2017 ACP Colorado Chapter Residents' Meeting

May 16, 2017

Saint Joseph Hospital, Denver, Colorado

ACP American College of Physicians
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# RESIDENT ABSTRACTS

## PRESENTED:

## MAY 16, 2017

**Name:** Jacob Ludwig, DO **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

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**Abstract Title: Pharming**

## Abstract Information:

**Case Study:**

YK is a 19 y/o female with intractable seizures who presented to the epilepsy-monitoring unit for further evaluation after failing to respond to antiepileptic drug (AED) therapy. YK's neurological issues began at 15 y/o, which included tremor, difficulty concentrating and episodic involuntary limb/torso movements. These movements progressed after starting high school often triggered by studying, focusing, reading or playing piano. At age 18, YK began complaining of worsening insomnia and depression, and her father began noticing focal seizures that would progress into generalized bilateral convulsions with loss of consciousness. YK's father noted that the seizures often occurred around the 20th of every month despite increasing doses of Levetiracetam. At age 19, YK's seizures became more frequent and she experienced worsening of symptoms that included colorful visual hallucinations. Her insomnia and depression were treated with amitriptyline and her AED medications were changed to Lamotrigine and Topiramate. But her seizures continued to be refractory despite increased AED doses.

YK then presented to the epilepsy-monitoring unit for further work-up. Throughout the first day of monitoring, she suffered numerous brief seizures, periods of hemodynamic instability, and had a negative lamotrigine serum drug level. On hospital day 2 she had a prolonged seizure aborted with ativan. A CNA then discovered numerous diphenhydramine pills underneath her in a recliner chair. The neurologist reviewed the recorded video, which revealed YK taking several handfuls, 300-500 mg at one time, of diphenhydramine pills overnight and, notably, one hour before her prolonged seizure. YK was subsequently intubated and sedated with propofol for seizure control, which was transitioned to Phenobarbital. Her AED's were increased and changed to IV formulations. She remained mechanically ventilated for several days with frequent hyperkinetic movements, hemodynamic instability, fevers and urinary retention, but without further seizure activity. Her QRS remained stable, her symptoms eventually subsided and she was successfully extubated.

YK eventually endorsed taking 5 tablets of diphenhydramine usually 3 times daily beginning at 15 y/o. Her initial motivation was insomnia, but she added daytime doses to help with allergies. She then began experiencing a "high," which prompted her to take more. In the week prior to admission, she also started taking antler tea, a Korean homeopathic remedy for her seizures. Her depression and insomnia were worsening, and her seizures were even more frequent, so she again increased her diphenhydramine intake in conjunction with suicidal ideations. She was ultimately discharged in stable condition to inpatient psychiatric care for further evaluation.

**Discussion:**

With advancements in biotechnology and imaging the ability to determine a patient's specific epilepsy etiology has improved, often identifying genetic, structural or metabolic causes. However, the etiology of up to one third of all cases remains unknown. YK's epilepsy was

associated with strange features that included visual hallucinations, altered mental status, urinary retention, hyperkinetic movements and hemodynamic instability, and was refractory to several antiepileptic drugs. When it was discovered that her seizures were from chronic daily diphenhydramine overdose the picture came together and she ultimately endorsed motivations for use that included insomnia, depression, intoxication and suicidal ideations. She was also taking amitriptyline, which undoubtedly worsened her anticholinergic state, and also had absent blood levels of lamotrigine, likely due to alterations in drug metabolism from the herbal tea. The controversial antidotal therapy of physostigmine, an acetylcholinesterase inhibitor, was not used given its risk of precipitating more seizure activity.

Diphenhydramine abuse is common, especially as treatment for insomnia and for intoxicant effect, and symptoms associated with the anticholinergic overdose are dose-dependent. With ingestion of greater than 300-1000 mg daily, symptoms can include visual hallucinations, seizures, coma and life threatening QRS or QT prolongation. “Pharming,” or the use of prescription or OTC drugs for intoxication purposes, among teens has been trending up in recent years. Recreational use of diphenhydramine for hallucinogenic effect is reported in the literature at doses of 300-700 mg. Furthermore, with chronic use, addiction ensues and withdrawal symptoms can be very distressing. YK was likely using similar doses for a year or more, and her chronic ingestion was nearly fatal. Luckily the etiology of her epilepsy remained under investigation. Through the efforts of diligent physicians, assisted by advances in technology, the cause of her seizures was ultimately determined and she is now receiving the psychiatric care that she truly needs.

**Name:** Jan Denkmann, MD **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Eric Seger, DO

**Abstract Title: A Rare Case of Motor Neuropathy**

## Abstract Information:

**Case**

Case Description

58yo male presented with a 3 week history of a rash, subjective fever and chills, night sweats and nausea for more than 48 hours. He also noted enlarged lymph nodes in in his neck for 3-4 months and an enlarging right neck mass for the past 6-8 weeks.

Past medical history was limited to untreated hypertension and depression.. He has a remote history of smoking, drinking, cocaine and marijuana abuse in the 1970's.

Physical exam was remarkable for enlarged bilateral anterior cervical lymph nodes, a diffuse vesicular rash with innumerable erythematous papules, in different stages of development including eschar, closed, pustular, and hemorrhagic lesions noted on face, scalp, torso, arms and legs. Oral mucus membranes of his lower lip demonstrated a single painful blister, and there was no conjunctival involvement. Neurologic assessment on the day of admission was significant for RUE weakness of the shoulder with 2/5 strength for abduction and extension.

Laboratory evaluation was significant for a lymphocytic leukocytosis (WBC 105K, Lymphocytes 92%) with noted smudge cells on peripheral blood smear. A peripheral blood flow cytometry showed a population of monotypic B cells with a phenotype consistent with chronic lymphocytic leukemia. Chemistry was significant for mild hyperkalemia (5.1 mmol/L), elevated creatinine (1.48 mg/dL) and elevated uric acid (7.3 mg/dL). Virology assessment included negative PCR for HIV and HSV 1 and 2. The pathologic assessment of the skin biopsy showed changes to keratinocytes consistent with VZV.

Treatment was initiated for acute VZV infection with one dose of IVIG and a 14 day course of IV acyclovir. His treatment course was complicated by multiple catheter-associated superficial thrombophlebitides and acute kidney injury on day 12 that necessitated dose reduction of acyclovir.

The patient was provided aggressive physical therapy for his motor neuropathy and regained full strength in his left shoulder. The right-sided motor neuropathy persisted, and he was discharged with home physical therapy.

Case Discussion

Immunocompromised patients are at increased risk for disseminated VZV infection. This is more commonly seen in transplant recipients on chronic immunotherapy and advanced HIV-infection. This patient presented with previously undiagnosed chronic lymphocytic leukemia, which put him at risk for disseminated infection.

Complications of disseminated VZV infection may include visceral organ involvement including disease patterns of pneumonia, hepatitis and encephalitis. The most common neurologic complications are Ramsay-Hunt syndrome and segmental limb paresis. Peripheral motor neuropathy is a relatively rare complication and thought to occur in approximately 3% of patients with VZV infection. The peripheral motor weakness is thought to result from a spread of VZV from the dorsal root ganglia to the anterior root/horn. Treatment options for VZV most commonly include intravenous vidarabin or acyclovir and PT rehab for motor strength training.

**Name:** Kristin Hesterberg, DO **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Rachel de Andrade Pereira, MS

## Abstract Title: Rare Cause of an Unresponsive Patient

### Abstract Information:

**Case**

A previously healthy 58-year-old male presented for revision of left hip arthroplasty. Surgery was prolonged with significant bone manipulation. Patient received spinal anesthesia only, without paralytics agents. Intra-operatively, he developed atrial fibrillation. Rate was controlled with esmolol and he converted back to sinus rhythm. Blood pressure remained stable throughout procedure. Intra-operatively, he received, fentanyl, propofol, lidocaine, tranexamic acid, ketamine and 2.5 liters of LR solution.

Post-operatively the patient was unresponsive but protecting his airway. CT scan was without acute abnormality. MRI showed scattered punctate foci in watershed type distribution consistent with fat emboli. Foci were also seen in the thalamus and basal ganglia. Chest X-ray showed evidence of significant bilateral interstitial prominence consistent with fat emboli associated ARDS. Transthoracic echocardiogram was negative for any significant valvular disease; bubble study was negative for patent foramen ovale. Given continued unresponsiveness, there was concern for seizure activity. Electroencephalogram showed seizure activity and intermittent status. He was started on Keppra and Ativan, without improvement in mental status. EEG showed resolution of seizure activity.

