2019 ORAL CLINICAL VIGNETTE PRESENTATIONS

NAME	TITLE	PROGRAM
Dr. Jasmine Manley **ORAL CV 1**	Out of Sight – Out of Mind: An Unexpected Cause of Painful Ophthalmoplegia	**GBMC**
Dr. Janelle Ho **ORAL CV 2**	When a Balanced Diet is No Small Potatoes: Hemolytic Anemia in an 18 Year Old	**Hopkins/Bayview**
Dr. Bernard Landry-Wegener **ORAL CV 3**	Necrotizing Tracheobronchitis as an Extra-Intestinal Manifestation of Ulcerative Colitis	**Hopkins/Broadway**
Dr. Monika Kulasekaran **ORAL CV 4**	The Tip of the Iceberg: Ocular Lambert-Eaton Myasthenia Syndrome from an Uncommon Origin	**MedStar Harbor**
Dr. Cassandra Egwom **ORAL CV 5**	The Winter That Made Us Wonder	**MHIM**
Dr. Asadullah Jat **ORAL CV 6**	Tetanus after a Skin Abrasion	**St. Agnes**
Dr. Gregory Vo **ORAL CV 7**	Hyponatremia and Brain Mass – SIADH or Something Else?	**Sinai Hospital**
Dr. Victor Peng **ORAL CV 8**	Overcoming Bias: Shedding Light on an Unusual Cause of Chronic Abdominal Pain	**UMMC/Midtown**
Dr. Nina Millman **ORAL CV 9**	A Case of Babesia-Associated Hemolytic Anemia in a Patient with Evan's Syndrome	**UMMS/VA**
Dr. George Kontogiannis **ORAL CV 10**	Paced Coronary Vasospasm Occurring during Intravenous Nitroglycerine	**Walter Reed**

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OUT OF SIGHT, OUT OF MIND: AN UNEXPECTED CAUSE OF PAINFUL OPHTHALMOPLEGIA

Jasmine Manley, MD, Bongu Ramachandra, MD, Talha Khan, Pauline Daley, MD, Greater Baltimore Medical Center, Towson, Maryland

There are several menacing differentials for painful ophthalmoplegia, and some are more difficult to diagnose than others. Tolosa-Hunt Syndrome (THS) is a particularly rare etiology that is highly treatable. Since most patients who present with orbital pain and paresis won't have THS, this was an excellent exercise in physical exam and diagnostic exclusion, which uncovered a 1 in 1 million case.

Mrs. N is a 47-year-old woman from Kenya who presented to the hospital with unremitting headache and blurred vision. She described left-sided facial fullness and left orbital pain that had been worsening over the past 3 weeks. The pain was associated with persistent intermittent horizontal diplopia when driving, and general unsteadiness without falls. A physical exam revealed left-sided cranial nerve (CN) 6 palsy and a partial CN 3 palsy with an inability to elevate the left eye (the left eye diving below the midline on adduction). The remainder of the physical including the neurological exam was normal. CTA, Brain MRI and LP were performed to rule out common causes of painful ophthalmoplegia including neoplasm, vascular abnormalities, infectious and inflammatory etiologies. Radiographic findings on Brain MRI showed soft tissue thickening along the medial margin of the left temporal lobe extending into the superior orbital fissure, which was concerning for a meningioma. Orbital MRI revealed characteristic findings of THS: soft tissue thickening abutting the lateral aspect of the cavernous sinus extending into the orbital apex via the superior orbital fissure. Resulting mass effect and compression of the structures within the left orbit was consistent with her pain and palsies. Rapid remission of pain shortly after treatment with glucocorticoids is a defining feature of THS.

Mrs. N received IV glucocorticoids and saw drastic resolution of her pain within 2 days. She is undergoing a prolonged prednisone taper with plans for follow up with neuro-ophthalmology to assess for radiographic improvement, which often lags clinical improvement by several weeks.

