

Case Report

Esophageal Rupture in a Young Man after Alcohol Ingestion: Boerhaave Syndrome. Case Report and Review of Literature.

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Abstract

Barogenic rupture of the esophagus, the so-called “Boerhaave Syndrome,” is the most serious and rapidly lethal perforation of the gastro-intestinal tract. The pathology involved is a complete, transmural laceration of the oesophagus. The diagnosis it’s very difficult because very few patients present with the classic triad of retching, sudden epigastric pain and shock, at the time of recovery. Most series in the literature report essentially a 100% mortality within 7 days without surgery and only a 70% overall survival with surgical intervention. The outcome essentially depends on prompt diagnosis and treatment. We present the case report of a prompt diagnosis spontaneous esophagus rupture during vomiting after enough eating in 43 years old.

Keywords: Boerhaave’s syndrome; Spontaneous esophageal rupture; Thal technique

Introduction

Effort rupture of the esophagus, or Boerhaave syndrome, is a spontaneous perforation of the esophagus that results from a sudden increase in intraesophageal pressure combined with negative intrathoracic pressure for severe straining or vomiting. It compares in a normal esophagus or in pathological diseases such as Barrett’s esophagus and less frequently with childbirth, seizure, prolonged coughing or laughing or weightlifting. It is the most serious and rapidly lethal perforation of the gastro-intestinal tract. The pathology involved is a complete, transmural laceration of the esophagus. The diagnosis it’s very difficult because very few patients present with the classic triad of retching, sudden epigastric pain and shock, at the time of recovery. Esophageal perforations are rare, with an incidence of 3.1 per 1,000,000 per year. We present the case of a 43 years old Caucasian male patient who has had a spontaneous esophagus rupture during vomiting after an abundant meal.

History

Boerhaave’s syndrome was first described by a Dutch physician, Hermann Boerhaave in 1724. His patient was a 50-year-old Grand Admiral of The Netherlands, Baron Jan van Wassenaer, who died in 1723 after 18 hours of self-induced vomiting which

resulted in esophageal rupture. At autopsy, Boerhaave recognised the smell of duck flesh and found olive oil and roast duck flesh in the left pleural cavity plus a transverse tear (not the usual linear rent) in the distal esophagus [1].

Case Report

A 43-year-old Caucasian man, was seen in the Emergency Room at 9:00 pm after some episodes of vomiting approximately one hour prior to admission. He has had an abundant meal with alcohol abuse. On admission he was oriented with a Glasgow Coma Scale (GCS) 15, his temperature was 36 C, pulse rate 100/min, respiratory rate 15/min, and blood pressure 150/90 mm Hg. During the visit he appeared acutely ill and he reported strong pain in epigastrium posterior irradiated. Physical exam revealed clear breath sounds bilaterally, marked epigastric tenderness, and rigidity with rebound. Peristalsis was absent and np crepitus was palpable in the neck. No evidence of an acute myocardial infarction was present on the initial EKG. Initial clinical impressions included a differential diagnosis of a perforated peptic ulcer, pancreatitis, alcoholic gastritis or a pneumothorax. Plain films of the abdomen were normal and no pneumoperitoneum was present. A gastrographin swallow Chest-TC scan was then performed which demonstrated marked extravasation of contrast from the distal esophagus to the left posterolateral mediastinum and diffuse atelectasia (Figure 1). The patient underwent an emergency open laparotomy six hours later. He was subjected to emergency intervention with a classic

Mercedes incision laparotomy. Was discovered a perforation of the lateral distal esophagus, approximately 6 cm in length and 1.5 cm above the hiatus and 400 cc of serosanguinous fluid was present in the left pleural cavity with a marked pleural and mediastinal inflammatory reaction. The mediastinum was intact. We performed a repair primarily with a two-layer silk closure and gastric fundus patch with Thal's Technique. The mediastinum and pleural spaces were irrigated with saline and drained trans-hiatus. A nasogastric tube was passed into the stomach intra-operatively in aspiration form and the mediastinum was drained with a trans-hiatal tube. A left chest tube was placed. He was placed on therapeutic doses of antibiotics (penicillin, cephalothin and gentamicin). A following Ct Scan demonstrated the anatomic status (Figure 2). He was discharged on the seventy-hospital day on a normal diet and follow-up upper GI x-rays were essentially normal. (Figure 3) One month after follow-up and chest radiography demonstrated a complete pathological response. (Figure 4)

