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Case Report Open Access

Esophageal Stenosis as a Cause of Spontaneous Esophageal Perforation (Boerhaave Syndrome): A Case Report and Explanation of Possible Mechanism

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Abstract

Introduction: Boerhaave syndrome (BS) is a spontaneous perforation of the esophagus which depends on increased intra-esophageal pressure, while the upper sphincter is closed during excessive vomiting.

Case: A 75-year-old man was referred to hospital with sudden chest pain after vomiting. A computed chest tomography demonstrated mediastinal emphysema, thickening of the wall at the esophago-gastric junction level, and left pleural effusion. An upper gastrointestinal endoscopy showed an esophageal stenosis at mid thoracic portion and a perforation detected just 1 cm above of the lower gastro-esophageal sphincter. The patient underwent left posterolateral thoracotomy in the 12th hour of event. Stenotic segment is dilated and the mucosal perforation was repaired.

Conclusion: BS is a serious disease with high morbidity and mortality rates. While BS usually occurs in a normal esophagus; in our case, BS was due to esophageal benign stenosis instead of upper sphincter esophageal sphincter closure. Benign stenosis may facilitate perforation as seen in our patient due to increased intraluminal pressure following vomiting.

Keywords: Boerhaave's syndrome; Esophageal stenosis; Spontaneous esophageal perforation

Introduction

Boerhaave syndrome is a spontaneous perforation of the esophagus that is characterized by triad of vomiting, chest pain and subcutaneous emphysema. Perforation depends on increased intra-esophageal pressure, while the upper sphincter is closed during excessive vomiting [1,2]. In the case which is presented here, a spontaneous esophageal perforation occurred due to increased intra-esophageal pressure caused an esophageal stricture, instead of upper esophageal sphincter.

Case Report

A 75-year-old man was referred to hospital with sudden chest and back pain after vomiting. He was cured for a cerebral tumor 15 years ago and had no neurologic deficit. He had alcohol and fizzy drinks abuse. From his past history, it was understood that he had suffered of progressive dysphagia for 7 months.

A physical examination revealed epigastric tenderness, minimal left cervical subcutaneous emphysema and decreased respiratory sounds in the left lower zone. His pulse was 110 beats/min, blood pressure was 110/70 mmHg and body temperature was 38°C.

In laboratory findings; sedimentation rate measured 96 mm/h, C-reactive protein (Crp) level was 41 mg/dL (0-6), and white blood cell (WBC) count were 8100/dL. Postero-anterior chest X-Ray revealed a closed left cardio-phrenic sinus (Figure 1). A computed chest tomography (CT) scan showed diffusing emphysema to the mediastinum adjacent to the esophagus, thickening of the wall at the esophago-gastric junction level, and significant left side pleural effusion (Figure 2).

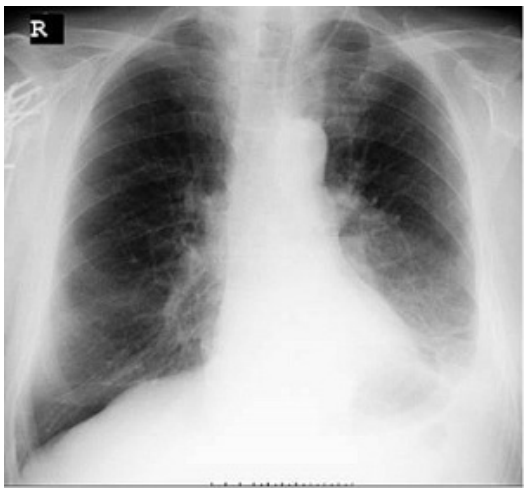

Figure 1: Closed left cardio-phrenic sinus in chest X-Ray.

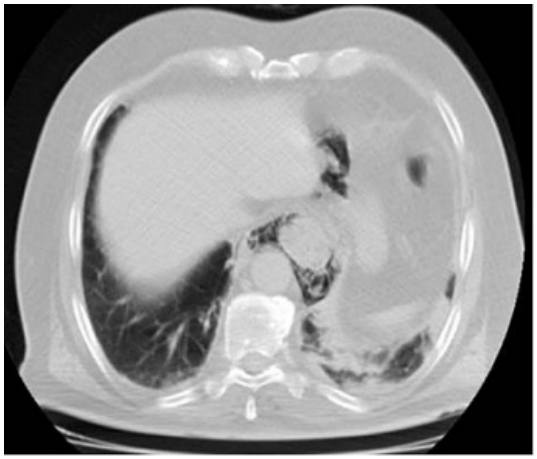

Figure 2: Mediastinal emphysema in chest CT.