The patient showed minimal improvement, but continued to be non-verbal. He was able to open his eyes and make some vocalizations. After discussion, family decided to transition to comfort care given patient previously expressed wishes about quality of life.

**Discussion**

Fat embolism Syndrome (FES) is a rare clinical entity, classically developing 12 – 72 hours after pelvic and long bone fracture. FES has been reported rarely after pancreatitis, bone marrow transplant, liposuction and more commonly after arthroplastic surgery with significant bone manipulation. Incidence of FES after long bone fracture is reported at <1%, though other studies have cited up to 30%. FES is primarily a clinical diagnosis characterized by the triad of petechial rash, respiratory distress and neurological dysfunction. There is no diagnostic gold standard, but Guard's criteria are frequently cited to assist in diagnosis. Two theories for how FES develops have gained acceptance: The Mechanical Theory and The Biochemical Theory. The former suggests that physical obstruction of microcirculation in critical vascular beds by fat particles released into damaged venous sinusoids of the long bones or pelvis.

The latter suggests hormonal changes secondary to trauma or sepsis trigger release of free fatty acids (FFA) that cause cascading damage to vulnerable populations of cells in critical vascular beds.

Lungs are the most common organ system affected in fat emboli syndrome, with nervous system involvement less frequently observed. Cases where neurologic symptoms are the predominant finding are referred to cerebral fat embolism syndrome (CFES). Neurological symptoms have a broad range including headache, lethargy, delirium, stupor, convulsions, and coma. MRI is the most helpful imaging modality in diagnosing cerebral FES as CT is usually without acute abnormality. Diffusion weighted MRI will present with a "starfield" pattern where bright spots representing punctate foci ischemia are seen in a watershed distribution. These imaging changes are thought to represent cytotoxic edema which develops immediately after the injury. T2 weighted image finding develop later and likely represent vasogenic edema.

Mortality for FES is downtrending, likely due to advances in resuscitative care strategies, but is still upwards of 10%. Prior studies have not shown a benefit of steroids or heparin after development, though steroids may provide benefit in prevention. Management continues to be supportive care and early surgical fixation of fractures. Delayed recovery is also a common characteristic associated with cerebral FES, with >50% of cases returning to baseline functional status (need citation). Prior case reports document complete neurologic recovery at 6 or more  months.

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**Name:** Jonathan Hickmann, DO **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: Not Your Typical Tango

### Abstract Information:

**Introduction**

Non-ketotic hyperglycemia has been described as a rare cause of choreoathetoid movements. We present the case of an 87 y/o diabetic female who was found to have hyperglycemia and hyperosmolarity in the clinical setting of left upper and lower extremity chorea.

**Case Description**

87 year old female, with a history of diabetes and prior stroke, presented with abnormal and uncontrollable left arm and left foot movement for 3 days. She reported running out of her anti-hyperglycemic agents 2 months prior. Physical exam was remarkable for choreiform and ballistic movements of the left upper extremity and choreiform movements of the left foot. No other focal neurologic abnormalities were elicited. Labs were significant for a blood glucose of 647 and calculated serum osmolality of 321. The patient underwent CT of the head which was significant for increased density in the right caudate and putamen. No other abnormalities were found to explain her chorea. The patient's blood glucose was corrected with complete resolution of her choreiform movements.

**Discussion**

Non-ketotic hyperglycemic associated hemichorea is a rare cause of hemichorea/hemiballism. This syndrome is more common in elderly females of Asian descent. It can be the presenting symptom for diabetes or present after periods of poor glucose control, as was the case with our patient. Prior studies report glucose of 170-1260 mg/dl and serum osmolarity of 290-335 mOsm/l. CT imaging may show hyperdense putamen or caudate nucleus on the contralateral side to the patient's symptoms. T1 -weighted MRI images show high density signal intensity in the basal ganglia on the contralateral side. T2- weighted images have more variability in findings. The changes seen on imaging usually resolve after resolution of symptoms. There are reports of imaging changes in the absence of hemichorea-hemiballism, but with hyperglycemia.

The mechanisms for this syndrome is unknown, though several theories exist. Hyperviscosity leading to neuron and neurotransmitter dysfunction, specifically GABA, may result in uncontrolled movements. One SPECT study showed decreased perfusion of the striatum contralateral to the affected side. Some have proposed petechial hemorrhages or myelinolysis in the affected areas, resulting in movement disorder.

Management involved normalizing blood glucose. In the majority of cases, treatment of the hyperglycemia results in complete resolution. In refractory cases, postsynaptic dopamine antagonists, such as atypical antipsychotics may ameliorate the symptoms. Although not well studied, medications with GABA antagonism, such as benzodiazepines have been used. Repeated episodes may result in permanent movement disorders.

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**Name:** Carly Hurley, MD **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: A Tale of Deceitful Hemodynamic Stability

**Abstract Information:**

Case: A 66-year-old male with chief complaint of dyspnea on exertion, lower extremity swelling, and decreased urine output is found to have renal failure and bilateral deep vein thrombosis (DVT). He denies chest pain. Vitals: initial tachycardia resolves with 2L oxygen, normotensive, respiratory rate of 32. IV heparin is started. Labs: Pro-BNP 24,993; troponin 0.09 and lactate 5.5. Echocardiogram reveals large mobile thrombus in the right atrium. Pulmonology, nephrology, interventional radiology, and cardiology discuss treatment options including systemic tissue plasminogen activator (TPA), catheter directed TPA, and inferior vena cava filter. Given patient's clinical stability, anticoagulation is continued and a lasix drip started for hypervolemia with plans for thrombolysis should patient become hemodynamically unstable.

The following morning patient has a pulseless electrical activity arrest. Bedside ultrasound shows no pericardial effusion, pneumothorax, or right atrial thrombus. Patient is declared dead after 50 minutes of resuscitation efforts, including TPA.

Discussion: There are no treatment guidelines for right atrial thrombus (RAT) in hemodynamically stable patients. UpToDate recommends considering thrombolysis on a case by case basis. Consideration is particularly recommended when "patients develop signs of deterioration, including increasing tachycardia, clinical signs of shock, worsening right heart dysfunction, worsening blood pressure, significant hypoxemia," which were not present in our patient. [9]

Torbicki et al analyzed an international pulmonary embolism (PE) registry, including 1,011 patients with PE and echocardiogram evaluation. 37 patients had RAT with PE. Despite treatment, fourteen-day mortality was 23.5%, 20.8%, 25% among those with RAT with PE treated with heparin, thrombolysis and embolectomy, respectively, vs 11% with isolated PE.[6] Rose et al analyzed 177 cases of RAT and showed mortality rates: 100% no therapy (n=16), 29.6% heparin alone (n=35), 23.8% surgical embolectomy (n=63), and 11.3% thrombolysis (n=62) suggesting lower mortality with thrombolysis.[3] Unfortunately, 2016 CHEST guidelines regarding antithrombotic therapy in venous thromboembolism fail to address RATs.[8]

Several studies demonstrate success with novel interventions.[1,4,5] The AngioVac is a percutaneous device used with venovenous bypass to remove thrombi. Donaldson et al evaluated the AngioVac in 14 consecutive cases between April 2010 and July 2013 at Massachusetts General Hospital. Intracardiac thrombi were present in 11 of the 14 cases; all thrombi were completely removed. Two patients did not survive to discharge.[1] Resnick and Salsamenid both reviewed 7 cases where AngioVac was used for thrombectomy and also found promising results.[4][5]

Despite numerous therapeutic options, there are no guidelines for optimal management of RAT. In her editorial in Insights in Chest Disease, Bushra et al highlights the challenging situation posed by RAT. They recommend multidisciplinary evaluation of treatment options based on clinical experience and available resources.[7] Unfortunately, this strategy was ineffective in our case. Further literature review, randomized control trials, and ultimately evidence based guidelines would aide management of RAT.

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**Name:** Ajay Major, MD **Presentation Type:** Oral Presentation

**Residency Program:** University of Colorado School of Medicine

**Additional Authors:** Melver Anderson III, MD, FACP

## Abstract Title: The Exceptionally Rare Coumadin Associated Side Effect of Cholesterol Embolization

### Abstract Information:

Introduction:

Cutaneous squamous cell carcinoma (cSCC) is the second most common cancer in the United States and has a significantly increased risk of recurrence and metastasis as compared to basal cell carcinoma, with a five-year recurrence rate of 8% and a five-year metastasis rate of 5%. However, not all cutaneous squamous cell carcinomas are alike. The size and location of the primary lesion in cSCC confers a widely variable risk for recurrence and metastasis.

