires caution to avoid overcorrection. The patient's thyrotoxicosis was managed acutely with methimazole, as well as propranolol as the addition of beta blockade has been shown to prevent recurrent attacks of paralysis. THPP is fairly well described in Asian populations with an incidence of 1.8-1.9% compared with only 0.1-0.2% in North America. Hispanics and Native Americans are also at increased risk. The pathophysiology of THPP results from combined thyroid and adrenergic stimulation of the membrane associated sodium-potassium ATPase, resulting intracellular potassium shifts, especially into myocytes. This effect is potentiated by insulin, exercise, and androgens, (possibly explaining the higher incidence in males). Proximal muscles are usually more affected than distal muscles, and patients often report preceding muscular stiffness and/or cramping. Respiratory and bulbar muscles and bowel/bladder function are generally unaffected. A mildly elevated CK may be seen, especially if exercise precipitated the event. This patient had first degree AV block, but other cases have been associated with life threatening arrhythmias, depending on the degree of metabolic derangement. This case demonstrates an unusual manifestation of a comparatively common endocrine disorder, and highlights the integration of history and physical examination in arriving at the appropriate diagnosis.

**Name:** Michelle Kowalis, DO

**Presentation Type:** Oral

**Residency Program:** Parkview Medical Center

**Additional Authors:** Johnny Cheng, DO, Benjamin Ruckman, DO, Alexandria Nowacyk, DO, Jade Norton, DO

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## **Abstract Title: ACE Inhibitor-induced Angioedema of the Small Bowel**

### **Abstract Information:**

**Introduction:** Angiotensin-converting enzyme (ACE) inhibitors are routinely prescribed for patients with hypertension, coronary artery disease (CAD), heart failure with systolic dysfunction, diabetes and chronic kidney disease (CKD). This category of medications is also the leading cause of drug-induced angioedema, occurring in 0.1% to 0.7% of patients. Typically this adverse effect presents with swelling of the lips, tongue or face; however, swelling can also affect the intestinal tract causing episodic abdominal pain. The differential for episodic abdominal pain with nausea and vomiting is extensive and in this specific case included: small bowel obstruction, celiac disease, irritable bowel syndrome, inflammatory bowel disease, malignancy and gastroparesis. In this case, we discuss an unusual presentation of angioedema related to ACE inhibitor use.

**Case Presentation:** A 49-year-old female presented for evaluation of worsening abdominal pain starting in the left upper quadrant and radiating to the entire abdomen. For six weeks prior to presentation, the patient noted episodes of cramping abdominal pain associated with nausea, vomiting, and severe bloating. When present, the episodes would last for approximately three days, then resolve. The patient reported bloating so severe that she was unable to button her pants. Over the course of the next 12 months, the episodes increased in frequency and intensity.

Past medical history: hypertension, depression/anxiety and a hiatal hernia.

Surgical history: abdominal hysterectomy 20 years prior.

Family history: colon cancer in both mother and father.

Medications: Lisinopril 20mg once daily, started 2 years prior.

Work up included evaluation by gastroenterology (EGD, colonoscopy), multiple lab studies and imaging studies (CT abdomen/pelvis, enhanced; abdominal x-rays; small bowel follow through). The patient experienced symptoms for a total of 15 months, at which point her ACE-inhibitor was stopped. Her symptoms resolved and did not return, suggesting angioedema caused by her ACE inhibitor. This was likely seen in her initial abdominal CT as borderline wall thickening of distal small bowel loops, at that time considered possible enteritis or inflammatory bowel disease.

**Discussion:** JNC 8 guidelines suggest thiazide diuretics, calcium channel blockers, ACE inhibitors and ARBs as first line treatment for hypertension. Currently, there are more than 40 million Americans taking an ACE inhibitor, accounting for 35% of all prescriptions written for hypertension medications in the United States. As this number continues to grow under JNC-8 recommendations, along with treatment for CAD, CKD, diabetes and heart failure, it would be expected that angioedema will occur more frequently. While angioedema involving the face is more evident and easier to diagnose, angioedema of the small bowel must be considered in evaluation of all hypertensive patients on ACE inhibitors reporting abdominal pain.

