New Jersey Chapter

American College of Physicians

Student Abstract Competition

2018 Submissions

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Anne Boguslavsky	Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	Teratoma of the Thymus – A Rare Finding	**INTRODUCTION:** Mediastinal masses are most commonly found in the anterior compartment. These lesions have a broad differential, including lymphoma, germ cell tumors/teratomas, thyroid tissue, and thymomas. **CASE:** A 66-year-old woman presented with cough and congestion for 1 month. She had no history of cigarette smoking, weight loss, fever, hemoptysis or shortness of breath. Her symptoms failed to resolve with over-the-counter cough medications, and broad spectrum empiric antibiotic therapy. Her vital signs were within normal limits. Her physical examination was unremarkable. A chest X-ray showed a suspected anterior mediastinal mass. A chest CT with contrast showed a 4.1x3.7x4.0 cm anterior mediastinal mass without fat or calcification without invasion of surrounding structures, and mild mass effect on the adjacent main pulmonary artery. The patient underwent a robotic resection of the mass. The histopathology showed fatty membranous tissue and a cystic mass consistent with a teratoma within thymic tissue. An 8-month follow-up chest CT showed no recurrence of the mediastinal mass. The patient remained asymptomatic. **DISCUSSION:** Mature teratomas of the thymus contain tissues from two or three of the three embryonic cell layers. Primary germ-cell tumors of the mediastinum are rare representing 1–3% of all germ cell neoplasms. Teratomas can be found to have well-differentiated tissues such as fat, hair, and teeth. They tend to be benign, slow-growing tumors. Teratomas are usually found in the gonads, retroperitoneum or sacrococcygeal region. Rarely aberrant migration of primordial germ cells during early embryological development allow them to present at extragonadal locations that are usually in or near the anatomical midline, among which the mediastinum happens to be the most common site. While usually asymptomatic and found incidentally, they may cause a number of symptoms due to mass effect such as SVC obstruction, including chest and back pain, cough, and shortness of breath. Surgical resection is the definitive treatment and is associated with a very good prognosis, with 5-year survival rates nearing 100%. The mass in our patient also contained thymic tissue. Thymomas account for about 20% of anterior mediastinum neoplasms in adults. Up to half of patients with thymomas are found to have associated myasthenia gravis. Our patient did not have myasthenia gravis. **CONCLUSION:** Teratomas of the thymus are a rare mediastinal lesion. Early diagnosis is key because they are often asymptomatic. Surgical removal carries a good prognosis.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Chandani Desai	Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	Clinically Significant Antiphospholipid Antibody Syndrome in Patient with Hepatitis C Virus Infection	**INTRODUCTION:** Antiphospholipid (aPL) antibody syndrome produces a hypercoagulable state that manifests clinically with thrombosis, pregnancy complications, and thrombocytopenia resulting in bleeding. The presence of aPL antibodies in patients with hepatitis C virus (HCV) infection is considered as an epiphenomenon lacking clinical significance. **CASE:** A 64-year-old male with chronic genotype-1 HCV, diagnosed 16 years prior, presented with virological relapse. After a failed interferon treatment, he was treated with Viekira (ombitasvir/paritaprevir/ritonavir) antiviral therapy. He successfully achieved viral suppression after completing a 12-week course. One-week later he presented with nausea and constipation refractory to suppository and laxative use. A CT-scan of the abdomen showed extensive thrombosis in the patient's main portal vein, superior mesenteric vein, and splenic vein. There was no evidence of infection or inflammation. Lab tests were mostly unremarkable except for a positive hexagonal phase phospholipid (HPP) result. During the following 8-month management with warfarin, an underlying malignancy was ruled out through a series of diagnostic tests, including MRI scans. Patient decided to discontinue warfarin, on his own, due to burdensome monitoring. Two-months later he presented with small bowel obstruction secondary to mesenteric ischemia. That was his fourth clinical presentation of thrombosis. Hypercoagulable workup demonstrated a positive lupus anticoagulant (LA), positive DRVVT and positive HPP, confirming a diagnosis of primary aPL antibody syndrome. **DISCUSSION:** Antiphosphoplipid antibody syndrome is an autoimmune hypercoagulable state more commonly affecting women and occurring in individuals with other autoimmune, or rheumatic disorders. Types of aPL syndromes include primary, secondary, and catastrophic, which presents with simultaneous multi-organ failure. Diagnosis requires at least one clinical event of thrombosis and two antibody blood tests spaced three months apart demonstrating lupus anticoagulant (LA) and/or anti-cardiolipin (aCL). Infectious diseases, including HCV, are known to induce LA or aCL antibodies. However current literature suggests that these markers are an epiphenomenon lacking true clinical significance. While aCL-antibodies are more frequently found in chronic HCV, most patients fail to present with clinical manifestations of aPL syndrome. Patients presenting with hypercoagulability are often primarily worked-up for infection, inflammation, or malignancy. Autoimmune etiologies are considered less likely than malignancy, resulting in a work-up that can take months, require hospital admissions, and create undue anxiety to the patients. Repeat antibody assessment should be considered for all patients, whether malignancy is highly suspicious, or not. These test results offer a cost-effective and timely diagnosis for the patients, possibly preventing the fatal complications associated with an autoimmune hypercoagulable state including stroke, myocardial infarction, renal failure, pulmonary embolism, and most importantly, catastrophic aPL antibody syndrome. **CONCLUSION:** Our case demonstrates that aPL antibodies may not always be an epiphenomenon in HCV infection.