The diagnostic challenges in this case inspire physicians to review oculomotor physiology and pathologies that should be ruled out when patients present with painful ophthalmoplegia. For patients previously diagnosed with THS and develop similar symptoms, physicians are encouraged to do a rigorous evaluation in the usual fashion, avoiding early closure or anchoring bias as THS is rare.

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When a Balanced Diet is No Small Potatoes: Hemolytic Anemia in an 18-year-old

An 18-year-old recent high school graduate and lover of video games presented to an urgent care clinic with 3 days of nausea, non-bloody, non-bilious vomiting and generalized weakness. He was found to be profoundly anemic with a hemoglobin of 5.8 and scleral icterus and was transferred to the ED for further evaluation. He denied any recent illnesses, sick contacts or constitutional symptoms. He had no history of bleeding, bruising or new rashes. He also had no personal or family history of sickle cell disease, blood disorder or cancer. He denied illicit drug use or significant alcohol intake. His diet consisted only of starches, specifically fried potatoes and rice. He had not consumed meat, vegetables, dairy or fruit since early childhood because of reported aversion. His family confirmed this fact.

On exam, he was pale-appearing but his vital signs were normal. He had scleral and sublingual icterus and conjunctival and palmar pallor, but no palatal petechiae or bleeding gums, and no thyromegaly. His heart was tachycardic but regular with a 2/6 early SEM heard best at the LUSB. He had hyperactive bowel sounds, but no ascites and his liver was not enlarged. His neurologic exam was normal.

Admission labs were notable for hemoglobin of 5.5, hematocrit of 15.3 with MCV of 96.8 and RDW of 22.6. His WBC and platelet counts were normal. His alkaline phosphatase was 160, AST 176 and ALT 115. His total bilirubin was 4.6 and direct bilirubin 0.5. LDH was >4000, with haptoglobin <6 and low C3. His D-dimer was 16.63 and fibrinogen 145 consistent with a hemolytic process. Serum B12 was mildly low at 150 and MMA was mildly high at 341. Remarkably, his folate was profoundly low at <2 and his homocysteine was extremely high at 187.5 (upper limit 15). Peripheral blood smear revealed anisopoikilocytosis with a few schistocytes and hyper-segmented neutrophils, consistent with hemolysis and folate deficiency. Subsequent autoimmune and GI malabsorption work up was unrevealing.

The final diagnosis was severe folate and mild B12 deficiency, secondary to dietary insufficiency, causing hemolytic anemia. After just three months of folate and B12 supplementation and diet liberalization, his hemoglobin normalized to 12.5, MCV 83, with folate >20 and B12 of 787. He is now focusing on chronic disease prevention and healthy living.

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NECROTIZING TRACHEOBRONCHITIS AS AN EXTRA-INTESTINAL MANIFESTATION OF ULCERATIVE COLITIS

Bernard Landry-Wegener, MD; Chris Haas, MD, PhD; Kelly Arps, MD; Kevan Salimian, MD; David Furfaro, MD

Necrotizing tracheobronchitis is a rare diagnosis that is characterized by mucosal airway sloughing leading to airway obstruction and progressive hypoxemic/ hypercarbic respiratory failure. In adults, the majority of case reports describe a necrotizing/ pseudomembranous tracheobronchitis in the setting of an underlying Aspergillus infection, or lymphoproliferative disorder with associated paraneoplastic pemphigus. There have been rare case reports of necrotizing tracheobronchitis associated with inflammatory bowel disease (IBD). Ulcerative colitis (UC) is an IBD that traditionally manifests with mucosal inflammation in the distal gastrointestinal tract. Additionally, UC has a host of extra-intestinal manifestations. Necrotizing tracheobronchitis is one such rare, albeit critical extra-intestinal manifestation that requires immediate medical management and thus prompt recognition. Here, we describe a unique case of necrotizing tracheobronchitis as a manifestation of UC in a patient who has not undergone colectomy.

