A Rare Case of Spontaneous Pneumothorax in a Young Woman

Lindsay A. Lucas¹, Tariq Cheema²,¹¹ Medicine, West Penn Allegheny General Health System, Pittsburgh, PA, United States. ²Pulmonary and Critical Care Medicine, Allegheny General Hospital, Pittsburgh, PA, United States. (Control ID: 1322870)

Learning Objective 1: Recognize the clinical features of thoracic endometriosis.
Learning Objective 2: Describe the hypothesized pathophysiology behind catamenial pneumothorax.

Case: A 20-year-old woman presented with shortness of breath and stabbing left-sided chest pain that started at a concert that night. Pain was constant, worse with inspiration, and associated with anxiety. She is a nonsmoker, and denied trauma or recent illness. History was notable for congenital hypothyroidism and three spontaneous pneumothoraces, the first of which occurred at age 16. Family history was significant for endometriosis in her mother. In the last 18 months, she was hospitalized twice for pneumothoraces requiring chest tube decompression and had been to the emergency department several times for panic attacks with similar pain. Recently she left college and returned home due to missed classes and anxiety. Her only medication was levothyroxine. Physical exam disclosed a tall, anxious-appearing, thin young woman, BMI 18. She was mildly hypoxic on room air. Increased work of breathing and decreased breath sounds over the left apex were noted. Chest radiograph revealed a large pneumothorax with collapse of the left upper lobe. A chest tube was placed in the ER, and she underwent video-assisted thoracic surgery the next morning for pleurodesis, wedge resection of the bleb, and pleural biopsy. Alpha-1 antitrypsin, CBC, CMP, and thyroid studies were normal. Pathology revealed nonspecific fibrosis and chronic inflammation. Further history revealed she was menstruating prior to the onset of symptoms. She was referred to gynecology and started on oral contraceptives for catamenial pneumothorax. At six month follow up she has had no further pneumothoraces, anxiety has improved, and she doing well in community college.

Discussion: Catamenial pneumothorax (CP) and catamenial hemothorax are uncommon clinical entities related to thoracic endometriosis, characterized by recurrent pneumo- or hemothorax within 72 hours of menstruation. Once thought to be rare, new data suggest thoracic endometriosis is under recognized as a cause of secondary pneumothorax. In three large studies of recurrent pneumothorax in women of reproductive age, prevalence of CP ranged from less than 1% to 24.6%. Little is known about the exact mechanism by which CP occurs. Histological evidence of endometrial tissue is identified in less than 13% of biopsied blebs, suggesting the mechanism of injury is not necessarily related to sloughing of endometrial implants on visceral pleura, but may also include indirect effects of hormones or prostaglandins on existing blebs, and progressive destruction of alveoli from circulating endometrial cells that enter the thoracic cavity through congenital diaphragmatic defects. CP should be considered a clinical rather than histopathological diagnosis. Initial treatment includes rest for mild cases and thoracostomy for larger lesions. Long-term management is not well defined due to limited identification of the condition. Invasive strategies include pleurectomy, pleurodesis, diaphragmatic defect repair, or ablation of endometrial implants. Medical therapies are aimed at hormonal management including oral contraceptives and GnRH agonists. Thoracic endometriosis is a rare but potentially treatable cause of recurrent hemothorax/pneumothorax in women of reproductive age, and a source of significant stress on those affected. Identification and continued research will be the keys to identifying optimal treatment in the future.
An unfortunate complication of anti-platelet therapy: A case report

Subhraleena Das1, Sujith Cherian1, Wasim A. Hamarneh1, Ehtesham Ul Haq1, David Landsberg1 1Internal Medicine, SUNY Upstate Medical University, Syracuse, NY, United States. (Control ID: 1339683)

Learning Objective 1: Recognize the rare but potentially fatal complication of diffuse alveolar hemorrhage (DAH) with clopidogrel.

Learning Objective 2: Manage clopidogrel induced DAH.

