CLINICAL VIGNETTE

Paraesophageal Hernia Presenting as Chronic Cough: A Case Report

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Introduction

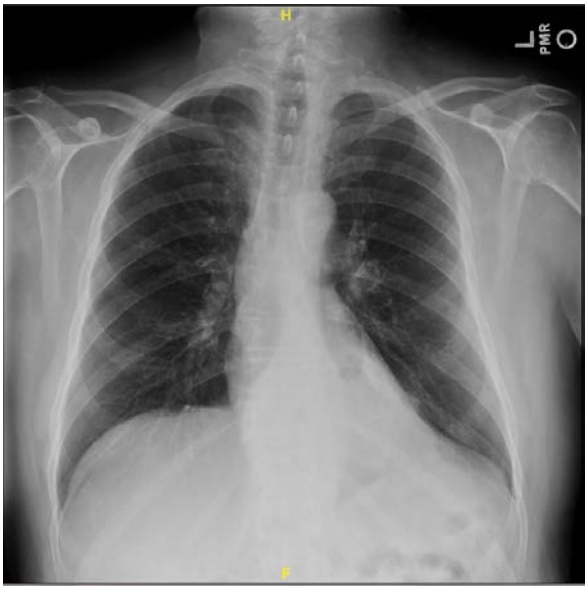
Hiatal hernias are characterized by protrusion of abdominal contents into the thoracic cavity through a widening of the esophageal hiatus of the diaphragm. Hiatal hernias are broadly divided into sliding and paraesophageal hernias, although the most comprehensive classification recognizes four different types of hiatal hernias. Type I (sliding) hernia may arise from congenital malformation, trauma, and iatrogenic factors. Type II, III and IV (paraesophageal) hernias may arise as complication from surgical dissection of the esophageal hiatus in antireflux procedures, esophagomyotomy, or partial gastrectomy. More common complications of hiatal hernia include gastroesophageal reflux disease, gastroesophageal erosion, ulceration and bleeding, laryngopharyngeal reflux disease, gastric volvulus and respiratory complications.[1] Less common complications of large paraesophageal hernia include splenic rupture[2] and cardiac syncope.[3] In this paper, I present a patient with a large type III paraesophageal hernia with pulmonary complications.

Case Report

A 78-year-old male, nonsmoker, with a past medical history of pyloric stenosis repair as a newborn, allergic rhinitis, hypertension and diabetes mellitus presented with chronic productive cough for 8 weeks. He described a productive cough with clear phlegm all day and night with wheezing and shortness of breath. He had no fever or chills, no hemoptysis or night sweats. He had a history of pollen allergies with mild nasal congestion but no loss of smell, taste or facial pain. Trial of glucocorticoid nasal spray, 2nd generation antihistamine and saline nasal rinse provided relief to his nasal and sinus congestion but not his cough. He reported an intentional 7 lb weight loss over a 3-month period through diet and exercise. He denied heartburn, acid taste in the mouth or voice changes. Empiric treatment with a proton pump inhibitor for 4 weeks did not improve his symptoms. Ace-inhibitor was held with no improvement of his cough. Fluoroquinolone antibiotic prescribed in urgent care 1 month before presentation led to a temporary improvement of his cough.

Physical exam was significant for mild wheezing in the right thorax, without crackles or egophony. His abdomen was soft with normal active bowel sounds, mildly distended but non-tender, no hepatosplenomegaly. Laboratory test included normal CBC and CMP, Negative sputum for culture TB and fungus.

Figure 1. 78-year-old male with paraesophageal hernia, a fluid-filled retrocardiac density is present on CXR.