**Name:** Scott Milch, MD

**Presentation Type:** Oral

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Aaron Calderon, MD, FACP

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## **Abstract Title: Don't Be Fooled By the Hemoglobin A1C**

### **Abstract Information:**

**Introduction:** Hemolytic anemia reduces the life span of RBCs and may lead to a falsely low HbA1c. We present a case of an incorrectly interpreted low HbA1c which led to medication management changes resulting in an ICU admission for hyperglycemic hyperosmolar syndrome (HHS).

**Case:** A 75 man with a PMH of type 2 diabetes and anemia presented to the hospital with the chief complaint of feeling poorly and was found to have HHS with an initial glucose of 849 that resolved with fluids and insulin. He was also discovered to have anemia with a hematocrit of 33.6% and an indirect hyperbilirubinemia. Further studies revealed an LDH of 403 U/L and a reticulocyte count of 10.2%, both consistent with hemolysis. He underwent an extensive workup for hemolysis that was unrevealing including a peripheral smear, a negative Coombs test, a normal glucose-6-phosphate dehydrogenase level, normal hemoglobin electrophoresis, and a normal urine hemosiderin. His blood sugars normalized on subcutaneous insulin and his hematocrit was stable prior to discharge.

Review of his outpatient medical record revealed that his HbA1c varied significantly over the last year (4.7% to 9.7% to 4.4%) and was 4.4% five months prior to presentation leading to discontinuation of his insulin regimen despite having high blood glucose levels. No other inciting event for his HHS was discovered.

**Discussion:** HbA1c is commonly used to estimate average blood glucose levels over the previous 3 months. Conditions that extend the life of the erythrocyte or result in decreased red cell turnover (i.e. iron or B12 deficiency, asplenia) expose RBCs to glucose for a prolonged period of time, resulting in higher HbA1c levels. In contrast, conditions that reduce the life of RBCs or result in increased red cell turnover (i.e. hemolytic anemia, acute/chronic blood loss, splenomegaly) shorten the exposure of erythrocytes to glucose, resulting in lower HbA1c levels.

Because the HgA1c currently plays such a vital role in the diagnosis and management of diabetes, it is imperative that clinicians recognize clinical scenarios that may falsely decrease or increase the HgA1c. As in our patient who had several outpatient glucose readings that were significantly elevated, clinical suspicion should be raised when HbA1c readings have dramatically changed from prior readings or when values do not seem to correlate with recently recorded glucose levels. Furthermore, caution should be used when medication adjustments are being made in these clinical scenarios. Failure to do so, will either result in undertreatment and an increased risk of HHS/DKA, as well as, long-term diabetes sequelae, or overtreatment and the risk of hypoglycemia.

**Name:** Aaron Strobel, MD

**Presentation Type:** Oral

**Residency Program:** University of Colorado Health Sciences Center

**Additional Authors:** David Elison, Brent Wagner, MD

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**Abstract Title: Pneumonic Plague: A Misdiagnosis of CAP with Pseudomonas Luteola Bacteremia**

**Abstract Information:**

**Case Description:**

42 year old male presented to an outside hospital with productive cough, fevers, and chest tightness, starting 6/28. On 6/30 at outside hospital he was febrile to 40.2, mildly tachycardic, with WBC of 22.5, and a right lower lobe infiltrate on CXR. He was started on ceftriaxone and azithromycin for CAP. His blood cultures grew out Pseudomonas luteola. He was switched to meropenem/levofloxacin for 7 days. He remained febrile and worsening oxygen requirement to 10L. CT-chest demonstrated bilateral ground glass opacities, at which point he was transferred to University MICU. On arrival Tm 39, pulse 99, respirations 34, and an oxygen saturation of 94% on 10L. Physical exam with accessory muscle use and coarse rales bilaterally. Given his sepsis, respiratory compromise and presumed ARDS he was urgently intubated, begun on broad spectrum coverage with vancomycin, piperacillin/tazobactam, streptomycin, and voriconazole, and underwent bronchoscopy with bronchoalveolar lavage (BAL). Blood, sputum, urine, BAL, fungal cultures, zoonotic pathogens and respiratory viral panel were all negative. The original positive blood sample was sent to the Colorado Department of Public Health, where it was determined to be Yersinia pestis. After a 10 day course of streptomycin, his ARDS recovered and he was extubated on 7/17, and discharged home on 7/23.