Case Summary:

A 69-year-old man presented with one week of worsening constipation, nausea, vomiting, and abdominal pain, as well as a new headache and an unintentional 30-pound weight loss over the past year. His past medical history was significant for prostate cancer treated with prostatectomy and a history of multiple skin cancers on his scalp, face, ears and arms.

Vital signs were within normal limits. Physical exam demonstrated a 2.5 cm ulcerated, hyperkeratotic lesion with a cutaneous horn on the vertex of the scalp, multiple erythematous scaly plaques on the glabella and left infraorbital cheek, and cutaneous horns on the midline chest and left forearm. Wheezes were auscultated in the right lower lung field. The abdomen was soft and nontender.

Both the CBC and BMP were unremarkable. Calcium was 9.4 mg/dL. Alkaline phosphatase was 180 IU/L. Chest x-ray revealed a 3.6-cm left perihilar nodular opacity and right lower lung patchy opacities. Subsequent CT of the chest revealed multiple mass-like airspace opacities, including a cavitary mass in the posterior right lower lobe that was 5 cm in diameter. Biopsies revealed the vertex scalp lesion as squamous cell carcinoma and the glabella lesion as folliculotropic mycosis fungoides. The right lower lung lesion was biopsied and revealed poorly-differentiated squamous cell carcinoma. A MRI of the brain also revealed two metastatic lesions. The presumptive diagnosis was metastatic cutaneous squamous cell carcinoma.

Discussion:

Cutaneous SCC lesions greater than 2 cm in diameter have a recurrence rate of 15% and a metastasis rate of 30%, three times higher than smaller lesions. Lesions located on the lip, ear, scalp, forehead, eyelid and nose have a rate of recurrence and metastasis that ranges from 10 to 25%, two to three times higher than lower-risk lesions. The presence of firm papules or plaques with ulceration, hyperkeratosis, or cutaneous horns, all of which were present in this patient, should alert clinicians to the possibility of recurrent disease in patients with a known history of cSCC. When these lesions are large and present in high-risk locations, as was the case in this patient, there should be a high suspicion for distant metastatic disease.

**Name:** Paola Roland, MD **Presentation Type:** Oral Presentation

**Residency Program:** University of Colorado School of Medicine

**Additional Authors:** Mark Foster, MD, Carlos Roldan, MD, Eric Young, MD

## Abstract Title: Shocking Amiodipine Toxicity

### Abstract Information:

Background: Calcium channel blocker (CCB) toxicity in a patient with cirrhosis and acute renal failure may present with shock due to peripheral vasodilation and depression of the myocardium and conduction system, which poses major diagnostic and therapeutic challenges.

Case: A 68-year-old male presented with hypotension, dyspnea and oliguria and admitted for undifferentiated shock and acute renal failure. His past medical history included chronic kidney disease stage III, type II diabetes mellitus, heart failure with preserved ejection fraction and paroxysmal atrial fibrillation on chronic anticoagulation. For a week prior to admission, despite feeling ill, he continued taking warfarin, amlodipine, lisinopril and insulin.

Decision-making: The patient was initially treated as septic shock with intravenous fluids, broad spectrum antibiotics and norepinephrine (NE) with minimal improvement and negative infectious workup. An abdominal ultrasound revealed cirrhosis explaining his supratherapeutic INR of 7.5. A transthoracic echocardiogram (TTE) showed a large pericardial effusion with right ventricular diastolic collapse and ventricular interdependence suggestive of cardiac tamponade. A pericardiocentesis removed 700cc of hemorrhagic fluid. However, the patient remained hypotensive, severely hyperglycemic, lactic-acidotic and oliguric. With pulmonary artery catheterization data suggestive of cardiogenic shock, dobutamine was started but ineffective. A repeat TTE showed preserved left ventricular function and no pericardial effusion, so he was placed on NE, an insulin drip, and calcium infusions, hemodialysis for volume overload, metabolic acidosis and hyperkalemia and mechanical ventilation for impending respiratory failure. The patient's hemodynamics improved and he was slowly weaned off hemodialysis, ventilator and infusions. Given the transient obstructive and mixed cardiogenic and distributive shock, severe acidosis, and hyperglycemia, we suspect the patient's hemodynamic collapse was due to amlodipine toxicity in the setting of cirrhosis.

Conclusion: This case illustrates that CCB toxicity can cause mixed cardiogenic and distributive shock.

**Name:** Christina Sass, DO **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: A Dizzying Diagnosis

### Abstract Information:

**Introduction**: Dizziness is a common chief complaint that is sometimes difficult to assess clinically. Prolonged dizziness with ocular finding should be suspicious for a paraneoplastic syndrome called obsoclonus/myclonus aka "dancing eye syndrome".

**HPI:**

Patient is a 76 y/o former smoker with multiple hospitalizations for chief complaint of dizziness. She initially presented to her PCP in September with sudden onset dizziness reported as "room spinning" that had gradually worsened. She was trialed on meclizine and valium with no improvement. She was admitted to the hospital when her symptoms persisted causing nausea and vomiting preventing any oral intake. The dizziness worsened with her eyes open. Patient noted having difficulty with eye movement on command. On physical exam she was noted to have horizontal nystagmus. CT and MRI of brain were negative for acute findings. Epley maneuver was performed and did not bring relief. Patient continued to have nausea, vomiting, and dizziness. LP was showed normal cell count and differential, cultures were negative and paraneoplastic panel sent which also returned negative. She was diagnosed with vestibular neuritis and discharged to rehab. She re-presented to the ED a month later with the same symptoms. Second workup including CXR, MRI brain and neuro consultation was again unrevealing. At different times during these 2 hospitalizations horizontal and vertical nystagmus were seen on exam. She also had episodes of uncontrolled shaking not consistent with seizures. The patient presented a third time in January for AMS. On CXR a new hilar mass was seen. CT chest revealed a large soft tissue mass in the superior mediastinum that extended into the right heart boarder with a small RUL nodule. Biopsy was performed and revealed small cell lung cancer.

**Discussion:**

Opsoclonus Myoclonus (OM) is characterized by conjugate, random directional nystagmus (opsoclonus) coupled with myoclonus +/- dysarthria, truncal ataxia, and confusion. The most common cause of OM in adults is a paraneoplastic syndrome secondary to small cell lung cancer. Symptoms usually precede the cancer diagnosis. Diagnosis is made clinically. There are no well characterized antibodies for OM, so often paraneoplastic testing is negative. With treatment of the malignancy there can be complete or partial recovery of symptoms. The earlier the treatment after symptom onset the better the symptom improvement which is unfortunate for our patient as she originally presented 4 months prior to her diagnosis.

**Conclusion:**

When a patient with a significant smoking history presents with dizziness and nystagmus with a negative brain MRI one should think of opsoclonus myoclonus. A chest CT should be obtained to rule out lung cancer as small lung nodules may be missed on a CXR.

**Name:** Eric Schultz, DO **Presentation Type:** Oral Presentation

**Residency Program:** Parkview Medical Center

**Additional Authors:** R. Argyle, DO, K. Coffey, DO, L. Longfellow, DO, S. Murphy, DO

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## Abstract Title: The Unrelenting Complications of Chronic Query Fever

### Abstract Information:

**Introduction:**

**Introduction:**
Among zoonotic diseases, Q fever is making its presence increasingly known. First recognized in 1935 in Australia, it was referred to as Q (for query) fever, as the etiology of the disease was unknown. Eventually the cause of Q fever was identified as an infection of the bacteria Coxiella burnetii, an organism found worldwide in farm animal populations. Q fever was recognized in the United States by the early 1940s and its incidence has increased steadily since. Despite the increasing number of cases, Q fever remains a challenging disease to diagnose due to nonspecific and often times benign constitutional presenting symptoms.