The patient was a 55 year old male with recently diagnosed UC, who presented with sore throat, cough, and blood-tinged sputum, in the setting of active UC flare. He rapidly developed hypoxemic respiratory failure necessitating mechanical ventilation. Imaging demonstrated serial opacifications in the right and left upper lobes with subsequent complete left lung opacification consistent with migrating areas of severe atelectasis. Bronchoscopy demonstrated proximal tracheal cartilaginous damage with necrotic, fibroinflammatory secretions. These findings were consistent with necrotizing tracheobronchitis, and caused tracheobronchial obstruction requiring serial bronchoscopy for airway clearance. Infectious, rheumatologic, and vasculitis workup remained unrevealing and he was treated with pulse dose steroids with improvement in his clinical condition. He was ultimately transitioned to Infliximab for maintenance therapy.

While IBD is primarily an intestinal disorder, it remains a systemic disease with multiple extra-intestinal manifestations. This case serves to remind clinicians of one of its rare, but critical complications: necrotizing tracheobronchitis.

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THE TIP OF THE ICEBERG: OCULAR LAMBERT-EATON MYASTHENIA SYNDROME FROM AN UNCOMMON ORIGIN

Monika Kulasekaran, MD; Valentine Baez Sosa, MD; Jason Chien, MD
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Introduction
Lambert-Eaton myasthenic syndrome (LEMS) is an uncommon disorder of neuromuscular junction transmission with muscle weakness as the primary clinical manifestation. Early recognition is particularly important because of its strong association with certain malignancies. It has been commonly associated with small cell lung cancer (SCLC), with only a few reports showing an association with non-small cell lung cancer (NSCLC).

Case Presentation
A 62-year-old incarcerated Egyptian man with a smoking history of 20 pack-years and no other significant past medical history presented from prison with diplopia and ptosis of the left eye. The patient denied any respiratory symptoms. Physical exam was positive for impaired ocular movements in downward gaze in both eyes with diplopia and the weakness improved with repetitive contraction. Computed tomography showed a left lung mass and anterior mediastinal mass with multiple pleural nodular masses. Acetylcholinesterase binding antibody was elevated. A biopsy of the pleural/mediastinal mass revealed poorly differentiated carcinoma with squamous cell differentiation. The patient was treated with pyridostigmine, corticosteroids, intravenous immunoglobulin, plasmapheresis, and pembrolizumab, a PD-L1 checkpoint inhibitor. Subsequently, his neurological symptoms improved.

Discussion
LEMS is a disorder of reduced acetylcholine (Ach) release from the presynaptic nerve terminals, despite usual ACh vesicle number, normal ACh presynaptic concentration, and normal postsynaptic acetylcholine receptors. LEMS may be the first sign of an underlying malignancy.

Conclusion: While SCLC in the primary underlying cause of LEMS, NSCLC is a rare cause.

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The Winter That Made Us Wonder

Cassandra Egwom, MD; Peter Sloane, MD; MedStar Health Internal Medicine, Baltimore, MD

Hypersensitivity pneumonitis (HP) is an alveolitis that is triggered by various extrinsic antigens. The presentation of HP is non-specific and can mimic other respiratory illness, making it diagnostically challenging without a detailed history.

An 82-year-old woman was referred to a pulmonologist in February for dry cough, dyspnea, and fatigue for three weeks. She is a lifetime nonsmoker and has no prior history of respiratory issues. She recently failed a course of antibiotics for presumed pneumonia. Physical exam was notable for hypoxia on room air and clear lungs to auscultation. CT Chest was consistent with cryptogenic organizing pneumonia. She was treated with prednisone taper and 4L home oxygen. Months later, she had remarkable clinical improvement and no longer required oxygen. However, by November, she had recurrence of symptoms. Simultaneously, her 86-year-old husband was referred to a separate pulmonologist for dry cough, worsening fatigue, and exertional dyspnea for 3 months. He is also a lifetime nonsmoker and maintains an active lifestyle. He reported minor relief with inhalers and antihistamines. Lung exam, chest x-ray, and spirometry were normal. He was prescribed a therapeutic trial of prednisone for management of post infectious bronchitis and his symptoms significantly improved. However, the following winter, he had a severe recurrence of dry cough, dyspnea, and fatigue. His symptoms were now refractory to steroids. Further investigation revealed that the couple's symptoms initially presented after they began using a humidifier in their bedroom during the winter a few years ago. The humidifier had never been cleaned. They were diagnosed with "Humidifier Lung"; a HP triggered by breathing humidified air contaminated by microorgamisms. Upon removal of the humidifier, the couple had complete resolution of symptoms as well as normalization of imaging and pulmonary function.