**Discussion:**

Since the invention of antibiotics, the pandemics of Yersinia pestis infection that historically killed millions have disappeared. Some 21,725 cases were reported worldwide from 2000-2009. (57 cases in U.S.)

Yersinia, a Gram negative coccobacillus, is a flea borne disease transmitted to humans primarily via mammalian hosts (rodents and house pets). Handling of infected or aerosolized animal tissues can spread the bacteria. Yersinia infection manifests as either bubonic (80-90% of cases), septicemic (10-20%), or pneumonic (rare, though most deadly) plague.

Flea bites inject the bacteria into the skin which travel to regional lymph nodes which become inflamed, creating "bubo". Fevers, malaise, weakness, headaches and painful swollen nodes. This can progress to bacteremia, sepsis, pneumonia, DIC and death in 50% if untreated, though with treatment mortality is 10-20%. Septicemic plague typically presents as advanced bubonic plague without lymphadenopathy. Pneumonic plague, with an untreated mortality of nearly

100%, presents with rapidly progressive pleuritic chest pain and dyspnea that can be fatal if proper antibiotic treatment is not instituted within 24 hours.

Diagnosis is typically made through history and positive blood culture. It is well documented that automated bacterial identifiers may misidentify *Yersinia* as other species of *Pseudomonas* or *Acinetobacter*.

Treatment is usually with streptomycin, with gentamycin as an acceptable alternative. Secondary coverage is usually added for severe disease with either doxycycline or levofloxacin.

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**Name:** Sweekriti Adhikari, DO

**Presentation Type:** Poster

**Residency Program:** Parkview Medical Center

**Additional Authors:** Camillo Franklin, MD, Kelly Buerger, DO, Kristen Scaff, DO, Johnny Cheng, DO, Benjamin Ruckman, DO, Eric Elliot, DO

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**Abstract Title: A Case of Thyrotoxic Periodic Paralysis**

**Abstract Information:**

Case Description:

A 36 year male presented to clinic with 2 year history of hip and shoulder arthralgia and upper arm and thigh myalgia. Episodes began 2 years ago and were occurring with increasing frequency which prompted his visit. The patient's current medications included Amlodipine 10 mg daily, Klor-Con 20 mEq PO daily, lisinopril 20 mg daily, and omeprazole 20 mg daily. He took potassium supplement for known hypokalemia but without known cause, which he found alleviated symptoms of muscle pains. Physical exam on initial visit revealed tachycardia with heart rate 102, 4/5 shoulder strength bilaterally, 4/5 right hip strength and 3-4/5 left hip strength as well as decreased patellar and Achilles reflexes on left.

Labs including CMP, Vitamin D, CK, ESR, TSH, and hepatitis panel were obtained and significant for a TSH of <0.01, free T4 elevated at 3.97, T3 elevated at 20.01, Potassium was normal. Vitamin D was low at 13.7. Thyroid receptor-stimulating antibody level were drawn and found to be elevated at 154%. Within short interval of lab results patient presented to ER with 8 day history of severe bilateral lower extremity weakness. His potassium was found to be 1.9, his TSH <0.01. During his admission potassium was replaced and muscle aches improved. He was diagnosed with thyrotoxic periodic paralysis. Patient was subsequently sent for Thyroid uptake and scan which revealed an enlarged and homogeneous thyroid with a pattern consistent with Grave's disease. He was treated for his Grave's disease and is now hypothyroid undergoing titration of thyroid replacement, his potassium has not been low since correcting his thyroid.

Discussion:

Thyrotoxic periodic paralysis (TTP) is a sporadic form of periodic paralysis. This differs from Hypokalemic Periodic paralysis which is autosomal dominant. TTP is commonly seen in the Asian population and presents with severe hypokalemia and profound proximal paralysis. The hypokalemia is due to intracellular shift of potassium by sensitization of the Na/K pump by thyroid hormone. This is curable once euthyroid state is achieved. It is important to diagnose this quickly as hyperkalemia can result from excessive potassium replacement.

In our case, the patient had symptoms for 2 years without a diagnosis. Given the uncommon nature of this disease and presentation usually in Asian patients TPP was not included in the differential diagnosis. Patient may have remained without severe weakness because he was taking potassium replacement. TTP is uncommon yet a disabling condition, once euthyroid state is achieved hypokalemia resolves.