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**Case Description:**
Our case discusses the presentation of a forty-four year old male on Etanercept whose occupation of several years was in the removal of dead animals from farmlands. Presenting originally with a cough, fevers and shortness of breath, he was found to have C. burnetti antibodies on serologic workup. Tricuspid valve vegetations were noted on a transesophageal echocardiogram. Aggressive antibiotic administration led to eventual discharge from the hospital on intravenous ceftriaxone. Two months later he returned with similar complaints and persistent vegetations were visible on transthoracic echocardiogram. He was referred to a tertiary center for tricuspid valve replacement. Nearly a year post valve replacement he returned with complaints of chest pain. Serology revealed persistent C. burnetii infection, with IgM and IgG antibodies, and he was referred to an outpatient infectious disease physician for prolonged monitoring on antibiotics. Six months later, presenting for his fifth admissio n since originally diagnosed a year and a half earlier, he presented with a new onset intense headache. A head computerized tomography scan revealed parenchymal hemorrhage with intraventricular hemorrhage. Patient had a slow recovery and was discharged with close outpatient follow-up.

**Discussion:**
Q fever is an uncommon cause of endocarditis accounting for only 3 to 5% of cases and there is often a delay in diagnosis. C. burnetii is a difficult pathogen to treat given it multiplies in macrophages which results in chronic infection, a process which takes approximately 6 months. Both immunosuppression and prior valvular disease have been noted to be associated with increased risk of complications from Q fever.
A well-established sequela of infective endocarditis includes neurologic complications which occur in approximately 35% of endocarditis cases. Intracranial hemorrhage is a serious

neurologic complication that occurs as a transformation after a cerebral infarct or secondary to ruptured mycotic aneurysms. It has been reported that only 0.7% to 5.4% of all cerebral aneurysms are caused by infective emboli. Thus, a mycotic aneurysm believed to be secondary to Q fever infective endocarditis is a rare but potentially fatal outcome.

**Name:** Chelsea Springer, MD **Presentation Type:** Oral Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: A Common Infection with a Rare Fatal Complication

### Abstract Information:

Case Description

A 34 year old female with recurrent small bowel obstructions, pancreatitis, recent *Clostridium difficile* colitis, and extensive abdominal surgical history beginning in childhood presented with abdominal pain, distension, anorexia and altered mental status. On a recent admission she was treated conservatively for small bowel obstruction. Since that time, she had slowly declined. CT scan on presentation demonstrated fluid-filled small bowel, stool-filled colon, and hyperenhancing mucosa. She was taken by surgery for exploratory laparotomy given continued elevated lactate and concerning abdominal exam. Intra-operatively, the bowel appeared healthy. After surgery, she was admitted to the ICU where she developed signs of septic shock and was started on broad-spectrum antibiotics. Three days after admission, blood cultures grew *Clostridium difficile* and the patient was switched to oral and rectal vancomycin with intravenous metronidazole. In the next two days, she developed recurrent hypotension requiring vasopressor therapy. She was taken back to surgery due to concern for bowel ischemia where they found a small perforation, which was repaired. Given continued fevers, Tigecycline and fungal coverage were added to her antibiotic regimen. The patient was taken again back to the operating room two days later due to concerning exam. More areas of the colon were perforated, but these were unable to be repaired due to friability of the tissue. The following day, given the patient's poor prognosis and continued decline, the family decided to withdrawal care and the patient passed.

Discussion

*Clostridium difficile* colitis is a commonly encountered infection in the hospital. Extraintestinal manifestations of *C. difficile* colitis are rare and few cases of *C. difficile* bacteremia have been reported in the literature. Most prior cases describe *C. difficile* bacteremia in the setting of a polymycrobial bacteremia, unlike this case. Risk factors based on previous reported cases include malignancy, recent antibiotic use, alcohol abuse and abdominal surgery. Several mechanisms to understand the pathogenesis of *C. difficile* bacteremia have been postulated. One hypothesizes bacterial transfer through injured intestinal mucosa. Bacterial translocation to blood, lymph nodes, and peritoneum through an inflamed or immunosuppressed gut has also been suggested. In our patient's case both mechanisms could have been possible given her small bowel obstruction and ileus after recent *C. difficile* colitis in the setting of chronic illness.

In patients with history of *C. difficile* colitis, abdominal surgery and recent antibiotic use, it is important to keep *C. difficile* bacteremia on the differential. From review of other cases, presenting symptoms including fever and abdominal pain are nonspecific creating further diagnostic dilemma. Given the high degree of fatality, early recognition of *C. difficile* bacteremia is imperative. Additionally, further research is needed to determine appropriate antibiotic therapy in cases where traditional treatment is not adequate to prevent mortality.

**Name:** Jacob Ludwig, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: The Head Cheese Stands Alone

### Abstract Information:

#### Introduction

Amiodarone is an antiarrhythmic drug often used to treat tachyarrhythmias. However, in modern times it is less commonly used to treat atrial fibrillation with rapid ventricular rates (RVR) due to alternative therapy with AV nodal blockers being widely available, safer and better tolerated. One of the most feared complications of amiodarone is pulmonary toxicity.

#### Case Description

The patient is an 89-year-old female with T1DM who was recently discharged from an outside hospital with a diagnosis of diastolic heart failure after an episode of DKA and atrial fibrillation with RVR. She was discharged on new medications that included amiodarone 400 mg bid and bumetanide 1 mg daily.

Two weeks later she presented to our hospital complaining of shortness of breath. She was clinically dry on exam and had hypoxemia. Chest X-ray was concerning for bilateral pneumonia, however, she had no other infectious symptoms except an unexplained leukocytosis. Procalcitonin was negative. During hospitalization, her oxygen requirements continued to worsen despite broad-spectrum antibiotics covering empirically for HCAP. A chest CT revealed a mosaic-like pattern of significant bilateral infiltrates, air bronchograms and interstitial ground glass opacities. This pattern was interpreted as a "head cheese sign," which is pathognomonic for hypersensitivity pneumonitis.

She was transferred to the ICU, intubated and mechanically ventilated. Despite aggressive corticosteroid therapy and discontinuation of her amiodarone, the patient's condition continued to deteriorate. After discussion with family members, the patient was terminally extubated with death attributed to respiratory failure from acute amiodarone pulmonary toxicity.

#### Discussion

Amiodarone is the most commonly prescribed antiarrhythmic drug in North America. However, it is also associated with several adverse drug reactions, including amiodarone pulmonary toxicity (APT). Amiodarone is lipid soluble and tends to accumulate in highly perfused organs. Lung damage is induced by a direct cytotoxic effect and by an indirect immunological reaction. The lungs of a patient with ATP will show a mosaic pattern of diffuse interstitial pneumonitis, hyperplasia of type II pneumocytes, widening of alveolar septae with an inflammatory infiltrate, varying degrees of interstitial fibrosis, and an accumulation of lipid-laden foamy macrophages in alveolar spaces leading to the pathognomonic "head cheese sign" seen on chest CT. Labs may be remarkable for an unexplained leukocytosis. The most severe manifestation of APT is a rapidly progressing diffuse pneumonitis with an ARDS picture[1].

Although toxicity can occur at any time, the greatest risk is in patients with high total cumulative doses, i.e. >400 mg/d for >2 months. Historically, when patients were put on high

doses, the incidence of APT was 5-15%. However, even at lower doses, the incidence is still reported at around 2%. APT is more frequent in men, and increases with age and co-morbidities, such as pre-existing lung disease and supplemental O2 exposure. The drug of choice for treatment is high dose corticosteroids. Prognosis is favorable unless ARDS develops[1].

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**Name:** Jen Denkmann, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Kirsten Hesterberg, DO

## Abstract Title: A Funny Looking EKG

### Abstract Information:

Introduction:

Arrhythmogenic right ventricular dysplasia (ARVD) is characterized by adipose and fibrous replacement of myocytes, classically in the right ventricle. Fibrofatty tissue in ARVD acts as re-entrant circuits, with adrenergic stimulation provoking arrhythmias. The infiltrated tissue may also result in delayed ventricular activation, manifested as epsilon waves. It is an important cause of sudden death in those less than 30, with up to 20% occurrence rates. The condition was first described in 1977; prevalence is thought to be approximately 1 in 5000, but it may be under recognized. Definitive diagnosis may be obtained with biopsy, but since infiltrates are frequently segmental, tissue may not show changes. Diagnostic criteria include family history, electrocardiogram (ECG), imaging, biopsy and genetic testing.