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Clinical Vignette	Nicholas Hoell	Olga Schweiker-Kahn MD, Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	A Case of Parsonage-Turner Syndrome	**INTRODUCTION:** Parsonage-Turner Syndrome (PTS) is a rare condition presenting with severe, acute-onset pain, sensory loss, and weakness of the shoulder and/or the upper extremity. Hereditary and idiopathic forms of PTS have been described, the latter being associated with chemotherapy, infections, vaccinations, pregnancy, surgery, and exercise. **CASE DESCRIPTION:** A 45-year-old, left hand dominant, college professor with a history of lymphoma in remission post chemotherapy presented 10 days after the acute onset of left arm weakness associated with 9/10 left shoulder pain refractory to ibuprofen. The patient denied headache, visual changes, nausea or vomiting, chest pain, shoulder, neck or arm injury, or other acute event. His vital signs were within normal limits. Physical examination was only abnormal for left shoulder and proximal arm weakness (power 2/5), decreased range of motion, and diffuse sensory deficit. His grip strength was preserved. A cervical radiculopathy was suspected. Imaging studies of the cervical spine and left shoulder were unremarkable. He was treated with oral corticosteroids, NSAIDs, tramadol and physical therapy. One month after presentation, neither the patient's strength nor pain showed significant improvement. An EMG study showed diffuse denervation in the left C5-C6 distribution suggestive of brachial plexopathy. Two months from the presentation, with continued physical therapy, the patient reported complete resolution of his pain and near complete resolution of his weakness. **DISCUSSION:** Parsonage-Turner Syndrome (also referred to as brachial plexitis, brachial neuropathy, and brachial radiculitis) affects men more commonly than women with a reported incidence of 1.64 cases per 100,000 person-years. The idiopathic form is associated with viral infections (25% to 50%) and recent vaccination (15%). Chemotherapy, radiation, surgery, pregnancy, and exercise have also been associated with PTS. The hereditary form results from autosomal dominant mutations in the Septin 9, manifesting in recurrent episodes. PTS is heralded by severe, acute-onset pain in 95% of patients. Pain is typically localized to the shoulder and proximal arm, but may extend to involve the neck and distal arm. The onset of weakness and sensory deficits may lag behind the onset of pain. No lab test has been found to be diagnostic, but positive sharp waves and fibrillation potentials on EMG as well as T2 hyperintensities on MRI may favor the diagnosis. Treatment for PTS is conservative with NSAIDs for analgesia. Early administration of corticosteroids has been suggested to reduce pain and the time course of disease, but remains unproven. Physical therapy is beneficial in recovering strength and range of motion. PTS is ultimately a time-limited disease with excellent prognosis. About 60% of brachial plexus lesions resolve in less than one year. **CONCLUSION:** This case demonstrates the need for a high index of suspicion in identifying PTS, which yields a favorable prognosis despite its alarming presentation.