This case illustrates the challenges in diagnosing HP and the invaluable utility of a detailed history when assessing the etiology of relapsing, chronic respiratory issues. The recognition of patterns of illness severity/symptom improvement can lead to avoidance of unnecessary pharmacologic treatment, reduction in healthcare utilization, and improved quality of life.

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Tetanus after a Skin Abrasion

Introduction: Tetanus is a life-threatening, neurotoxin mediated disease that is caused by anaerobic spore forming Clostridium tetanus bacterium.[3] Tetanus is very rare in developing countries and is almost always found in the unimmunized population. We hereby present a case of severe tetanus after minor skin abrasion.

Case: A 40-year-old known asthmatic male presented with generalized muscle cramps, throat tightness and fever. Patient reported an accidental injury from rusted tape gun blade, 15 days before presentation, that resulted in small abrasion on his right hallux for which he did not seek any medical care. Over the next 2 weeks his symptoms progressed from fever, night sweats and fatigue to dysphagia, throat tightness and right leg cramps. He was evaluated by a pulmonologist and was treated for a possible asthma exacerbation. The following 5 days his symptoms evolved into generalized muscle spasms, limited range of motion of the jaw, rigidity, photophobia, phonophobia, palpitations and dizziness. His tetanus immunization status was not up to date. Physical examination was remarkable for trismus, neck rigidity, generalized hypertonicity, brisk deep tendon reflexes at the knees, photophobia and facial muscle twitching when exposed to bright light. A 0.5cm linear abrasion was observed on right hallux. A diagnosis of tetanus was made and treatment with tetanus immunoglobulin, metronidazole and diazepam was started. Hypertonicity and light sensitivity started to improve within 24 hours of treatment, but he was hospitalized for 8 days. At the time of discharge, he still had mild facial muscle twitches, mild muscle spasms of the lower extremities and a spastic gait. At 1 month follow up his gait was still spastic, and he had tetani of the lower extremities with exertion. His symptoms fully resolved in 3 months.
Discussion: Tetanus is a life-threatening disease which can be difficult to identify early on, due to the nonspecific nature of the initial symptoms. Tetanus is commonly associated with puncture wounds and major injuries. This case was different in the sense that the patient suffered a minor injury that did not prompt seeking medical attention and led to a missed opportunity for prophylaxis.[4] Management includes neutralization of unbound toxin, removal of the source of infection, control of muscle spasms and rigidity, monitoring for and treatment of autonomic dysfunction. Recovery is slow process as it requires regeneration of the affected axons.[1,3]
Conclusion: This case emphasizes the importance of a thorough history

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HYPONATREMIA AND BRAIN MASS - SIADH OR SOMETHING ELSE?

Gregory Vo MD, Amteshwar Singh MD, Kellen Mulhern DO, Lauren Berninger DO

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW) are uncommon causes of hypotonic hyponatremia often juxtaposed given similar findings and presentation (encephalopathy from a neurologic insult). Distinguishing them has paramount significance as treatment of each are diametrically opposed. To further convolute this distinction, primary adrenal insufficiency (PAI) and secondary (SAI), can also present with hyponatremia (albeit by different mechanisms), and both respond rapidly to steroids. Hypotonic hyponatremia can often be a leading presentation of PAI due to impaired mineralocorticoid production, but hypotonic hyponatremia secondary to SAI is rare, occurring due to the loss of cortisol's inhibitory effect on ADH secretion. We present a case of hypotonic hyponatremia, where these rare diagnoses were considered causing a diagnostic dilemma.