Case Description:

A 64 year old male with known history of ARVD, with history of paroxysmal ventricular tachycardia, presented for elective ventricular tachycardia ablation. Baseline ECG showed t-wave inversions in V1-4, without epsilon waves. On morning after procedure, patient became acutely hypoxic and hypotensive with standing. CT angiography showed bilateral pulmonary embolisms. Bedside echocardiogram showed acute RV dysfunction. ECG showed development of right heart strain, incomplete right bundle branch block and epsilon waves. Given his recent procedure, and relative hemodynamic stability, tissue plasminogen activator (tPA) was not administered. He was treated with low molecular weight heparin and warfarin. ECG prior to discharge showed regression of incomplete right bundle and epsilon waves.

Discussion:

ECG findings can be instrumental in the diagnosis of ARVD. In the prior case, the patient did have ECG changes consistent with ARVD (T wave inversions in V1-3), but not epsilon waves at baseline. He developed epsilon waves after acute pulmonary embolism with right heart strain by bedside echocardiography. Epsilon waves represent delayed RV activation, likely due to infiltration of myocytes. In this case, the combination of right ventricular stretch, due to the pulmonary embolism, as well as fibrofatty tissue likely resulted in delayed activation, as manifested by epsilon waves. The epsilon waves appeared only in the setting of acute right heart strain in this patient and resolved with treatment. This is unusual in that epsilon waves are normally persistent, if present, in patients with ARVD.

**Name:** Aaron Strobel, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** University of Colorado School of Medicine

**Additional Authors:** Brianna Dix, MS, Lela Mansoori, MD

## Abstract Title: Snowboarding: Shredding More Than Just Snow

### Abstract Information:

Introduction: Acute, atraumatic, exercise-induced bilateral compartment syndrome of the legs from low-endurance activities such as snowboarding or skiing are uncommonly reported in the literature. Acute compartment syndrome (ACS) has potential lifestyle-changing consequences with functional loss that requires high clinical suspicion and emergent treatment.

Case Description: A previously healthy 42-year-old man with prior snowboarding experience developed bilateral lower leg cramping and pain after only 3 hours of snowboarding. He presented to an outside facility after the pain worsened and noticed his urine to be red in color. He was treated for rhabdomyolysis and evaluated by orthopedic specialists who ruled out compartment syndrome based on physical exam at that time. The patient had persistent creatinine kinase elevation and developed elevated liver enzymes so he was transferred to another hospital for further management. On arrival, the patient had extreme pain in his bilateral lower legs with decreased sensation and 3/5 dorsiflexion on physical exam. His compartment pressures were significantly elevated and he required a four-compartment fasciotomy with complete left anterolateral compartment debridement of dead muscle tissue and hyperbaric oxygen treatment. At discharge, the patient had a persistent footdrop that is expected to b e permanent.

Discussion: Although an uncommon cause of leg pain, ACS should be considered in any patient presenting with severe lower leg pain after engaging in physical activity for any duration of time to prevent potential devastating consequences. Nearly 75% of ACS cases are associated with trauma or long bone fractures, but there have been few low-endurance cases reported in the literature. During atraumatic ACS cases, such as those caused by extreme physical exertion, muscles can experience partial ischemia related to the relative oxygen deprivation. This can lead to cell necrosis, release of cell contents, and fluid accumulation in addition to the muscle hypertrophy whereby the muscle can increase by up to 20% in size. Due to the confined compartments created by the fascial planes, this is a surgical emergency for which decompression is sometimes needed. Diagnosis requires directly measuring the pressures within these compartments with a handheld manometer with a suggested threshold of 20mm Hg to consider fasciotomy. Although fasciotomy should only be done in those with compartment pressures meeting surgical criteria and under the clinical judgment of surgical specialists, timely diagnosis and treatment is important to prevent patients from developing long-term

consequences. Mortality in patients requiring fasciotomy for ACS has been estimated to approach 15% in some studies, but more common are permanent sensory loss, footdrop, and other functional deficits.

**Name:** Keleigh McLaughlin, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: Itching for A Reason

### Abstract Information:

**INTRODUCTION:**

Early signs of relapse lymphoma may include 'B' symptoms (weight loss >10% of body weight over the last six months, fevers >38°c, and drenching night sweats), as well as biochemical parameter changes, such as elevated LDH. These symptoms may precede actual nodal enlargement by several months (1). We report on a 37-year-old male with a three-month history of nightly urticaria, presumed to be the first indication of relapsed Grey Zone Lymphoma (GZL). After a thorough literature review, we believe this is the first case of relapsing and remitting lower extremity urticaria as an initial indicator of relapsed lymphoma.

**CASE PRESENTATION:**

A 37-year-old Caucasian male presented to clinic complaining of a recurrent pruritic rash that would appear on his lower extremities every evening for the past three months. Over this period, he used oral antihistamines, topical steroids and dietary modification, with no evident benefit. Night sweats began during the third month, and two weeks prior to review he developed a tender 4cm mass in his left groin.

His past medical history included Stage III Grey Zone Lymphoma (treated in 2009 with R-CHOP achieving a Complete Response), and mild hypertension.

On clinical review the only abnormality evident was inguinal adenopathy. The urticaria was never evident during clinical review, however the patient's wife documented the nightly presentation of these skin changes (Images 1, 2). The lesions never involved the hands, feet, or mucus membranes.

Laboratory data revealed an LDH two times normal. Imaging revealed mediastinal, hilar, inguinal, and retroperitoneal adenopathy. Excisional biopsy of the enlarged lymph node confirmed recurrent lymphoma with similar characteristics to initial disease from 2009. He was diagnosed with recurrent Stage III GZL.

He received three cycles of R-ICE chemotherapy in preparation for an autologous transplant. By day two of his initial R-ICE chemotherapy, the urticaria had resolved and has not returned.