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Clinical Research	Ryan Mathern		Cooper University Hospital (Brian Gable)	Rate of Force Relaxation-Scaling Factor and Its' Clinical Utility	**INTRODUCTION:** Ability to generate quick submaximal muscle forces followed by quick relaxations is not only important in activities that require consecutive agonist and antagonist contractions but it is also crucial for postural control and fall prevention. Variables such as rate of force development (RFD) and RFD-Scaling Factor (RFD-SF) have high day-to-day reliability and have demonstrated clinical significance when analyzing patients with MS and Parkinson. **PURPOSE:** In aging, and diseases such as Parkinson's and Myotonic Dystrophy Type I, relaxation is slowed or malfunctioning. Given the power of RFD-SF, it is important to quantify the ability of muscle relaxation after a quick submaximal force generation. Our purpose was to quantify this through the development of a new variable, the rate of Relaxation-Scaling Factor (RFR-SF) and to show comparable utility to RFD-SF. **METHODS:** Thirteen healthy adults were tested in grip force (GF), elbow extension (EE), and knee extension (KE) on two different days separated by one day. The slope, R2, and y-intercept were calculated for each individual and their corresponding muscle groups. However, out of these calculations, the slope RFD-SF and RFR-SF were of most importance. They were obtained from the relationship between peak force and the corresponding peak RFD and peak RFR representing RFD-SF and RFR-SF, respectively. The reliability of RFD-SF and RFR-SF were determined using the intra-class correlation and the test-retest design. **RESULTS:** Results of a two-way ANOVA (3 muscles and 2 scaling factors) indicated that RFR-SF was smaller than RFD-SF in all of the selected muscles ($p<0.001$). Bonferroni corrected pairwise comparisons revealed that RFD-SF obtained from GF and KE were similar and both of them were smaller than those obtained from EE. Regarding RFR-SF, those obtained from EE and GF were similar, and they were higher than the ones obtained from KE. The reliability of RFR-SF obtained from the studied muscle groups was fair to high (ICCs of GF, EE, and KE were 0.67, 0.6, and 0.74, respectively). **CONCLUSION:** Rate of force relaxation scaling factor was extracted from the brief force production tasks of three muscle groups. RFR-SF was similar to RFD-SF indicating a potential variable that quantifies neuromuscular quickness Future studies should develop day to day reliability of RFR-SF and assess the variable in clinically significant populations.

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Clinical Vignette	Jenna McClane	Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	Capgras Syndrome in Parksinson's Disease: An Imposturous Account	**INTRODUCTION:** Capgras Syndrome (CS) is a delusional misindentification syndrome that most commonly presents in patients with dementia. **CASE PRESENTATION:** A 72-year-old man with Parkinson's disease (PD) was brought by his wife for abnormal behavior for one month. According to her the patient has been repeatedly telling her to leave his house as she was not his wife. He had been making phone calls to his son inquiring about his wife and complaining that someone in his house claims that she was his wife. When his son arrived, he claimed that he was an imposter and not his son. The patient had been stable on amantadine and carbidopa-levodopa therapy for 5 years. He had a recent evaluation by his neurologist which concluded as stable. There was no history of travel, fever, headache, double vision, vomiting, weakness in the extremities, urinary or bowel incontinence, change in medication, or fall. His vital signs were within normal limits. The patient was alert and awake. He had a flat affect, mild cogwheel rigidity and slow but steady gait. The rest of the physical examination was unremarkable. He was referred to the emergency room. His complete blood count, complete metabolic panel, urinalysis, serum ammonia level, CSF examination and brain MRI were within normal limits. A detailed neuropsychology evaluation concluded Capgras syndrome. Patient was treated with quetiapine and supportive care. After 3 months, patient's wife reported that barring occasional episodes his behavior was normal. **DISCUSSION:** CS has been identified in patients with both psychiatric diagnoses and organic brain lesions, and is typically characterized by the belief that one's spouse or close relative has been replaced by an imposter. Less commonly it is associated with idiopathic PD, and in almost all reported cases of CS in PD, there is evidence of preexisting cognitive impairment or dementia. Case reports have documented the emergence of CS following increases in dose of dopamine agonists and/or levodopa. CS-like delusions following methamphetamine use have also been reported, suggesting the role of dopamine dysregulation in the pathophysiology of CS. Further, evaluation of patients with CS has shown a decreased autonomic response to faces of close relatives, implying the neocortical regions involved with emotional attribution as a possible etiological target. Taken together, the dopaminergic pathways that connect facial recognition and emotional response are likely primed by degenerative lesions and other insults for disruption, specifically by drugs and other disease-modifying agents. Atypical antipsychotics, such as Quetiapine, are effective in treating CS. **CONCLUSION:** CS can present in both psychiatric and neurodegenerative disease. The symptoms are distressing both to the patient and his or her family and friends. Though its etiology is uncertain, patients can be successfully managed with atypical antipsychotics.