Case: A 72-year-old man was transferred to the ICU with hemoptysis and dyspnea. The patient had been admitted for an elective carotid endarterectomy a day prior and had been given 150 mg of clopidogrel before the procedure and was continued on aspirin and clopidogrel post procedure. Examination revealed tachypnea and bilateral rales. A Chest X-ray and CT scan of thorax revealed bilateral alveolar infiltrates. Both aspirin and clopidogrel were held, with improvement in respiratory status. Aspirin was restarted without complications. However, an attempt to restart clopidogrel resulted in sudden respiratory deterioration. The patient was started on pulse dose steroids with no improvement and was subsequently intubated. 2 D echo revealed diastolic dysfunction, vasculitis workup was negative for ANA, ANCA, anti-GBM antibody with normal complement levels. Flexible bronchoscopy revealed blood throughout the broncho-alveolar tree. Broncho-alveolar lavage was negative for infectious agents and cytology negative for malignant cells. Clopidogrel was again held with marked improvement, thus confirming the diagnosis of clopidogrel induced DAH.

Discussion: Clopidogrel is a thienopyridine derivative used to inhibit platelet aggregation by irreversible binding of adenosine diphosphate to the low affinity type 2 platelet receptors. It is commonly associated with increased incidences of gastrointestinal and intracerebral hemorrhage, and very rarely with DAH. DAH is most likely, an idiosyncratic hypersensitive response to clopidogrel, with a potential fatal outcome. Diagnosis is established by demonstrating a temporal relationship with the drug and ruling out other infectious, vasculitic and neoplastic processes by bronchoscopy. Due to the long half life (277-433 hours) of the drug, even after stopping the drug, DAH may worsen. Therefore, supportive treatment with mechanical ventilation, if needed, is key. Of note, platelet infusions are futile as donor platelets are also irreversibly inhibited by clopidogrel in the system. Thus, our case highlights the importance of identifying DAH secondary to clopidogrel, which if not diagnosed in time may prove to be fatal. We reiterate the need for maintaining a high index of suspicion in these patients as it may easily be overlooked as pulmonary edema in the setting of acute coronary syndrome, where the drug is frequently used.
My Teeth Won’t Let Me Eat

Ravi J. Patel¹, James M. Sosman¹¹ Internal Medicine, University of Wisconsin Hospitals and Clinics, Madison, WI, United States. (Control ID: 1334808)

Learning Objective 1: To recognize the differential diagnosis for a patient presenting with trismus.
Learning Objective 2: To distinguish disease manifestations and review management of a patient with Tetanus.

Case: A 78 year-old female presented to the ED by EMS after being found on the floor of her home by neighbors. She was found unresponsive with possible seizure-like activity. An oral airway was initially placed, but removed after improvement in her mental status. The patient lived in unsanitary conditions with a dirt floor and each room filled with hoarded items. In the ED, the patient complained “my teeth won’t let me eat.” She was hungry but stopped eating due to fear of choking. She also complained of weakness and sacral pain due to a fall. Her VS: BP 138/48, HR 84, RR 22, T 37.8°C. Her exam revealed episodes of interrupted speech due to clenching her jaw which lasted only a few seconds. She had no other neurologic deficits. She had no visible wounds, however her feet and toe-nails were caked with dirt. Her labs revealed WBC 12.0 K/uL, CK 2369 (N 0-175), troponin 0.29 (N 0-0.05), Ca 9.1, BUN 60, Cr 1.6. EKG showed anterolateral T wave flattening and the CXR had RML infiltrates. Initial therapy included a heparin drip for possible NSTEMI and IV Ceftriaxone for aspiration pneumonia. The differential diagnosis included Tetanus, so she was begun on IV Metronidazole and Tetanus IG which required several hours to obtain sufficient doses from other health care facilities. Subsequent tests revealed a normal TTE and EEG negative for seizure-like activity. The patient's symptoms quickly progressed with frequent episodes of trismus along with apnea and hypoxemia. She also developed spastic contractions of her upper extremities, muscle rigidity, and 1st and 2nd degree AV block with 5 second pauses. The patient was given muscle relaxants, and 3000 units of IM Tetanus IG. She refused intubation or CPR. She developed progressive apnea and bradycardia despite atropine and died within 48 hours. Her blood cultures revealed no growth.