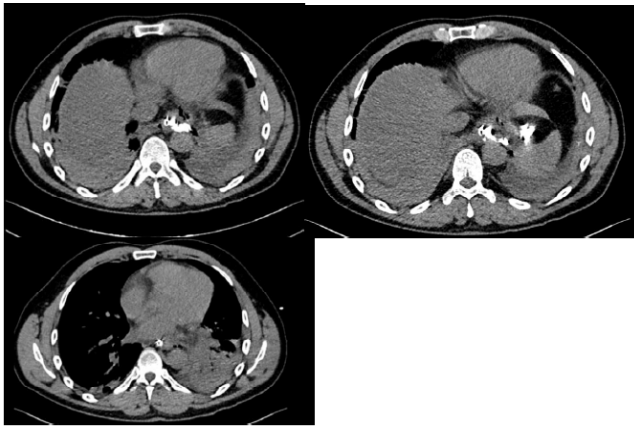


Figure 1: Chest/Abdomen CT Scan: Extraluminal oral contrast with left pleural effusion, retrocardiac air.

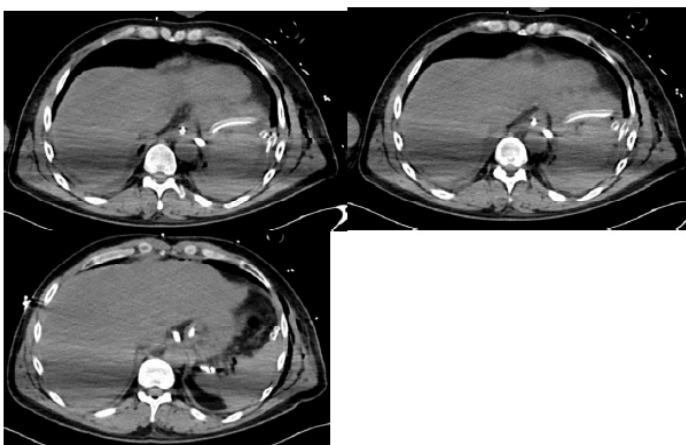


Figure 2: CT Scan after Thal's technique and left chest tube and trans-hiatal mediastinal tube.

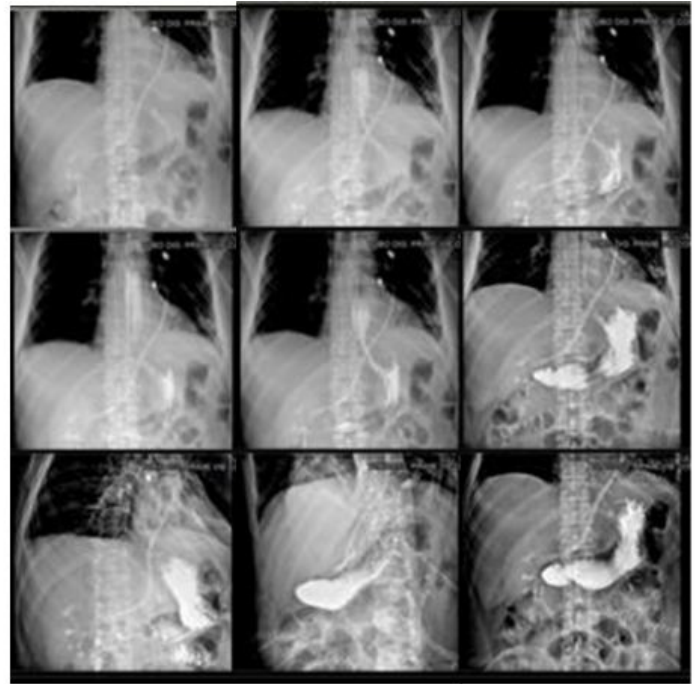


Figure 3: Oral contrast Radiography after Thal technique: no extra esophageal contrast.



Figure 4: Chest Radiography at first follow-up.

Pathogenesis

The cause of the high morbidity and mortality is serious cardiorespiratory insufficiency due to fulminating mediastinitis secondary to the accumulation within the mediastinal and pleural spaces of corrosive gastric juices, enzymes, food and bacteria. Severe sepsis and Septic Shock follow associated to major fluid

losses, and suppuration of the mediastinum and pleural cavities. The mediastinal pleura ruptures are digested at a later stage by gastric contents which are then drawn into the pleural space by the negative intrathoracic pressure. This occurs on the left side in 75 - 90% of cases, but can be bilateral in 5 - 10% of patients or rarely, as in the present case, on the right. It has been postulated that the esophageal rupture is caused by a sudden rise in the intraluminal esophageal pressure produced by actual or suppressed vomiting with full stomach contents being ejected against a closed cricopharyngeus muscle. There are several reasons for the predilection of the perforation in the distal esophagus for the left side, including thinning of the musculature of this area, segmental defects in the circular layer, weakening of the wall by entrance of vessels and nerves, anterior angulation of the esophagus at the left diaphragmatic crus, and lack of adjacent supporting structures [2-4].