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Clinical Vignette	Kristina Moller		Cooper University Hospital (Brian Gable)	Bowels on the Brain	**INTRODUCTION:** Irritable bowel disease (IBD) presents in a multitude of ways in the pediatric patient. **CASE PRESENTATION:** A 9 year old male presented to the hospital complaining of new onset scrotal swelling and puffiness around his eyes. He also endorsed constipation and diarrhea for the past 4 years. Limited PCP records showed that he had gained 9 pounds in the past 4 years. His current height and weight were below the 5th percentile. On physical exam, he was noted to be pale and thin. His mucosa, palms and conjunctivae were pale. He had significant periorbital, scrotal, penile and lower extremity edema. His abdomen was distended and he had a positive fluid wave. Initial labs showed an overlapping iron deficiency anemia and anemia of chronic disease. He had microabluminemia and his fecal occult blood test was positive. The patient was worked up for conditions such as lupus, celiac disease and nephrotic syndrome; all tests were negative. The patient was set up to have an endoscopy with small bowel follow through. However, during the exam he had a seizure. The patient was stabilized and sent for imaging which showed extensive thrombosis in the right jugular vein and multiple venous sinuses as well as chronic brain atrophy and multiple acute venous hemorrhagic infarcts. The patient was transferred to the PICU and began anticoagulation. A full thrombophilia workup came back negative. The patient continued to improve and returned to baseline at which point an endoscopy and colonoscopy were performed confirming a diagnosis of IBD. The colonoscopy was impeded by impaction and only a biopsy from the rectosigmoid area was obtained. The patient was discharged from the hospital and has since been treated with Sulfasalazine, Iron and Folic acid as well as continued on Lovenox and Keppra. His iron studies have normalized along with his ESR and CRP. The patient has begun to gain weight. **DISCUSSION:** IBD has many complications including fistulas, decreased growth velocity and delayed sexual maturation. One of the more rare complications is venous thrombosis. In this patient, the unusual location of the clots with no detected underlying coagulation disorder is unique. It is well known that inflammation can predispose to coagulation. Additionally, the dysregulation of vascular endothelium in IBD is thought to predispose to platelet adhesion and aggregation. However, a more novel theory points to the severe iron deficiency as a cause. Studies show that platelets produced in iron deficient environments displayed a higher mean platelet volume and increased aggregation as well as accelerated megakaryocyte differentiation in the bone marrow. **CONCLUSION:** This case demonstrates an unusual connection between IBD, severe iron deficiency anemia and a predisposition to cerebral venous sinus thrombosis.

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Clinical Vignette	Lakeisha Mulugeta-Gordon	Deborah Park MD, Shivani Gandhi DO, Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	Warfarin Related Nephropathy in a Patient with Underlying Chronic Kidney Disease	**INTRODUCTION:** Approximately 4 million people in the United States are currently taking warfarin for anti-thromboembolic therapy. Warfarin is known to be a culprit for bleeding, but acute kidney injury due to warfarin itself has been more recently described as warfarin related nephropathy (WRN). **CASE PRESENTATION:** A 66 year-old male with history of laryngeal cancer status post radiation, COPD, hypertension and CKD-III presented with acute onset shortness of breath. His physical examination was remarkable for atrial fibrillation with heart rate of 180 beats per minute. The rest of the vital signs were within normal range. The rest of the examination was normal. His electrocardiogram showed atrial fibrillation with increased heart rate. His serum creatinine was 4.0 mg/dL, BUN 45 mg/dL, hemoglobin 11.5 g/dL. The rest of the complete metabolic panel, complete blood count, troponins, TSH wee within normal limits. Patient received medical management for rate control for atrial fibrillation with rapid ventricular rate, followed by warfarin 5 mg daily therapy. After 5 days his INR increased to 3.2 and serum creatinine increased to 8.0 mg/dL. The etiology of his rapid decline in renal function remained unclear even after extensive diagnostic workup, including normal renal ultrasonography. A renal biopsy showed mild diffuse mesangial proliferative glomerulonephropathy with multifocal RBC casts and diffuse acute tubular injury. A diagnosis of WRN was made. Patient was started on hemodialysis and subsequently he remained stable and hemodialysis dependent. **DISCUSSION:** Since 2009, cases of unexplained acute kidney injury has revealed a rare and novel entity called WRN. WRN is defined as a rise in serum creatinine by 0.3 mg/dL with concomitant rise of INR greater than 3.0. Histologic findings include presence of glomerular hemorrhage and acute tubular obstruction from RBC casts. WRN occurs more frequently in patients who have underlying chronic kidney disease (CKD) but can also occur in non-CKD patients. Warfarin increases the risk of hemorrhage in the microvasculature of the kidneys causing progressively worsening renal disease, especially in patients with underlying CKD. Patients diagnosed with WRN have been noted to have increased mortality within two months of diagnosis, or they develop accelerated progression of CKD. A retrospective analyses of patients who experienced over-anticoagulation (INR>3) 37% had unexplained AKI, making WRN a reasonable possible etiology and may be more prevalent than previously thought. Given the risks of WRN, a cautious approach towards the dosing of warfarin in the setting of CKD may prove to improve mortality rate of CKD patients receiving warfarin therapy. **CONCLUSION:** Anticoagulation with warfarin has been a standard therapy for stroke prevention and treatment of thromboembolic diseases. It is important for the medical community and our patients to understand the risks associated with its use prior to initiating therapy, especially WRN.