A 68 year old male with no medical history presented with altered mentation and vision changes while on a Caribbean cruise. On initial exam, he was hypertensive, febrile, and lethargic. Labs revealed a hypotonic hyponatremia and cranial imaging revealed a pituitary macroadenoma with evidence of apoplexy. Serum and urine studies, as well as physical exam were consistent with a presumptive diagnosis of SIADH, and fluid restriction was started. Overnight, the hyponatremia worsened and vaptans were considered but were withheld due to acute polyuria and hypotension. Studies were now suggestive of CSW and hypertonic therapy was initiated. Coincidentally, concurrent endocrine workup revealed impending panhypopituitarism and the patient was started on hormone replacement. The hyponatremia quickly resolved and the patient successfully transsphenoidal pituitary resection.

Although likely multifactorial, the primary etiology of the hyponatremia is theorized to be SAI due to pituitary apoplexy based on clinical response and workup. This case underscores the importance of distinguishing four potentially life threatening conditions that present similarly, but have distinct treatments that can cause considerable morbidity and mortality if not properly employed. The differential for encephalopathy should remain broad and anchoring bias on infectious etiology may lead to premature closure and errors.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!

Overcoming Bias: Shedding Light on An Unusual Cause of Chronic Abdominal Pain

Introduction: Chronic abdominal pain is a common presenting complaint in the inpatient setting. While in many cases, the cause is unknown but benign and self-limiting, identifying the correct etiology can be critical to the patient's longevity and well-being.

Case Presentation: A 27 year old woman with past medical history of Hepatitis C and essential hypertension was admitted with a chief complaint of abdominal pain. She has visited the emergency department on multiple occasions in the past, up to 12 – 15 times a month, with several sonograms and CT scans of her abdomen/pelvis done, all without significant pathology. The patient has been using illicit opiate-containing pain medications since the age of 12, and claims to have had this same abdominal pain as long as she can remember. She notes that heroin provides temporary relief of her pain. She has a history of multiple spontaneous abortions. She was taking no medications at home.

Physical exam was significant for BMI of 19.4 kg/m^2, blood pressure of 155/90 mmHg, diffuse abdominal tenderness to palpation without guarding or rebound. There were no cutaneous findings. Patient was crying and writhing in the bed and preferred staying in the dark. She had flat effect and manic episodes during her stay. Testing for anti-phospholipid syndrome was negative. However, the urinary porphyrin levels were elevated, most notably coproporphyrin I and coproporphyrin III. A repeat urine sample collected during the acute attack of abdominal pain was significant for coproporphyrin elevation III>I.

Conclusions: Her chronic abdominal pain triggered by light exposure could be due to abnormal metabolism of porphyrins related to Hepatitis C, causing an acquired porphyria presenting as abdominal pain. Hepatitis C virus core proteins are known to cause deactivation of uroporphyrinogen decarboxylase and coproporphyrin oxidase, causing an acquired porphyria with symptoms overlapping between classic porphyria cutanea tarda and hereditary coproporphyria.
The patient unfortunately left the hospital before she could be tested for fecal porphyrin levels or for treatment to be initiated. She continues to visit emergency departments to receive narcotics for her abdominal pain.

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A CASE OF BABESIA-ASSOCIATED HEMOLYTIC ANEMIA IN A PATIENT WITH EVAN'S SYNDROME. **Millman N, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.**

It is well documented that Babesia infection can cause hemolytic anemia especially when associated with a high parasite count or in an asplenic patient. The incubation period is between one and eight weeks after initial infection and transmission occurs through a tick bite.