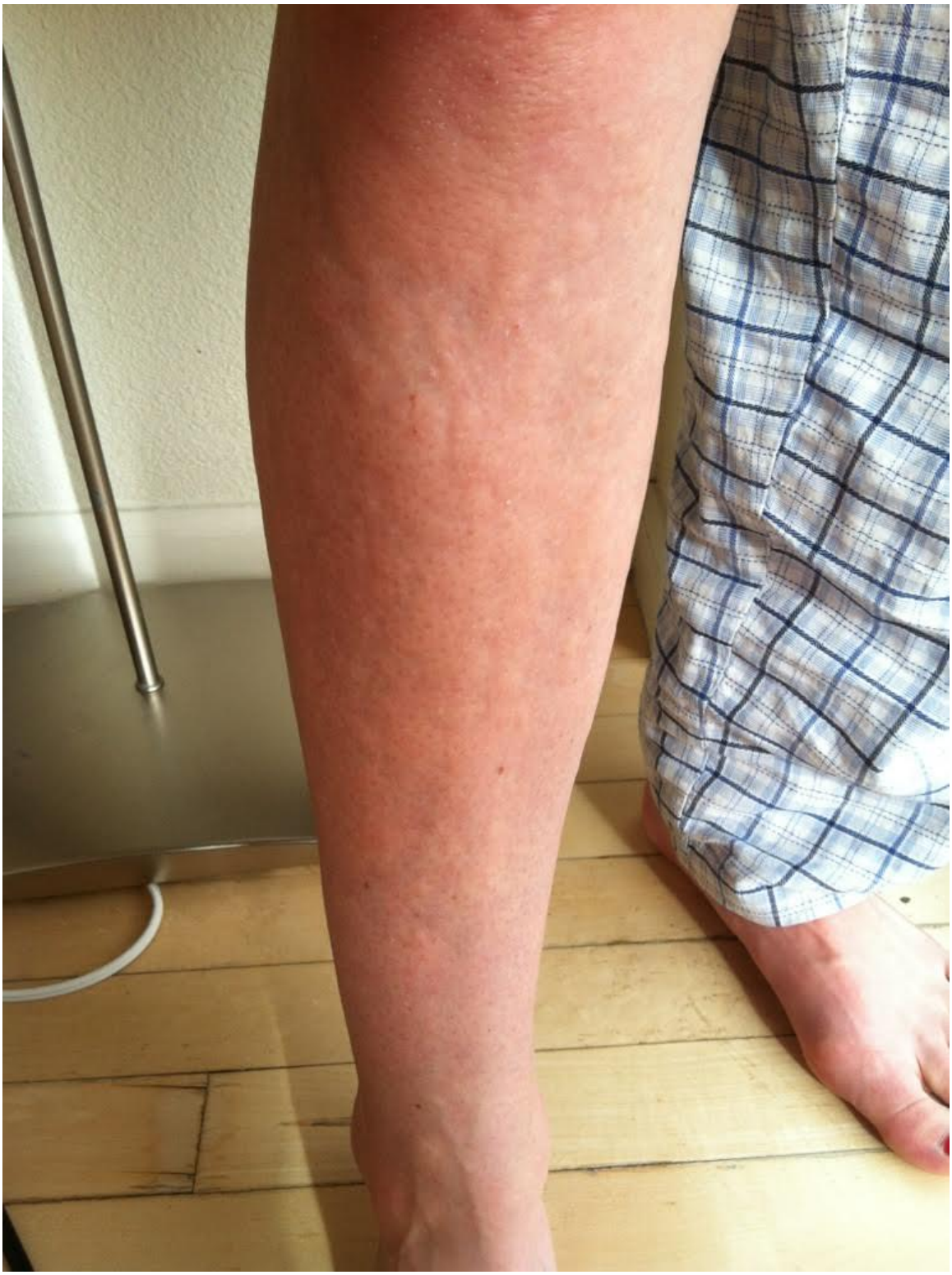
**DISCUSSION:**

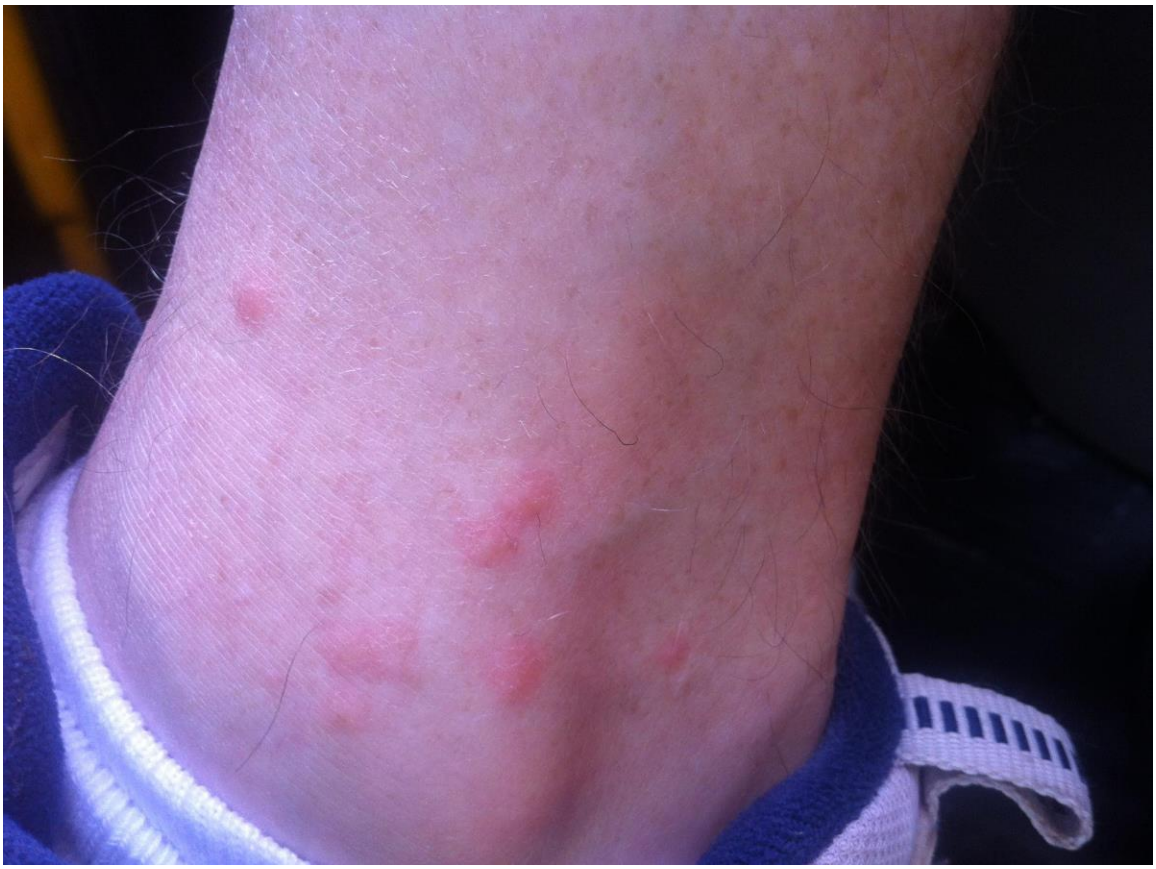
Although the connection between the urticarial lesions and return of the lymphoma was suspected, imaging had not confirmed the disease relapse until almost 3 months had passed from their first presentation.

In two studies following patients with chronic urticaria, there had been no increase in detected malignancies (2,3). Castelli et al reported angioedema occurring as a first presentation in lymphoproliferative disorders. These cases presented with involvement of the face, hands, feet, and mucus membranes, with biochemical abnormalities associated with C-1 esterase inhibitor deficiency (4). We had no laboratory data suggesting this possible etiology.

The escalation and rapid disappearance of the urticaria as the lymphoma worsened and was successfully treated suggest these symptoms represented a preclinical immunological expression of the lymphoma. The nature of this abnormality is unknown and merits further exploration.

Images:

1) 

2) 

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**Name:** Melissa O'Meara, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: Oh Crap, Now What Do We Do?

## Abstract Information:

**Introduction:**

Fecal microbiota transplant (FMT) is a highly effective treatment for *Clostridium difficile* colitis, with cure rates around 94%. With rates of recalcitrance as low as 6%, many providers may not have first hand experience providing care for patients with FMT resistant *C diff.* While consensus opinion favors FMT after failure of first line therapies, there is not yet consensus on what to do when FMT fails.

**Case Description**:

A 66 year old woman with multiple medical problems presented to her doctor for nausea, vomiting, and frequent watery diarrhea—greater than 12 bouts per day. She had recently completed courses of clindamycin and amoxicillin. She had received the clindamycin for a dental abscess. On the heels of this infection, she was treated for pneumonia with amoxicillin. Her diarrhea began midway through the course of amoxicillin. She was found to be *C diff* positive and showed signs of volume depletion and was hospitalized for further care. Her original hospitalization was a 36 day stay. She was initially treated with PO vancomycin 125mg four times a day. However, as her condition worsened, treatments were escalated to include IV metronidazole, vancomycin enemas, and high dose PO vancomycin, without clinical improvement. Antibiotics were discontinued and FMT was performed. The patient had initial improvement, but 6 days later, she had recurrent diarrhea. Antibiotics were resumed, and alternative causes of pseudomembranous colitis were ruled out. The patient was discharged to a skilled nursing facility on IV metronidazole, PO fidaxomicin, and IV tigecycline. She returned to the hospital four weeks later having completed antibiotics, again with copious diarrhea. She underwent a second FMT, but again has had recurrence of diarrhea. Current treatment options include considering bezlotoxumab, a novel human monoclonal antibody against *C diff* toxin A & B, for prevention of recurrence.

**Discussion**

This case provides an illustration of the potential avenues for escalation of treatment for *C. diff* colitis, including escalation of antibiotic therapy, evaluation for other causes, and novel agents. Bezlotoxumab is one such novel agent. Recent results from global phase 3 trials show that bezlotoxumab infusion along with standard antibiotic treatments can reduce rates of reinfection by up to 51%. The number needed to treat to prevent reinfection at 12 weeks for patients over 65 is 6 (NEJM Jan 2017). An estimated 15-30,000 people die of *C diff* infection annually. Given

this sobering statistic and the fact that *C diff* incidence is rising, it is important to understand treatment options for infections that do not response to first and second line therapies.

**Name:** Avi Salamon, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

## Abstract Title: Acute Coronary Syndrome in a Young Woman with SLE

### Abstract Information:

Introduction
Patients with systemic lupus erythematosus (SLE) are known to be at increased risk of coronary artery disease (CAD), even in young individuals. Often, these patients are young women presenting with symptoms of atypical chest pain. Acute coronary syndrome (ACS) can be initially missed due to their perceived low risk.

Case Presentation
A 28 year-old woman with history of SLE diagnosed at age 15 presented to the ED with chest pain. Her SLE has been quiescent for 10 years after an episode of pericarditis and lupus nephritis treated with steroids and Cyclophosphamide. She has had regular laboratory evaluations which have not demonstrated ongoing disease.