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Clinical Vignette	Spencer Ng	Dawnette Urcuyo, MD	Cooper University Hospital (Brian Gable)	A Case of Pembrolizumab Induced Diabetic Ketoacidosis in a Patient with Tonsillar Squamous Cell Carcinoma	A 60-year-old male with a diagnosis of metastatic squamous cell carcinoma of the right tonsil (T1N2Mx) and papillary thyroid carcinoma (TxN1Mx) on current treatment with Pembrolizumab presented with a two-day history of increased urinary urgency and frequency. He was initially diagnosed with tonsillar squamous cell carcinoma in 2016 and was treated with Cisplatin and radiation therapy. His cancer persisted despite three months of treatment. Palliative immunotherapy with Pembrolizumab began in September 2017. The patient presented to the hospital with the symptoms of urinary incontinence and increased urgency after receiving his second cycle of Pembrolizumab. On admission to the hospital, his blood glucose was 536mg/dL, anion gap of 28 and a beta-hydroxybutyrate level of 6.18mmol/L. He was found to have an HbA1c of 7.9%. Prior to his treatment, his random blood glucose readings ranged from 85-104mg/dL. He had no personal or family history of diabetes. Islet Cell Antibodies and Insulin Auto-Antibodies (<0.4U/ml) were negative. However, he did have a mildly elevated anti-glutamic acid decarboxylase antibodies [anti-GAD 6IU/ml (reference <5IU/ml)]. His hospital course was uncomplicated and his urinary symptoms resolved after treatment of hyperglycemia. He was started on both long-acting insulin glargine and prandial short-acting insulin with improved glycemic control upon discharge. Patient did well after discharge but Pembrolizumab therapy was held due to uncontrolled diabetes. **DISCUSSION:** Pembrolizumab is an IgG4 monoclonal antibody that targets PD-1. It is approved for the treatment of metastatic melanoma, renal cell carcinoma and non-small cell lung cancer. It has been associated with the development of type 1 diabetes mellitus in seven patients (incidence of 0.1%). New diagnosis of diabetes occurred between one week and fifty-one weeks after receiving pembrolizumab. The association of immune related adverse events with the use of immune checkpoint inhibitors has been increasingly recognized. Besides type 1 diabetes mellitus, thyroid disorders, hypophysitis and adrenal insufficiency have occurred. Here, we describe a case of diabetes ketoacidosis in a patient who had recently started pembrolizumab therapy. In most cases, the patient had positive anti-GAD and ICA, suggesting an association of PD-1 inhibitors with autoimmune diabetes. Of note, diabetes does not seem to resolve spontaneously after termination of the treatment and requires long term insulin therapy. **CONCLUSION:** The PD-1 inhibitors have been associated with multiple autoimmune adverse events. Although rare, pembrolizumab induced autoimmune diabetes can occur and has become more frequently recognized in recent years.