**Name:** Johnny Cheng, DO

**Presentation Type:** Poster

**Residency Program:** Parkview Medical Center

**Additional Authors:** Sweekriti Adhikari, DO, Jennifer Chang, OMSIV, Kelly Buerger, DO, Brita Krabacher, DO, Marcel Junqueira, MD

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**Abstract Title: Stroke After Paraganglioma Resection**

**Abstract Information:**

Parasympathetic paragangliomas or carotid body tumors are highly vascular structures, typically found in the head and neck with 60-65% involving the carotid arteries. Patients often present with a non-tender mass, as 80-90% are nonfunctional and slow growing with a median growth rate of one mm/year. Differentials for nontender lateral neck masses should include lymphadenopathy, branchial cleft cyst, salivary gland tumor, neurogenic tumor, thyroglossal duct cyst, and parasympathetic paragangliomas. In this case, we discuss an unusual course of a paraganglioma.

37-year-old male presented for evaluation of a left neck mass. CT of the neck revealed a 6x4x2cm homogeneous mass in the sternocleidomastoid and carotid bifurcation. During excision, the mass was found to engulf the internal and external carotid arteries with adherence to deep cervical musculature. There was dehiscence of the cephalad edge of the carotid bifurcation with the tumor remaining adherent and eroding into carotid adventitia that continued to bleed. Hemostasis was controlled with Surgicel and gentle pressure. A 3.1x2.3x1.8cm portion of the tumor was removed with 800mL blood loss. After the procedure, he became aphasic with complete right hemiparesis and enlarging left sided neck mass. Repeat head and neck CT revealed left temporoparietal lobe density changes indicating acute infarction secondary to occlusion of the cervical and petrous portions of the left internal carotid artery and left cerebral hemisphere edema indicating a very large embolic stroke. Neck CT showed mass effect with displacement of the oropharynx and hypopharynx to the right of midline and narrowing of airways due to edema and hemorrhage. Patient underwent immediate intubation and craniectomy, but was not a tPA candidate. The patient was extubated without complication and his right lower extremity weakness resolved. He continued having right upper extremity weakness and aphasia and was subsequently sent to a stroke rehabilitation facility.

Paragangliomas are treated by surgical resection as they have historically been radioresistant, and fine needle biopsy is contraindicated due the highly vascular nature and risk of hemorrhage. Surgical resection should be considered for tumors that are Shamblyn stage III, greater than 5cm, have a doubling rate of 100 days or less (malignant in 6-13% of cases), carotid stenosis, and low risk for neurologic complications. Initial imaging should include CT or MRI angiography to delineate the relation of the tumor to adjacent neck structures and allow visualization of the arteries in an attempt to prevent bleeding and stroke. Given the complications associated with treatment of these tumors, we recommend a team approach with vascular surgery, ENT, and neurosurgery.

**Name:** Brittany Folks, MD

**Presentation Type:** Poster

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** None

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**Abstract Title: The Perfect Storm: A Case of Iatrogenic Hypokalemic Periodic Paralysis**

**Abstract Information:**

**Introduction**

Hypokalemic periodic paralysis occurs in various populations due to genetic mutations that result in channelopathies causing increased intracellular shifts of potassium and muscle paralysis. However, the right combination of medications in certain individuals may also create the perfect milieu to mimic this phenomenon.

**Case**

A 40 year-old African-American gentleman with neurosarcoidosis and resultant pan-hypopituitarism on thyroid hormone replacement presented with several hours of acute onset global weakness. He had been treated the previous day with high dose solumedrol for a flare of his sarcoidosis and had awoken with paralysis of all four limbs. Upon arrival to the Emergency Department, he was found to have a potassium level of 1.9. Initial exam demonstrated 4/5 muscle weakness in his bilateral upper and lower extremities without appreciable reflexes. Imaging of his brain and cervical spine were unremarkable for signs of a new acute process. Creatine kinase, calcium, magnesium, and phosphorus were all within normal limits. Thyroid stimulating hormone was found to be appropriately low in the setting of pan-hypopituitarism, however, free thyroid hormone levels were elevated. Discussion with the patient revealed a recent increase in dosage of his levothyroxine after many months of poor compliance. His potassium levels corrected without repletion to 4.9 within two hours, resulting in slow resolution of his symptoms. His potassium was closely monitored with no further decreases or recurrence of symptoms, and he was discharged the following day.