An upper gastrointestinal endoscopy showed an esophageal stenosis didn't allowed pass at 35 cm mid thoracic portion (Figure 3). After a balloon dilatation, endoscope passed through the distal esophagus. Approximately 15 mm oblique perforation was detected on 1 cm above of the lower gastro-esophageal sphincter at the posterolateral wall (Figure 4). Fundus, cardia, and gastric mucosa were normal.

The patient immediately underwent left posterolateral thoracotomy from the seventh intercostal space in the 12th hour of event. At the exploration, approximately 200 cc of serous fluid was observed in the left pleural space. When the distal esophagus was explored, an oblique perforation was observed 1 cm above the cardia. The mucosa was dissected along the proximal side, and a significant stenosis was detected almost 5 cm above the perforation site. Frozen section examination of mucosal and muscular layer of the distal esophagus revealed benign pathology. A vertical incision was done to muscularis layer of stenotic segment and sutured transversely for dilatation. The mucosal perforation was repaired using 00 absorbable polyglactin sutures. The muscular layer was closed by using 00 silk sutures. Furthermore, the perforation site was supported by a pleural flap.

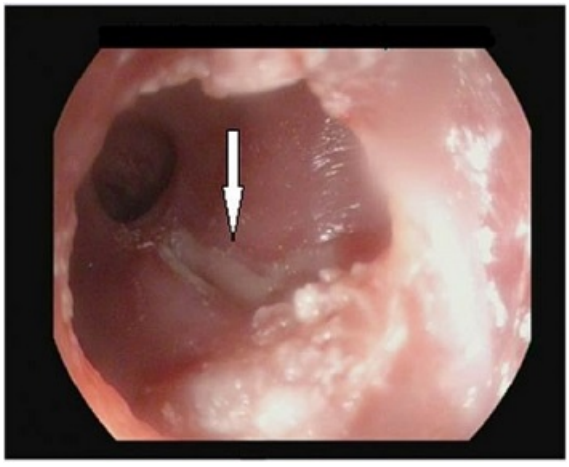

Figure 3: Esophageal stenosis.

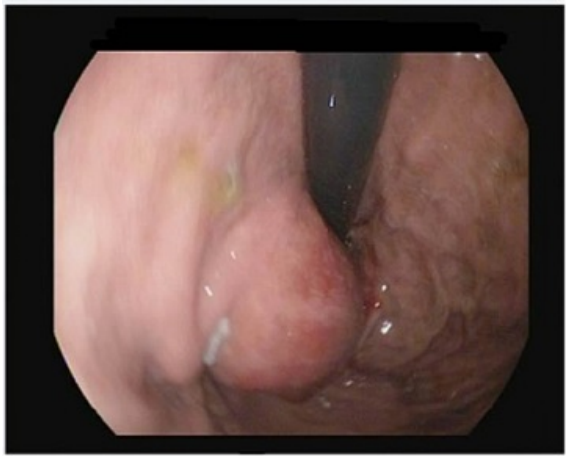

Figure 4: Esophageal rupture.

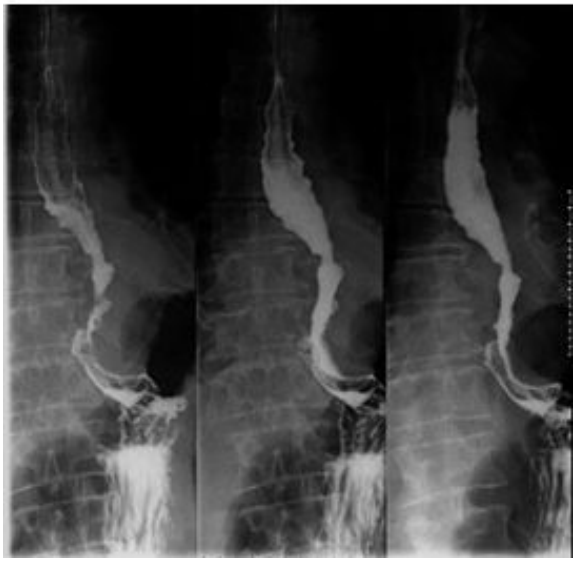

Figure 5: Barium esophagogram showed no leakage on 8th day of operation.

After one night stay of Intensive Care Unit, the patient was followed in the ward with nasogastric decompression, intravenous fluids, total parenteral nutrition and broad spectrum antibiotics. In his follow up barium esophagogram demonstrated no leakage at 8th days of surgery (Figure 5) and oral feeding was started. The patient was discharged on the 16th day after admission and has been followed-up without dysphagia for 10 months.

Discussion

Boerhaave syndrome is a spontaneous perforation of the esophagus and characterized by triad of vomiting, chest pain, and subcutaneous emphysema [1]. The condition was first defined by Herman Boerhaave in 1724 during autopsy of an admiral who died after excessive eating.