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Clinical Vignette	Jessica Richmond	Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Nickel Dental Implant and Recurrent Asthma	**INTRODUCTION:** Bronchial asthma is a common clinical presentation which may have several identifiable and non-identifiable trigger factors. **CASE PRESENTATION:** A 15-year-old female with asthma presented with progressive worsening of coughing and vomiting during the night, occasional wheezing, and a scaly pruritic rash on the dorsum of her hands. Patient had suffered multiple exacerbations of asthma throughout her life. Her medications included albuterol multi-dose inhaler, inhaled corticosteroids, and montelukast, without much relief of her symptoms. Her mother recalled worsening of her symptoms weeks after a dental procedure; which required a nickel crown. Due to the pruritic rash on her hand and worsening of her asthma symptoms, she was tested for nickel allergy; which was positive. Her dental crown was removed. She started seeing significant improvement after 6 weeks of removal. A year later, the patient no longer required medical management for asthma. She experienced only one asthma attack since the removal of the cap, but remained asymptomatic subsequently. **DISCUSSION:** Bronchial asthma has many common triggers such as exercise, infections, and allergens (dust, pollen, animal dander). However, other triggers such as nickel are often more associated with contact dermatitis and rarely considered when it comes to asthma exacerbations. Nickel is found almost everywhere from jewelry, coins, and cellphones to braces, dental crowns, stents, and knee replacement implants. No routine testing for nickel allergy exists in the general or asthmatic population and the exact prevalence in the U.S. population is unknown to justify such testing. In one study a significant difference in worsening of nasal flow was observed in patients with nickel allergy before exposure to nickel and after the nickel provocation test. Avoidance of nickel exposure in this group resulted in reduction in nasal and bronchial symptoms, such as cough and dyspnea, as well as improvement in peak expiratory flow and FEV1. This showed that there may be a correlation between nickel allergies and obstructive airway disorders. **CONCLUSION:** This case demonstrates the importance of paying attention to common preventable environmental factors, such as nickel exposure, when it comes to treating asthma in order to improve quality of life and reduce the need for medications.

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Clinical Vignette	Michelle Safferman	Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	When Diarrhea Dominates, Check the Duodenum for Rare Possibilities	**INTRODUCTION:** Chronic refractory diarrhea can be challenging in terms of finding an accurate diagnosis, which at times, can be due to a rare but correctable etiology. **CASE:** A 43-year-old Caucasian male with hypercholesterolemia and history of injection heroin abuse on methadone maintenance presented with intermittent, crampy abdominal pain and 5-6 watery bowel movements daily for the previous 6 months. He was taking Atropine-Diphenoxylate to manage his symptoms with minimal relief. He denied fever, heartburn, weight loss, hematochezia, melena, anorexia, mucous or blood in stool, or recent international travel. His vital were within normal limits. Physical examination revealed a soft, non-tender, non-distended abdomen. His complete metabolic panel, complete blood count, thyroid stimulating hormone and erythrocyte sedimentation rate were unremarkable. Abdominal ultrasound revealed no abnormality. CT abdomen identified a filling defect within the proximal small intestine. Follow-up small bowel series localized the filling defect to the 4th portion of the duodenum. Stool evaluation revealed borderline elevation in fat content with a normal WBC count and negative culture and ova and parasite. Stool osmolality was less than 50 mOsm/kg, suggesting a secretory form of diarrhea. Twenty-four hour urine 5-HIAA was elevated at 32 mg and octreotide scan demonstrated increased uptake in duodenum. EGD and biopsy of distal duodenum demonstrated tumor cells which were markedly positive for CD56, synaptophysin, and chromogranin consistent with a carcinoid tumor. He underwent resection of the distal portion of the duodenum and proximal jejunum with removal of 2.6 cm tumor with pathology consistent with grade I neuroendocrine carcinoma with no angiolymphatic invasion. Since resection, the patient has remained asymptomatic. **DISCUSSION:** Carcinoid tumors are a relatively rare, well-differentiated neuroendocrine tumor arising from enterochromaffin cells (EC). The age-adjusted incidence for non-pancreatic primary carcinoid tumors is 4.7 per 100,000. Most carcinoid tumors originate in the gastrointestinal tract (73.7%), with the appendix being the most commonly involved organ; the remainder of tumors originate in the respiratory system (25.1%) and reproductive system (0.56%). Typically, carcinoid tumors are discovered incidentally as many patients are asymptomatic. However, carcinoid tumors can secrete bioactive substances including serotonin and kallikrein. 5-hydroxyindoleacetic acid (5-HIAA) is a degradation product of serotonin that is often elevated in the urine of patients with carcinoid tumors. Carcinoid syndrome is seen in 5% of patients with carcinoid tumors and occurs when bioactive substances enter systemic circulation; patients can present with skin flushing, diarrhea, bronchoconstriction, and secondary restrictive cardiomyopathy. This is an uncommon presentation with primary gastrointestinal carcinoid tumors due to hepatic degradation of vasoactive substances prior to their entry into systemic circulation. **CONCLUSION:** Duodenal carcinoid tumors are a rare cause of chronic diarrhea. Confirmation of secretory diarrhea by stool osmolality assists in further work-up. Early diagnosis and treatment results into favorable outcome.