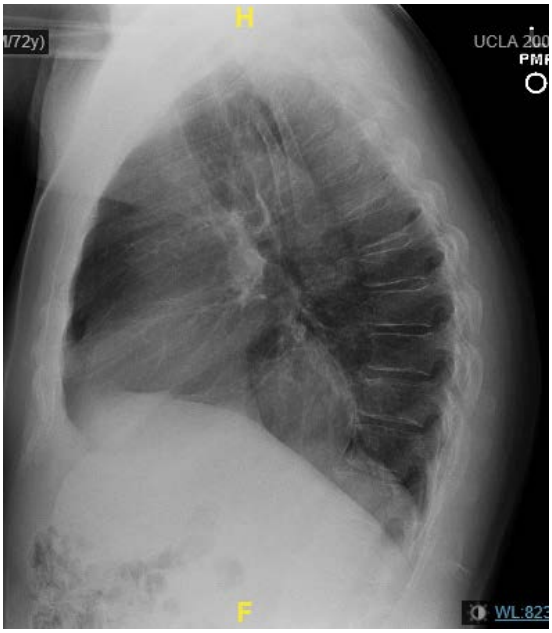
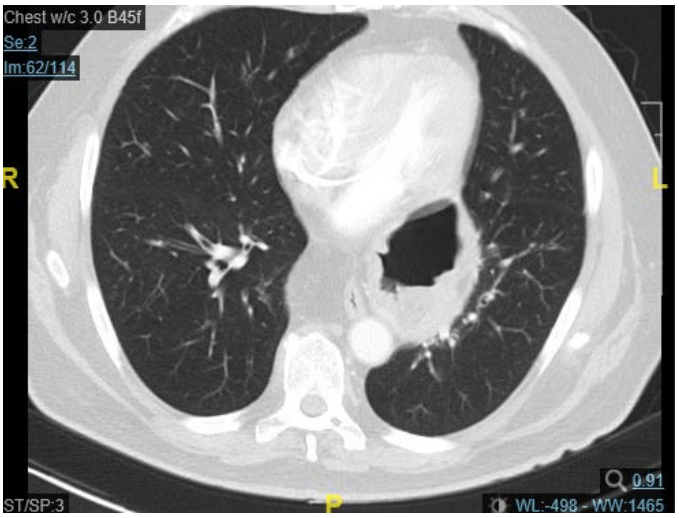


Figure 2. CT chest with contrast: large hiatal hernia containing most of the stomach; diffusely abnormal airways affecting all lobes



CXR: Showed no mass or pneumonia but a fusiform retrocardiac fluid-filled density. CT chest with contrast: Revealed diffusely abnormal airways affecting all lobes of the lung, consistent with chronic inflammatory airway disease. A large hiatal hernia containing most of the stomach. CT sinus: Showed bilateral sinus disease, with thickening of the bony walls, compatible with chronic sinus inflammation with no paranasal sinus air fluid level. Pulmonary function testing: Normal.

Endoscopy with wireless pH capsule placement revealed esophageal spasms and a large hiatal hernia. High Resolution Esophageal Impedance Manometry showed ineffective esophageal motor function. Bravo 48-hour Intraesophageal pH Study: Normal number of reflux events and acid exposure, no correlation between symptoms and reflux. It did not support diagnosis of symptomatic GERD.

Management

The patient underwent laparoscopic hernia repair with Toupet fundoplication and biologic mesh. His cough resolved 3 months after surgery and his lung findings improved radiographically after 6 months. He remains asymptomatic 1 year after surgery.

Discussion

Classification of Hiatal Hernia

Type I: Sliding hernia-This is characterized by the displacement of the gastroesophageal (GE) junction above the diaphragm. The stomach remains in its usual longitudinal alignment and the fundus remains below the GE junction.

Type II-IV: Paraesophageal hernias- These are true hernias with a hernia sac. The GE junction remains in its normal anatomic position but the gastric fundus herniates through the diaphragmatic hiatus adjacent to the esophagus.

Type II: This is a pure paraesophageal hernia, resulting from a localized defect in the phrenoesophageal membrane where the gastric fundus herniates through the defect while the GE junction remains in its normal anatomic position.