**Discussion**

Hypokalemic periodic paralysis occurs in approximately 1 out of every 100,000 persons and appears to be more prevalent in those of Asian descent. Intracellular shifts of potassium result in weakness and flaccid paralysis of skeletal muscles, usually with relative sparing of the cranial and respiratory musculature.

Thyroid disorders are thought to be a predisposing factor in susceptible persons, termed thyrotoxic periodic paralysis (TPP). TPP may be provoked by high carbohydrate meals, ingestion of alcohol, rest after strenuous exercise, trauma, and cold environments. Glucocorticoids have also been reported as a rare precipitant of TPP. It is presumed that the activity of the Na/K ATPase pump is increased in the setting of hyperthyroidism, causing excess intracellular potassium shifts. Steroids upregulate the number of Na/K ATPase pumps present in skeletal muscle. They also raise blood glucose levels which stimulates insulin release and further increases pump activity.

All previously reported cases of steroid-induced TPP have been in the setting Grave's disease. In our patient, glucocorticoids and manufactured thyroid hormone produced "the perfect storm," resulting in the first reported case of thyrotoxic periodic paralysis in the setting of iatrogenic hyperthyroidism.

**Name:** Miriam Freundt, MD

**Presentation Type:** Poster

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** R. Feyrer, M. Weyand, M. Pizon

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**Abstract Title: Impact of Surgical Ablation of Atrial Fibrillation on Atrial Natriuretic Peptide Levels in 6 months Follow-up. Can Preoperative ANP Level Predict Success?**

**Abstract Information:**

**Introduction**

Atrial fibrillation (AF) is a common supraventricular cardiac arrhythmia. Current guidelines recommend concomitant surgical ablation in patients with AF who undergo cardiac surgery for structural heart disease. Several biomarkers have been evaluated as predictors for success rate. The aim of this study was to evaluate atrial natriuretic peptide (ANP) as predictor for conversion into sinus rhythm in 6-month follow up after ablation with high intensity focused ultrasound (HIFU).

**Material und Methods**

We included 78 consecutive patients, who underwent elective cardiac surgery on cardiopulmonary bypass at the Medical Center Bayreuth, Germany. 42 patients with AF underwent concomitant surgical HIFU ablation (ablation group). 16 patients with AF underwent surgery without ablation (control group without ablation). 20 patients underwent cardiac surgery and had normal sinus rhythm (SR control group). Patients within the ablation group showed the following AF characteristics on admission: paroxysmal (11.9%), persistent (23.8%) and long standing persistent (64.3%). For HIFU ablation we used the Epicor system (St Jude Medical). ANP levels were measured 1 day preoperatively, and on post-op day (POD) 1, 7, 90 and 180. Heart rhythm was evaluated by EKG at the same time.

**Results**

Mean ANP levels (nmol/L) were as follows: In the ablation group: preop = 7.32, POD 1 = 9.34; POD 7 = 9.09; POD 90 = 7.75; POD 180 = 8.98. In the control group without ablation: preop = 7.245, POD 1 = 8.61; POD 7 = 8.175; POD 90 = 6.78; POD 180 = 7.23. In the SR control group: preop = 3.78, POD 1 = 6.95; POD 7 = 6.7; POD 90 = 5.55; POD 180 = 4.86. In the ablation group 68.4% had converted to SR at 6-months follow-up. In multivariate analysis preop ANP levels were an independent predictor of heart rhythm 6 months post procedure only in patients with longstanding persistent AF (Odds Ratio 0.733; Coefficient -0.31;  $p < 0.05$ ). Sensitivity for preop ANP level at a cut-off value of 7.5 nmol/l was 85.7%, specificity 72.7%, positive predictive value 80.0% and negative predictive value 80.0%.

**Conclusion**

These results imply that preoperative ANP level can predict outcome of surgical ablation in patients with long standing persistent AF.