Meyer reported death due to the disease in 1852, and successful treatment was reported after 1947 [2,3].

80% BS cases is seen in males especially between age 40 and 60 years. A carefully medical history and physical examination as well as radiography and esophagoscopy help to diagnose of BS [4,5]. BS is often misdiagnosed as a myocardial infarction, aortic dissection, peptic ulcer perforation, pancreatitis, Mallory-Weiss syndrome (excessive bleeding due to mucosal rupture of the gastric orifice) and spontaneous pneumothorax [1,3,4].

Perforation is mostly caused by sudden increased pressure inside the esophagus while the upper sphincter is closed during excessive vomiting (Figure 6a and 6b). This is also related less frequently with sneezing, retching, excessive coughing, severe straining for toilet, laughing, weightlifting, prolonged seizures, asthma attack, and the Heimlich maneuver during labor [4-6].

The perforation is usually seen at the weakest portion of esophagus which is in the left posterolateral part of the distal intrathoracic esophagus. Cervical or intra-abdominal esophagus is rarely affected. It results in contamination of the mediastinal cavity with gastric contents. This leads to chemical mediastinitis with mediastinal emphysema and inflammation, with a subsequent aerobic and anaerobic bacterial infection. Rupture of the overlying pleura by mediastinal inflammation contaminates the pleural cavity, and results pleural effusion. Acute mediastinitis, septicemia and organ failure is inevitable in cases without immediate and appropriate treatment [3,6].

The treatment of BS is primarily surgical repair; however, in some special cases there are some endoscopic or conservative treatment options. Surgical repair with pleural, pericardial or intercostal muscle flap support is suggested for treatment within the first 24 hours [1,7]. Adequate mediastinal debridement can be done by open thoracotomy or video-assisted thoracoscopy (VATS). In the presence of a diseased esophagus, resection and primary anastomosis may be added to the procedure [7-9].

Conservative management (cessation of oral intake, total parenteral nutrition, intravenous broad spectrum antibiotics, nasogastric decompression, proton pump inhibitors and tube thoracostomy) may be considered in patients without mediastinal or pleural contamination on imaging studies and without systemic symptoms of infection [10,11]. Conservative treatment is also suggested for cases having intramural esophageal dissection [12].

Endoscopic repair with a self-expandable stent can also be a treatment option in patients with comorbidities that has high risk for surgery [13].

Mortality ranges varies from 28%-85% between series [2-4,8]. Ökten et al. reported a mortality rate of 45.5% with conservative treatment and 20% with surgical treatment [2]. Schmidt et al. reported hospital mortalities of 11.1% within 6 hours, 13.3% within 6-24 hours, and 22.2% for repairs performed after 24 hours in their 62 case series [3]. Pate et al. reported in their of 34 patients series, even though there was no significant difference in mortality between repair within the first 24 hours and later; but complication rates were higher in cases of delayed repair. Jougon et al. also reported that early or late operations did not affect mortality [14].

Possible mechanism of perforation in our case: BS usually occurs in patients with a normal esophagus. However, some of patients with Boerhaave syndrome have underlying esophageal problems such as medication-induced esophagitis, Barrett's, eosinophilic or infectious ulcers [1,6]. Our patient has got a benign stricture probably related with esophagitis.

In our case BS was due of esophageal stricture instead of upper sphincter closure (Figure 6c). BS with esophageal stenosis has not been previously reported in the literature. Benign stenosis may have facilitated perforation as in our patient by causing increased intraluminal pressure following vomiting.

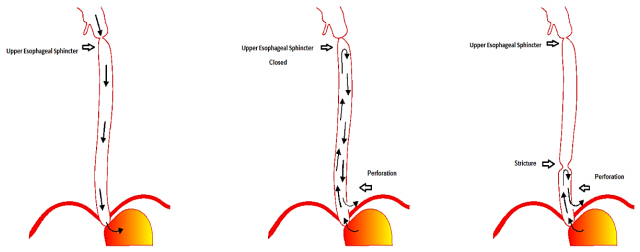

Figure 6a: esophageal anatomy, **6b)**BS mechanism, **6c)**tricture causes BS.

Conclusion

BS is a serious disease with high morbidity and mortality rates. The appropriate treatment depends on the extent, location and containment of the perforation and the comorbidities and patients' delay in presentation. Expeditious repair and suture line support in living tissue is the appropriate treatment method. Perforation occurs due the increased pressure in esophagus while the upper sphincter is closed during excessive vomiting. But unexpectedly in our case, BS was due of esophageal benign stenosis instead of upper sphincter closure.

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