She had had three episodes of chest pain over the previous weeks, all occurring during food or beverage intake. On initial evaluation in the ED, she had a negative troponin and her EKG was not suspicious for ischemia. While her symptoms resolved, her troponin increased to 14.8 and her EKG developed biphasic T-waves in the lateral leads. An echocardiogram did not demonstrate effusions or wall motion abnormalities, so she underwent coronary CT, which was equivocal. This prompted cardiac angiogram evaluation, which demonstrated diffuse plaque in the proximal-mid LAD and 80% stenosis of the ostium of the D2 branch. She received a cardiac stent and was discharged with dual anti-platelet therapy.

Interestingly, during her stay, she was evaluated for occult lupus activity. She had low levels of complement (C3) and elevated dsDNA at a titer of 1:160. She was also found to have mild proteinuria. Thus, it was suspected that her non-ST elevation MI might be attributed to subclinical lupus activity.

Discussion
SLE is known to increase a patient's risk of coronary artery disease. This association has been well studied, and the greatest relative risk is associated with patients who have concomitant hypertension, dyslipidemia, longer duration of SLE, long durations of steroid use, and high lupus activity. It has been reported that patients can have cardiovascular involvement without conventional atherosclerosis risk factors, even in the absence of clinical lupus activity. Although our patient had no cardiovascular risk factors, it is thought that her SLE may have had underlying subclinical activity, thereby putting her at increased risk for CAD and ultimately leading to a myocardial infarction.

It is unclear if patients with subclinical SLE should be screened for atherosclerotic disease in an effort to minimize CAD risks. Prospective trials have shown cardiac abnormalities in asymptomatic SLE patients. Though chest pain in young women with SLE is more often due to pericarditis or pleuritis, it is imperative to consider myocardial infarction in this population.

**Name:** Kiely Schultz, DO **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Parkview Medical Center

**Additional Authors:** J. Buchholz, DO, E. Elliott, DO

## Abstract Title: Tularemia: An Almost Missed Diagnosis

### Abstract Information:

**Introduction:**
Tularemia can present with a number of vague symptoms and non-diagnostic laboratory findings necessitating a high level of suspicion in order to not miss this rare, but important, diagnosis.

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**Case Description:**
A 32 year old male with past medical history significant for cirrhosis, alcoholic hepatitis and hepatic encephalopathy had initially presented with a chief complaint of productive cough. The patient, who was recently hospitalized for hepatic encephalopathy, was found to have a chest X-ray concerning for pneumonia versus ARDS, leukocytosis and elevated procalcitonin. He was admitted to the ICU and started on treatment for HCAP. Initially, he responded well to therapy and was transferred out of the ICU. Shortly after, however, he suffered respiratory distress and his leukocytosis worsened and, on hospital day six, he was transferred back to the ICU due to concerns for his respiratory status. Infectious disease was consulted and, at that time, a concern for zoonotic infection was raised as it was discovered he was frequently around goats, chickens, sheep and rodents. He underwent bronchoscopy and, with a concern for zoonosis, his antibiotics were changed to empiric doxycycline. He continued on doxycycline with a slow but steady improvement and was ultimately discharged thirteen days later. On the morning of discharge, his *Francisella tularensis* IgG antibody level, which had been drawn twelve days earlier, resulted positive at a level of 1:128. After having already had thirteen days of treatment, it was recommended he take one more day of doxycycline and have a repeat titer done in one month as an outpatient. The patient did not follow up with this but has made ER visits since then for unrelated complaints and appears to have had no further issues in regards to this case of pneumonic Tularemia.

**Discussion:**
This case demonstrates two important points. First, while an initial diagnosis of HCAP was reasonable and he seemed to initially respond to treatment, he slowly worsened and it took several more days of "staying the course" as well as a being transferred to the ICU for the management to change. Most importantly, it was six days into his admission that important risk factors (animal exposure) came to light, highlighting the importance of both taking a good history and maintaining a broad differential. While this patient had a number of lab abnormalities (thrombocytopenia, hyponatremia, transaminitis) that can be associated with tularemia, they could also be explained by his history of liver disease. When his leukocytosis persisted and then worsened, a return to HPI questioning could have led to changes in management much sooner.

**Name:** Jacob Ludwig, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:**

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**Abstract Title:** Pharming: A Case of Refractory Seizures Due to Chronic Diphenhydramine Overdose

## Abstract Information:

**Introduction:**

Diphenhydramine abuse is common, especially as treatment for insomnia and for intoxicant effect, and symptoms associated with anticholinergic overdose are dose-dependent. With ingestion of greater than 300-1000 mg daily, symptoms can include visual hallucinations, seizures, coma and life threatening QRS or QT prolongation.

**Case Description:**

PT is a 19 year old female with intractable seizures who presented to the epilepsy-monitoring unit (EMU) for further evaluation after failing to respond to antiepileptic drug (AED) therapy. PT's neurological issues began at 15 years old, which included tremor, difficulty concentrating and episodic involuntary limb/torso movements often triggered by studying, focusing, reading or playing piano. At age 18, PT experienced worsening insomnia and depression, and her father began noticing focal seizures that would progress into generalized bilateral convulsions with loss of consciousness. PT's father noted that the seizures often occurred around the 20th of every month despite increasing doses of Levetiracetam. At age 19, PT's seizures became more frequent and she was experiencing colorful visual hallucinations. Her medications were changed to Lamotrigine and Topiramate, but her seizures continued to be refractory despite increased AED doses.

On the first day in the EMU PT suffered numerous brief seizures and periods of hemodynamic instability. On hospital day 2 she had a prolonged seizure aborted with ativan. A CNA then discovered numerous diphenhydramine pills underneath the recliner chair. The neurologist reviewed the recorded video, which revealed PT taking several handfuls, 300-500 mg at one time, of diphenhydramine pills overnight and, notably, one hour before her prolonged seizure. She was intubated and sedated with propofol for seizure control. Her QRS remained stable, her symptoms eventually subsided and she was successfully extubated.

**Discussion:**

With advancements in biotechnology and imaging the ability to determine a patient's specific epilepsy etiology has improved, often identifying genetic, structural or metabolic causes. However, the etiology of up to one third of all cases remains unknown. PT's epilepsy was associated with strange features that included visual hallucinations, altered mental status, urinary retention, hyperkinetic movements and hemodynamic instability, and was refractory to several antiepileptic drugs. When it was discovered that her seizures were from chronic daily diphenhydramine overdose the picture came together and she eventually endorsed motivations for use that included insomnia, depression, intoxication and suicidal ideations.

“Pharming,” or the use of prescription or OTC drugs for intoxication purposes, among teens has been trending up in recent years. Recreational use of diphenhydramine for hallucinogenic effect is reported in the literature at doses of 300-700 mg. Furthermore, with chronic use, addiction ensues and withdrawal symptoms can be very distressing. Through the efforts of diligent physicians, assisted by advances in technology, the cause of PT’s seizures was finally determined and she was ultimately treated for her diphenhydramine addiction.

**Name:** Kiely Schultz, DO **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Parkview Medical Center

**Additional Authors:** B. Pottebaum, DO, S. Baghi, MD

## Abstract Title: Transaminitis and Pruritic Rash in an 81-Year-Old Female: Primary Billiary Cholangitis With Possible Drug-Induced Liver Injury

**Abstract Information:**

**Introduction:**
This is a case of an 81 year old female who presented with a pruritic, lower extremity rash of several months duration for which she received multiple antibiotics. Incidentally, she was found to have transaminitis and extensive work-up including liver biopsy likely lead to a diagnosis of primary biliary cholangitis. This case highlights both the importance of maintaining a broad differential and judicious medication use.

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**Case Description:**
An 81 year old female with medical history of hyperlipidemia and depression presented to the ER with a complaint of several months of pruritic rash in varying locations as well as weakness with reported fifty pound weight loss over the past year. Per the patient's report, she had been on multiple antibiotics for these varying rashes but with little overall improvement and her main complaint of pruritus persisted. On admission, she was noted to have transaminitis with her AST 78, ALT 66, and AP 273. Infectious disease was consulted for the rash, whom also felt it was not cellulitic in nature and agreed with monitoring off antibiotics. An extensive work-up was undertaken for her transaminitis, which trended up for the first several days of her admission before slowly down trending. During her hospitalization her INR and total bilirubin remained normal and she had no signs or symptoms of obstructive disease, though she was noted to have cholelithiasis on right upper quadrant ultrasou nd and a mildly dilated duct at 0.9 cm on MRCP. She had a liver biopsy with non-specific inflammatory findings as well as onionskin fibrosis and multiple labs, including copper, ceruloplasmin, serum protein electrophoresis studies, infectious and auto-immune serologies were all negative, with the exception of an anti-mitochondrial antibody level elevated at 111. Gastroenterology was involved with her case and ultimately felt that, while she likely has primary biliary cholangitis and should be started on Ursodiol, with her multiple recent medications it was difficult to assess with her presentation and non-specific liver biopsy findings if drug-induced liver injury was playing a role. She was recommended to have close outpatient GI follow; two months later she has yet to follow up.