**Name:** Tyler Miller, MD

**Presentation Type:** Poster

**Residency Program:** University of Colorado Health Sciences Center

**Additional Authors:** David Katz, MD, Douglas Ney, MD

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**Abstract Title: An Unusual Case of Syncope in a Lymphoma Patient**

**Abstract Information:**

**Case presentation:** A 59 year-old male with a history of diffuse large B-cell lymphoma in remission was admitted after a prolonged syncopal episode during which CPR was performed. This and other recent syncopal episodes were characterized by pain in his right neck and arm followed by lightheadedness, diaphoresis, anxiety, and then loss of consciousness. He additionally noted diplopia, difficulty using his right arm, hoarseness, and difficulty swallowing. On hospital day one the patient experienced another syncopal episode, and telemetry revealed transient heart block with a heart rate of 30. On recovery of normal conduction the patient was found to be alert and oriented. His heart and lung sounds were normal. On neurological examination his right pupil was noted to be enlarged and sluggishly reactive. Additionally, he was unable to adduct his right eye and demonstrated weakness with right ocular elevation and depression. Right palatal elevation was impaired. Strength and coordination were impaired in his right arm.

A cardiac workup including echocardiography was unrevealing, and his bradycardia was thought to be vagally mediated. He experienced further characteristic episodes of syncope necessitating pacemaker placement. Lumbar puncture revealed a mild lymphocytic pleocytosis. MRI of the brain did not demonstrate any abnormalities in the brain or brainstem, but did note a conspicuous small mass in the superior nasal cavity extending through the cribriform plate. Biopsy of the mass demonstrated intraneural invasion of atypical lymphocytes. A diagnosis of neurolymphomatosis from recurrent DLBCL was made. The patient was started on chemotherapy, and at two months' follow up he has improvement in his cranial neuropathies and regained normal use of his arm.

**Discussion:** Our patient presented with recent onset, recurrent syncope and multiple subacute cranial nerve deficits. Syncope often presents a diagnostic challenge. In patients with cancer the differential diagnosis may include several additional considerations: Seizures, carotid sinus hypersensitivity, dysautonomia, or cardiomyopathy have all been described as etiologies of syncope resulting from tumors or subsequent chemotherapy.<sup>1</sup> Drug-related causes were initially considered in our patient. However, no evidence of cardiomyopathy was found, and we learned vincristine had been withheld from his original chemotherapy due to a history of peripheral neuropathy. Rather, his episodes, which were characterized by unprovoked pain coinciding with periods of bradycardia, hypotension, and subsequent loss of consciousness, were most consistent with a vasovagal etiology. Additionally, our patient had physical signs of damage to his glossopharyngeal and vagus nerves (which overlap extensively) such as hoarseness, aspiration, and unilateral weakness of the palate. Damage to these peripheral nerves has been previously implicated as a cause of vasovagal syncope in cases of trauma,<sup>2</sup> neurosarcoidosis,<sup>3</sup> and schwannomas.<sup>4</sup> Neuropathic pain of the glossopharyngeal nerve has also been strongly associated with syncope.<sup>5</sup>

**Conclusion:** In a patient with a history of lymphoma and neurological symptoms, neurolymphomatosis, or infiltration of the peripheral nervous system by malignant cells, should be considered. Ours is the first description of neurolymphomatosis resulting in profound cardioinhibitory vagally-mediated syncope.

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- 5 Rushton JG, Stevens JC, Miller RH. Glossopharyngeal (vagoglossopharyngeal) neuralgia: a study of 217 cases. *Arch Neurol* 1981; 38: 201-5.

**Name:** Kara Mizokami-Stout

**Presentation Type:** Poster

**Residency Program:** University of Colorado Health Sciences Center

**Additional Authors:** Paul Reynolds, Gerald Barber, Larry Golightly, Michael McDermott

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**Abstract Title: Evaluation of the Appropriate Use and Safety of Intravenous Levothyroxine at an Academic Medical Center**

**Abstract Information:**

**Purpose of Study:** Intravenous levothyroxine (IV T4) provides a rapid repletion of thyroid stores and is often used in endocrinologic emergencies such as myxedema coma. The efficacy and safety of IV T4 for other clinical conditions is uncertain. Given the cost differential, hormone repletion rates, bioavailability and half-life, a retrospective study was conducted to evaluate the usage of IV T4.