Type III: Combination of Type I and Type II. Both the gastroesophageal junction and the fundus herniate through the hiatus. The fundus lies above the gastroesophageal junction.

Type IV: This is associated with a large defect in the phrenoesophageal membrane and characterized by the presence of organs other than the stomach such as the omentum, colon or small bowel within the hernia sac.

Type I accounts for more than 95% of hiatal hernias. Type II is the least common type of paraesophageal hernia. Type III constitutes more than 90% of paraesophageal hernias.

Pathophysiology

A sliding hiatal hernia results from progressive disruption of the GE junction through widening of the diaphragmatic hiatus and circumferential laxity of the phrenoesophageal membrane. This allows the gastric cardia to herniate upward into the thoracic cavity.[4] Most small sliding hiatal hernias are asymptomatic but large sliding hiatal hernias may have symptoms of gastroesophageal reflux disease. Type I sliding hiatal hernia promotes acid reflux by affecting the competence of the GE junction and by prolonging acid clearance.[5]

In contrast, paraesophageal hernias are associated with abnormal laxity of the gastrosplenic and gastrocolic ligaments which allows the greater curvature of the stomach to roll up into the thorax. Because the stomach is fixed at the GE junction, the herniated stomach has a tendency to rotate around its longitudinal axis resulting in an “upside down stomach”. As the hernia enlarges, the entire stomach may herniate and bring other organs such as the colon, omentum or small intestine into the hernia sac located in the thoracic cavity.

Clinical Manifestations

Most small type I sliding hiatal hernias are asymptomatic. However patients with large type I hernias may have symptoms of indigestion, regurgitation and dysphagia.

Many patients with paraesophageal hernias are either asymptomatic or have vague symptoms. GERD symptoms are less common compared to sliding hernia. The most common symptoms are epigastric pain, nausea and intermittent abdominal fullness.

Diagnosis of Hiatal Hernia

Hiatal hernia is not a diagnosis that is typically pursued, but is commonly diagnosed incidentally. Plain chest radiographs may identify soft tissue opacity with or without an air fluid level within the chest. A retrocardiac air fluid level on chest x-ray is pathognomonic for a paraesophageal hiatal hernia.

Contrast studies may help determine the size and reducibility of the hernia and localize the GE junction in relation to the esophageal hiatus. Contrast studies may also help identify presence of a short esophagus. Identification of a short esophagus will allow the surgeon to prepare for an intra-operative lengthening procedure if needed. Computed tomography scan maybe useful to urgently visualize herniated organs within the chest cavity.

Esophagogastroduodenoscopy (EGD) allows visualization of the mucosa of the esophagus, stomach and duodenum to determine the size and type of hernia. Difficulty reaching the duodenum in the presence of a large hiatal hernia is diagnostic of a volvulized paraesophageal hernia.

Esophageal motility study enables proper placement of a pH probe above the lower esophageal sphincter in patients with a sliding hiatal hernia and symptoms of gastroesophageal reflux.

pH testing is important in identifying the presence of increased esophageal acid in patients with sliding hiatal hernias. These patients who may benefit from anti-reflux surgery. The presence of abnormal gastroesophageal reflux by identification of erosive esophagitis or Barrett's esophagus on upper endoscopy or increased esophageal acid exposure on pH monitoring assists the surgeon in determining operative intervention in patients with a sliding hiatal hernia.

Management

Management of patients with symptomatic sliding hiatal hernia consists of management of GERD. This involves lifestyle and dietary modification as well as medications for GERD. Patients with sliding hiatal hernia and proven GERD who fail medical management may be considered for surgical repair. Surgical repair of a type I hiatal hernia in the absence of reflux disease is not indicated.

Paraesophageal hernia: Symptomatic paraesophageal hiatal hernia is an indication for surgery. Emergent repair is indicated in patients with uncontrolled bleeding, obstruction, volvulus, strangulation, perforation and respiratory compromise. Routine elective repair of asymptomatic paraesophageal hernias is not indicated.

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