A sixty-five year old female with a past medical history of lupus and autoimmune hepatitis and immune-mediated thrombocytopenia status post splenectomy presented to the emergency department (ED) reporting malaise and fevers. She was seen at outside hospital two weeks prior for similar symptoms and diagnosed with community-acquired pneumonia and sent home on antibiotics. Initially, her symptoms improved but she soon redeveloped symptoms and worsening fevers. Her symptoms were thought to be due to adrenal insufficiency in setting of her long-term prednisone use, however the patient continued to be symptomatic despite an increase in her steroid dose and re-presented to the ED. She was found to have autoimmune hemolytic anemia and immune thrombocytopenia, a combination called Evan's syndrome, which was thought to be a manifestation of her lupus. She was treated with high dose steroids up to 1.5mg/kg, but continued to have hemolysis. On further questioning, the patient reported a trip to Maine two months prior and repeat peripheral smear revealed a large parasitemia with staining positive for Babesia.

Throughout the admission, the patient had undulating fevers to 39.4 C. She had a positive double stranded deoxyribonucleic acid antibody and her C3 and C4 complement levels were low, initially pointing to a lupus flair as the cause of her hemolysis. Her labs were significant for decreasing hemoglobin, elevated lactate dehydrogenase, and decreased haptoglobin. Her direct Coomb's test was positive. Human immunodeficiency virus, Bartonella, hepatitis and tuberculosis were negative. A computed tomography of her chest, abdomen and pelvis were also not significant for a source of infection. On a peripheral smear, pathology found evidence of Babesia with an extremely high parasitemia of > 14%. This was confirmed on her first smear as there was initial concern of a transfusion related infection. Lyme's titers were also positive

The patient was treated successfully with atovoquone and azithromycin for six weeks. While the patient's history and laboratory data pointed toward an immune mediated cause, her continued hemolysis despite high doses of steroids pointed toward another cause. This case highlights the importance of a thorough travel history.

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PACED CORONARY VASOSPASM OCCURING DURING INTRAVENOUS NITROGLYCERIN, George Kontogiannis, MD, National Capital Consortium, Bethesda MD

Introduction: Classically known as Prinzmetal's angina, vasospastic angina is a rest angina that responds well to sublingual nitrates. The Coronary Vasomotion Disorders International Study group diagnostic criteria describe a nitrate responsive angina with transient ischemic ECG changes without another obvious cause, and angiographic evidence of coronary artery spasm. However, the absence of nitrate responsiveness does not completely exclude the diagnosis. Patients with Prinzmetal's angina are often of Japanese descent and smoke.

Case: We present a 45-year-old female with past medical history of right coronary artery vasospasm, associated with AV block status post dual chamber permanent pacemaker, who presented after ROSC from a syncopal episode. Her history of vasospasm was discovered a year prior when she had syncope secondary to an inferior STEMI and complete AV block. Cardiac catheterization showed no coronary artery disease but inducible spasm. Recurrent syncopal episodes led to a permanent pacemaker. In the ED, she was continued on her home isosorbide mononitrate and verapamil. She developed chest tightness with inferior ST elevations and ventricular pacing, was started on nitro drip, and her symptoms and ECG changes resolved after 10 minutes. She was admitted to the ICU and a few hours later had a recurrence, accompanied by symptomatic hypotension. Nitro was stopped and the patient was given a fluid bolus. Her symptoms resolved after 10 minutes. On history the patient admitted to recently restarting tobacco abuse to cope with acute life stressors. She was started on Norvasc, and monitored with no recurrence.

Discussion: This case illustrates the diagnosis and treatment of refractory vasospasm with a unique consequence: complete heart block. It is refractory since it occurred while on a calcium channel blocker and a nitrate, as well as while on intravenous nitroglycerin. When such a patient presents, it is important to classify if they are a true non-responder, or if it is secondary to poor lifestyle adherence. Furthermore, the use of dual calcium channel blocker therapy is somewhat novel, having been described only in case reports. Her ECG is interesting as a learning point as well. When paced from an RV lead, the nidus of electrical impulse is inferior, and as such the QRS waves should all be inferior. In her ECG however, the inferior leads were positively deflected, correlating with a right coronary vasospasm.