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Clinical Vignette	Colin Sperling	Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Hard to Swallow: An Unusual Cause of Esophageal Dysphagia	**INTRODUCTION:** Dysphagia is prevalent clinical entity in the geriatric population with a plethora of etiologies ranging from common to extremely rare. **CASE PRESENTATION:** An 83-year-old female presented with progressive dysphagia. Her symptoms began more than 1 year prior to presentation and became progressively worse within the preceding 3 months. Consumption of soft foods such as mashed potatoes and scrambled eggs were particularly disconcerting. She denied weight loss, fatigue, cough, dyspnea, heartburn, abdominal pain, nausea, vomiting, pyrosis, melena, or diarrhea. Her past medical history was significant for stage IIB lung adenocarcinoma 9 years prior, upon which a lobectomy of the right upper and middle lobes was performed. Social history was positive for a 60 pack-year smoking history, with no reported alcohol or drug use. Her vitals were within normal limits. Her physical examination was unremarkable. Her complete blood count was within normal rage. Her endoscopicgastroduodenoscopy was unremarkable. A modified barium swallow was performed with a variety of textures, including nectar, pudding, and solids. This revealed a partial obstruction during the esophageal phase of swallowing due to a large ventrally protruding cervical osteophyte at the inferior endplate of C5. The patient was then instructed to turn her head to the right while swallowing, which drastically reduced barium residue above the crycopharyngeus. This position allowed subsequent boluses with varying consistencies to more easily traverse the esophagus with minimal evidence of retention. **DISCUSSION:** Esophageal dysphagia is a relatively common clinical diagnosis with a wide range of possible etiologies. These include structural irregularities such as strictures or malignancy, motility dysfunction secondary to achalasia or scleroderma, or extrinsic compression from a dilated left atrium or an anteriorly projecting cervical osteophyte. With an estimated prevalence in the adult population as high as 15%, some studies suggest that up to 25 per 100,000 individuals are diagnosed with dysphagia each year. Asymptomatic cervical osteophytes are quite common in the elderly population, occurring in up to 30% of individuals. Dysphagia is also most prevalent in this age group, with estimates as high as 38%. Cases of cervical osteophytes resulting in esophageal dysphagia are sparsely reported in the literature, although a number of case reports have described associations with Diffuse Idiopathic Skeletal Hyperostosis and Cervical Spondylosis. A modified barium swallow has the advantage of assessing both anatomical and functional characteristics of the esophagus. Medical versus surgical intervention should take into account the degree of dysfunction. The patient described here opted for conservative treatment with turning her head to the right while swallowing solids. She subsequently remained asymptomatic. **CONCLUSION:** The presence of a protruding cervical osteophyte should be considered in the setting of isolated esophageal dysphagia in the absence of any constitutional symptoms.

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Clinical Vignette	Swar Vimawala	Satyajeet Roy MD FACP	Cooper University Hospital (Brian Gable)	A Case of Posner-Schlossman Syndrome	**INTRODUCTION:** Acute onset unilateral eye pain has a myriad of etiologies. **CASE:** A 48-year-old female patient with no significant past medical history presented with acute unilateral severe ocular pain in right eye accompanied by ipsilateral watering, mild nausea, headache, and severe photophobia. She denied cigarette smoking, alcohol use, substance use, travel, contact lens wear, eye makeup use, or new medication intake. On exam, her vital signs were within normal limits, and exam was essentially benign other than circumciliary congestion in her right eye. A CT head and MRI brain with contrast were unremarkable. Detailed ophthalmologic exam revealed right intraocular pressure (IOP) of 46 mmHg and a normal left IOP. Slit lamp exam did not reveal glaucomatous cupping, keratic precipitates, or synechiae. The cornea was normal. Laboratory findings on comprehensive metabolic panel, complete blood count, and erythrocyte sedimentation rate were normal. A clinical diagnosis of Posner-Schlossman Syndrome (PSS) or glaucomacyclitic crisis was established, and she was treated with ophthalmic steroids and brimonidine. Within one day, her right IOP decreased to 25 mmHg and then to 19 mmHg. There was no recurrence. She remained asymptomatic subsequently, and recurrence free at 2 year follow-up. **DISCUSSION:** Characteristically, the highest incidence of PSS is between the ages 20-29 and is male predominant (50.5-71.4%). Furthermore, the mean IOP in studies range from 42.77 mmHg to 49.2 