Clinical Presentation

It consists of vomiting, lower thoracic pain, and subcutaneous emphysema. A provider should suspect Boerhaave's syndrome when a patient presents with retrosternal chest pain with or without subcutaneous emphysema when associated with heavy alcohol intake and severe or repeated vomiting. Up to one-third of patients do not present with these symptoms. The actual clinical presentation of Boerhaave syndrome will depend on the level of the perforation, the degree of leakage, and the time since onset of injury. Because of the lack of specificity of signs and symptoms, the diagnosis of esophageal perforation is often delayed and/or missed [4]. The classic clinical presentation as usually described in the literature is of overindulgence in food or drink with vomiting followed by severe chest pain, dyspnea, mediastinal or subcutaneous emphysema and cardiovascular collapse; however, some reviews suggest that the presence of the entire complex of symptoms is rare. The most striking feature is the excessive pain which is poorly relieved by narcotics. Usually it is a pleuritic left-sided chest pain which may radiate to the substernal area, epigastrium or back. If pneumo-mediastinum is present, mediastinal cracking coincident with each. Physical exam findings may include abnormal vitals (tachycardia, tachypnea, fever), decreased breath sounds on the perforated side, mediastinal emphysema, and Hamman's sign (mediastinal "crackling" accompanying every heart beat) in left lateral decubitus position [5].

Diagnostic Evaluations

Unfortunately, laboratory tests provide little help in the diagnosis; however, they can exclude more common conditions in the differential including myocardial infarction and pancreatitis. Chest/abdomen Radiography are the most valuable modality that give the most information. The most common findings are

pleural effusions and pneumothorax with incidences of 91% and 80%, respectively [5]. Pneumothorax is usually associated with pleural effusion and is unilateral but can be bilateral in a few cases. Mediastinal air should be carefully looked for, as it is easily missed if it is retrocardiac. Widening of the mediastinum is occasionally seen and the so-called 'V-sign of Naclerio' is another subtle X-ray finding which is easily overlooked [6-8]. In patients in whom esophageal perforation is suspected clinically Computed Tomography (CT) associated with trans-oral Gastrographin for a positive oral contrast is the best diagnostic evaluation by localizing collections of fluid for surgical drainage. Boerhaave's syndrome- CT findings are as following: air in the soft tissues of mediastinum surrounding the esophagus, abscess cavities adjacent to the oesophagus in either the pleural space or the mediastinum, demonstration of an actual communication between air-filled esophagus and an adjacent mediastinal or paramediastinal air fluid collection. During the CT scan examination, we found positive oral contrast in left mediastinum and retrocardiac air [9-11].

Treatment

Treatment is typically tailored to the patient's presentation. Decision making depends on: size and location of perforation, presence of underlying esophageal disease, interval between perforation and diagnosis, condition of esophagus extent of soilage or injury to adjacent organs and tissues, age and general condition of the patient. Conservative measurements are usually reserved for small or contained ruptures [12-14]. Endoscopic placement of stents has been used to prevent fistula formations or seal esophageal leaks in both patients with delayed diagnoses and those with the early diagnosis without widespread contamination. Principles of operative management are: wide debridement of esophageal and mediastinal tissue, proper and thorough cleansing of pleural cavity, correction or elimination of any distal obstruction, two-layer closure of perforation, if necessary reinforce with other tissues, provision of broad-spectrum antibiotic cover and elective ventilation, if required [15]. Alternately, positioning of T-Tube in esophagus perforation associated with thoracic tube and nasogastric tube or complete esophagus defunctioning esophagostomy, and feeding gastrostomy. In this case, for the prompt diagnosis, we decide to perform a primary double layer closure with gastric patch as Thal's techniques [16-18].

Conclusions

Spontaneous esophageal perforation is a life-threatening condition and if diagnosed early, life may be saved with urgent and aggressive management: a careful history with a high index of suspicion and careful study of chest film will give important clues to diagnosis. Timing of diagnosis is the most important condition. A delay of 12 hours or more between symptoms and operation is

associated with a 36-64% mortality. More hours more mortality. The common reason for delayed diagnosis is that it always mimics other more common acute cardiothoracic or upper gastrointestinal conditions. Chest/Abdomen CT scan with oral contrast is a useful diagnostic modality [19]. The surgical modality depends on the time of intervention. Delayed time: T-Tube or esophageal exclusion. Prompt diagnosis: primary and reinforced closure [20].

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