**Methods:** A survey was sent to the Division of Endocrinology and University Health Consortium to establish compelling indications for IV T4 in addition to the FDA indication (myxedema coma). These included: NPO greater than 3 days, cardiogenic shock, and suspected thyroid malabsorption. Hospital in-patients receiving IV T4 were retrospectively evaluated over 6 months. Patients were assessed for presence of compelling indications for IV T4, appropriate dose reduction from oral T4, and duration of IV T4. A safety analysis was conducted to describe adverse events to IV T4 at higher risk from rapid supplementation (geriatrics, history of congestive heart failure, atrial fibrillation, or coronary artery disease). A cost analysis was performed to evaluate patients without compelling indications to IV T4.

**Results:** 76 patients were evaluated in the study period (Table 1). Among these, there were diagnoses of 5 (6%) cases of myxedema, 3 (4%) suspected T4 malabsorption, and 2 (2.5%) cardiogenic shock. Of patients without compelling indications, 49 (79%) patients had PO access while receiving IV T4. Of patients receiving IV T4, 22% were not converted correctly from their oral form. In addition, 13.5% developed atrial fibrillation and 14% developed troponin elevations while on IV T4.

**Conclusions:** Evaluation of necessity for IV T4 administration revealed that use of IV T4 was often unwarranted. Further analyses projected that implementation of an order set including standardized laboratory requests and conversions of T4 dosage forms coupled with clinical decision support aimed at limiting use of IV T4 to compelling indications could result in drug acquisition cost avoidance totaling \$50,820 per year in our hospital. A post intervention analysis is to follow.

**Name:** Kristen Scaff, DO

**Presentation Type:** Poster

**Residency Program:** Parkview Medical Center

**Additional Authors:** Kelly Buerger, DO

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**Abstract Title: A Case of Primary Hepatocellular Carcinoma in a Young Female in Absence of Identifiable Risk Factors**

**Abstract Information:**

Hepatocellular carcinoma (HCC) is the second leading cause of cancer death in the world among men and sixth for women. It is commonly associated with risk factors including hepatitis B and C, heavy alcohol consumption, cirrhosis and hereditary hemochromatosis. However, it is important to realize that this malignancy can occur in patients without any identified risk factors.

A 63 year-old female presents with cramping epigastric abdominal pain for 3 weeks. Associated symptoms include early satiety, belching, occasional regurgitation and unexplained weight loss of 9 lbs. over an eight-month period. Past medical history was positive only for asthma and arthritis. Family history revealed melanoma in her brother. Patient is a never smoker and never drinker. She worked as a Safeway clerk for 39 years and was current on all preventative measures.

Physical exam revealed a palpable firmness in the right upper quadrant (RUQ). Abdominal ultrasound revealed multiple, large heterogeneous lesions within the liver with the largest lesion measuring 9cm. An enhanced CT of abdomen and pelvis confirmed liver lesions. No further pathology or adenopathy within the abdomen was noted. Chest imaging did not reveal pathology. CT guided liver biopsy revealed poorly differentiated carcinoma of unknown primary. Follow up immunohistochemistry staining strongly positive for HepPAR-1 and Glypican-3, both of which are specific for hepatocellular carcinoma. AFP tumor marker was significantly elevated at >36,000. Laboratory studies were positive only for mildly elevated AST and alkaline phosphatase. Hepatitis panel was negative. PET CT revealed massive hypermetabolic focus localizing only to the liver. No extrahepatic activity was noted.

Patient traveled to MD Andersen for expert opinion. The case was presented at a multidisciplinary clinic where it was determined she would be a candidate for yttrium90 due to her multifocal, multilobar liver lesions. While awaiting approval for this treatment, systemic therapy with soratinib was initiated. Two weeks later, patient developed shortness of breath and was found to have acute, bilateral pulmonary emboli. Soratinib was stopped during this acute illness. One month later, patient developed right-sided pleural effusion and interval development of multiple pulmonary metastases. Liver lesions also grew in size and in number. Patient had developed ascites and edema and succumbed to her disease three months from diagnosis.

This case illustrates the importance of understanding that risk factors are only tools to guide our differential. HCC in the absence of known risk factors is rare. Although rare, it is a reminder of the importance of physical exam and keeping a broad differential diagnosis when typical risk factors are not present.