mmHg. Clinically, patients present with recurrent attacks of mild, unilateral, nongranulomatous, anterior uveitis and elevated intraocular pressure. Vision may be affected, but anterior chamber drainage angles remain open and optic disk appearance is normal. The differential diagnosis is broad, including acute angle closure glaucoma (fixed, dilated pupil), and Fuchs uveitis syndrome (nonresponsive to steroids). Diseases other than PSS predisposing patients to glaucoma include sarcoid, HSV, Behcet's, HLAB27 uveitis, Vogt-Koyanagi-Harada disease, and human T-lymphotropic virus type 1 uveitis. However, PSS is typically consistent with mild iridocyclitis and absence of posterior or peripheral anterior synechiae. Treatment of PSS includes both medical and surgical. First-line medical management involves the use of topical steroid and IOP-lowering medications such as a carbonic anhydrase inhibitor. If no improvement occurs, then topical valganciclovir for 1 month is appropriate as patients with PSS unresponsive to steroids are likely to have CMV infection of anterior chamber. The suggested surgical treatment for PSS is antimetabolite-augmented trabeculectomy and is indicated for patients with elevated IOP uncontrolled medically. The success rate is 82.86%. A patient with flare-ups of PSS could result in long-term sequelae such as glaucoma, optic nerve atrophy, and non-arteritic anterior ischemic optic neuropathy. PSS is usually not encountered in patients older than 50 years. **CONCLUSION:** Acute onset unilateral eye pain can be secondary to PSS. Early diagnosis and management can lead to a favorable outcome.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Matthew Norris	Dr. Christopher Kuriakose	Overlook Medical Center (Jeff Brensilver)	A Case of Ashwagandha Supplementation Preceding Onset of Optic Neuritis and Discovery of Demyelinating Disease	**INTRODUCTION:** In the United States, dietary supplements are available, accessible, and popular. Over half of American adults use them. Most people are self-medicating without their doctor's advice or knowledge. Although proposed benefits of supplements are often well-advertised, possible drug-drug and drug-disease interactions are often poorly understood by both patients and their physicians. This case highlights how a woman with a history of autoimmune diseases began self-medicating with a supplement called Ashwagandha that she was unaware had immune-stimulating properties and subsequently developed an unrelated autoimmune condition that revealed a different underlying autoimmune disease. **CASE DESCRIPTION**: A 28-year-old female with celiac disease and systemic sclerosis presented to our emergency department with complaints of blurred vision in her left eye of ten-day duration. This was associated with dyscromatopsia, painful eye movements, and a central scotoma of one-day duration. On physical exam of her left eye, visual acuity was worse than 20/200; furthermore, both a central visual field deficit and left afferent pupillary deficit were present. Although not taking medications, she had started taking Ashwagandha 800mg twice daily two weeks prior to onset of visual symptoms and continued their use up until time of admission. She was diagnosed with optic neuritis and admitted for treatment with IV methylprednisolone. During her hospitalization, she was evaluated for multiple sclerosis. Head MRI showed scattered foci of high FLAIR signal intensity within the cerebral white matter with several foci visualized in the periventricular region. A MRI C-spine showed foci of T2 hyperintensity within the cervical spinal cord at the C4 and C5 vertebral levels consistent with demyelinating plaques in a patient with multiple sclerosis. Of importance, there was no abnormal enhancement in the MRI C-spine suggestive of active demyelination. After four days, most of her symptoms resolved. She was advised to follow up with neurology and to discontinue Ashwagandha. **DISCUSSION:** Ashwagandha is a plant that has been found in vitro to have immune-stimulating properties. It has been shown to selectively stimulate Th1 immunity as well as enhance proliferation of CD4+/CD8+ and NK cells. Phytochemicals in Ashwagandha that contribute to Th1 immune polarization have also been identified. Optic neuritis is characterized in the acute phase by predominant T-cell activation with release of pro-inflammatory cytokines. It follows that a disease characterized by activation of T cells might be affected by a drug with Th1-predominant response. It is important to recognize our patient now has multiple autoimmune conditions characterized by T cell stimulation. Two studies link both multiple sclerosis and systemic scleroderma through a polymorphism at CD86. A polymorphism could further explain why Ashwagandha supplementation either initiated or aggravated a case of subclinical optic neuritis. This case emphasizes the need for patients to exercise caution before beginning a new supplement.