Discussion: Tetanus is a disorder caused by the toxin producing anaerobe Clostridium tetani. Now rare in the developed world, the CDC estimates an annual incidence of 1 per 10 million people in the US, and 2.3 per 10 million in those ages > 65. The bacterium is ubiquitous in soil and remains a threat to all unvaccinated people. Infection occurs through penetrating injury with a foreign body, or within devitalized tissue. In 10% of cases, no cause is identified. C. tetani spores produce tetanospasmin that irreversibly binds to receptors of anterior horn cells. This results in autonomic instability, increased muscle tone, and severe spasms. Tetanus is a clinical diagnosis with 50% of cases presenting with trismus, a forceful spasm of the masseter muscle. Differential diagnosis includes Strychnine poisoning, drug-induced dystonias, neuroleptic malignant syndrome, and Stiff-person Syndrome. Treatment includes wound care and antibiotics to halt toxin production, neutralizing unbound toxin with Tetanus IG, sedatives to control spasms, and intubation with paralysis for weeks. Though the majority of patients survive with optimal management, mortality is as high as 50% in those who are treated conservatively. Unfortunately, our patient had not received health care or vaccinations for over two decades. This case underscores the importance of continued tetanus vaccination even during a time of advanced medical care.
Duodenal Diverticula and Acute Pancreatitis

Sandipani Sandilya¹, Andrea Porrovecchio²¹ Internal Medicine, Montefiore Medical Center, Bronx, NY, United States. ² Internal Medicine, Montefiore Medical Center, Bronx, NY, United States. (Control ID: 1334444)

Learning Objective 1: Recognize duodenal diverticula as a possible etiology for acute pancreatitis
Learning Objective 2: Understand the pathophysiology of duodenal diverticula and how they can play a role in the etiology of acute pancreatitis

Case: A 67 year old woman presented with sharp abdominal pain radiating to the back, nausea, and vomiting for one day. She had a history of cholelithiasis and was status post-laparoscopic cholecystectomy. She was taking celecoxib, pregabalin, and cyclobenzaprine, and denied any recent changes in medication. She was a smoker and had a 50 pack year history, but denied drugs or alcohol. She was afebrile and hemodynamically stable, and had epigastric tenderness and guarding on examination. Significant laboratory values were amylase of 1,829 and lipase of 8,125. An ultrasound revealed mild dilatation of the pancreatic and common bile ducts with no gallstones and a hypechoic structure posterior to the head of the pancreas. Triglycerides were normal. The CT was consistent with pancreatitis and revealed a large juxtapapillary duodenal diverticula (JPDD) compressing the pancreatic head, pancreatic duct and common bile duct. The patient was treated with bowel rest, intravenous hydration, and pain control, and made an uneventful recovery.

Discussion: Acute pancreatitis is extremely common cause of hospital admission, with an incidence of 79.8 cases per 100,000 in the United States, frequently encountered by the internist. The majority of cases are caused by alcohol and cholelithiasis. Rarer causes include hypertriglyceridemia, trauma (including iatrogenic), medications, infections, and autoimmune processes. Cases in which no etiology is found (8-44%) are labeled “idiopathic” pancreatitis.

The patient had an unexpected finding of JPDD on imaging which was an etiological factor for her acute pancreatitis. The incidence of JPDD increases with age – retrospective analyses of ERCPs have revealed a 6% prevalence among patients below 50, 22% among patients between 60 and 69, and 30% among patients above 70. While a congenital factor may be involved, the major etiology is thought to be due to an increase in intraduodenal pressure and a weakening of duodenal smooth muscles. The majority of diverticula are asymptomatic, but they have been associated with serious complications like hemorrhage and perforation, which are surgical emergencies. Compression of the pancreatic duct from distension of the diverticulum is implicated in the etiology of pancreatitis.

A retrospective analysis on 433 patients who underwent ERCP over a 2 year period observed that the prevalence of “idiopathic” pancreatitis was 14% in patients with JPDD and 2% in patients without JPDD. The recommendation of the study was that JPDD be excluded before making a diagnosis of idiopathic pancreatitis, especially in older patients. There are no established guidelines on the management of duodenal diverticula. The main surgical option is diverticulectomy, which is not recommended for asymptomatic diverticula.

In summary, idiopathic pancreatitis is a common diagnosis, and a number of studies support a pathophysiological role for JPDD. While there are no guidelines on the management of diverticula, it is important for the internist to be aware of the increasing prevalence of these anatomical abnormalities and their relationship to pancreatitis in an increasingly aging population.
Mystery Case

Jonathan Katz¹, Jordan Brodsky¹, Dahlia Rizk¹, Ya Ju Chang¹, David Beyda², Jose A. Cortes¹. Medicine, Beth Israel Medical Center, Manhattan Campus of the Albert Einstein College of Medicine, New York, NY, United States. ² Medicine, University Hospital of Brooklyn at Long Island College Hospital, Brooklyn, NY, United States. (Control ID: 1296876)

Case: 72 year old man from Cuba with COPD was admitted for increasing dyspnea