**Discussion:**

Primary biliary cholangitis is a rare diagnosis, which can have an asymptomatic presentation, though the two most common presenting symptoms are fatigue and pruritus. While transaminitis is associated with PBC, a positive anti-mitochondrial antibody is considered a serologic hallmark

for the disease. In this case, the patient did have an alkaline phosphatase greater than 1.5 times the upper limit of normal, an elevated anti-mitochondrial antibody and pruritus, therefore she likely does have PBC. Interestingly, her pruritus and rash could arguably have been secondary to PBC, however with her multiple recent antibiotics, there could have been an additional element of drug-induced liver injury.

**Name:** Atouda Sobhi, MD **Presentation Type:** Clinical Vignette Poster Presentation

**Residency Program:** Saint Joseph Hospital

**Additional Authors:** Chelsea Springer, MD

## Abstract Title: Incessant Thrombosis: Recurrent Venous Thromboembolism in Malignancy Despite Appropriate Therapy

### Abstract Information:

**Introduction**: Malignancy is associated with increased risk of initial and recurrent thromboembolism. The CLOT trial provided evidence for superiority of low molecular weight heparin (LMWH) over vitamin K antagonists (VKA) in reducing risk of malignancy-associated recurrent venous thromboembolism (VTE). However, when recurrence does happen despite appropriate anticoagulant treatment, it poses a management challenge.

**Case Description**: A 55 year old female with history of metastatic cholangiocarcinoma initially presented to the hospital for staging exploratory laparoscopy. During that hospitalization, she was found to be hypoxic. CT-Angiogram revealed bilateral PE's, and enoxaparin at 1.5 mg/kg/day was started. She subsequently presented to the hospital 3 weeks later with left lower extremity edema and right upper extremity pain. These symptoms were not present at the time of her initial PE diagnosis. Ultrasound showed bilateral lower extremity DVTs and right upper extremity basilic vein thrombus. The patient's enoxaparin was then increased to 1 mg/kg BID. The patient again presented to the hospital five days later with worsening shortness of breath. Diagnostic workup revealed new and worsening clot burden both in the pulmonary arteries and lower extremities. The patient reported strict compliance with her anticoagulation. While in the emergency department, an unfractionated heparin drip was started which precluded obtaining an anti-Xa level to assess adequacy of the recently escalated LMWH dosing. Following the National Comprehensive Cancer Network (NCCN) guidelines, the patient was switched from LMWH to fondaparinux. A HIT antibody assay was also obtained and was negative. Patient was not an IVC filter candidate due to extensive IVC clot burden.

Our case demonstrates the management dilemma of recurrent malignancy-associated VTE. Even when treated with LMWH, about 9% of patients with active cancer experience VTE recurrence with subsequent increase in morbidity and mortality. There is very limited data to guide further management of these individuals. There are no head-to-head randomized controlled trials to compare the efficacy of LMWH with non-VKA classes of anticoagulants, such as the novel oral agents or fondaparinux, in active malignancy. There is some data in form of a few small cohort studies that supports effectiveness of the escalation of the initial LMWH dose in reducing rate of a second VTE recurrence. Obtaining factor-Xa-inhibitor levels is also considered helpful in guiding appropriate dosing; however, there is limited clinical validation for interpretation of assay results and their correspondence with patient outcomes. In conclusion, review of the literature indicates that there is very limited data for management of recurrent VTE in malignancy. The current NCCN guidelines are primarily based on expert opinion and recommend obtaining anti-Xa level, pursuing dose-escalation of LMWH or transitioning to fondaparinux, ruling out HIT, and evaluating for IVC filter placement.

**Name:** Brian Schulte, MD **Presentation Type:** Research Poster Presentation

**Residency Program:** University of Colorado School of Medicine

**Additional Authors:** Wei Zhang, Brett Stevens, Elizabeth Budde, Stephen Forman, Craig Jordan, Enkhtsetseg Purev

## Abstract Title: Anit-CD123 CAR T-Cell Therapy for Treatment of Myelodysplastic Syndrome

## Abstract Information:

Myelodysplastic syndrome (MDS) is a group of heterogeneous disorders caused by ineffective hematopoieseis that is characterized by bone marrow dysplasia and results in cytopenia. Currently, treatment options for MDS are limited to supportive care and hypomethylating agents, and most patients eventually succumb to the disease or progress to leukemia. We have demonstrated that CD123 is an important target for the treatment of MDS due to its aberrantly high expression in high-risk MDS stem cells compared to non-stem cells and normal stem cells. We generated a chimeric antigen receptor (CAR) vecotr containing CD123-specific single-chain variable fragment in combination with a CD28 costimulatory domain, CD3 signaling domain and tEGFR. Anti-CD123 CAR was expressed on healthy donor and patient derived T lymphocytes utilizing lentiviral vector delivery to target high-risk MDS stem cells. Healthy donor deriverd anti-CD123 CAR T-cells effectively eliminated MDS cell line (>99% eradication) and primary bone marrow derived MDS stem cells (>70% eradication) in vitro. The killing was associated with increased cytokine release and CD107 degranulation by anti-CD123 CAR T-cells, which did not occur with control anti-CD19 CAR T-cells. Additionally, we successfully transduced T-cells obtained from patients with high risk MDS to express anti-CD123 CAR with 50-70% transduction efficiency. Patient derived anti-CD123 CAR T-cells efficiently eradicated autologous CD123+ MDS stem cells (>99% eradication) in vitro compared to untransduce d control T-cells.
These results suggest that anti-CD123 CAR T-cells exhibit activity against high-risk MDS, and represent an effective therapeutic option for patients with high risk MDS.

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## Abstract Title: Psychosocial Distress Screening: Trends Among Hispanic and Caucasian Women with Newly Diagnosed Breast Cancer

### Abstract Information:

Background: We aim to describe our psychosocial distress screening program with attention to our female breast cancer population and symptoms of anxiety and depression among English-speaking Caucasians and Spanish speaking Hispanics.

Methods: We retrospectively examined the emotional distress scores of 170 women with newly diagnosed breast cancer at the Cancer Centers of Colorado at SCL Heath/Saint Joseph Hospital from January-December 2015. SCL Health IRB approval was obtained. Patients were provided a distress screening questionnaire (English or Spanish) at their initial visit. Data points included: gender, age, ethnicity, primary language, emotional distress (worry/nervousness or anxiety/fears and sadness/depression) and degree of distress ("thermometer" scale 0-10 or not at all, slightly, moderately, seriously or very seriously). The degree of distress measures were revised in August 2015 from a "thermometer" scale to a descriptive scale.

Results: Of 170 women studied, there were 104 Caucasian, 49 Hispanic, and 17 other race/ethnicity (including Black, Asian, Native American, and Pacific Islander). 76 women (40 Caucasian, 31 Hispanic, 5 other) did not participate in filling out the questionnaire. Of the 94 patients responding, 68% were Caucasian (n=64), 19% Hispanic (n=18), and 13% other (n=12). With regard to symptoms, 55% of Caucasians (n=35), 44% of Hispanics (n=8), and 50% of other (n=6) reported anxiety. Moreover, 36% of Caucasians (n=23), 56% of Hispanics (n=10), and 33% of other (n=4) recorded sadness/depression. Among the descriptive scale scores from August-December 2015, Hispanics were the only group to record "seriously" or "very seriously" in regard to anxiety or sadness/depression.

Conclusions: Our retrospective study of emotional distress screening demonstrated lower participation among Hispanics with breast cancer. Concerns of anxiety and depression were common among all groups, with a small number of Hispanics reporting the highest levels. Emotional distress screening is an important component of cancer care for women with breast cancer and barriers for non-English speaking Hispanic patients need to be examined. Further research aims to assess whether, with outreach from mental health and social work, there are also disparities between Hispanic and Caucasian women in acceptance of an intervention, and if differences in a successful response to these interventions exist.