A Case of Boerhaave's Syndrome

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Abstract

Transmural perforation of the esophagus following an effortful vomiting is a rare but fatal condition. This situation, known as Boerhaave's syndrome, is caused by spontaneously occurring perforations. The esophagus is not resistant to reflexes such as nausea and vomiting because it lacks serosal layers. Boerhaave syndrome, in which substantial amounts of spontaneous perforations occur, is generally seen after emesis. Its rarity and non-specific nature of symptoms make the diagnosis difficult. The immediate recognition of this potentially lethal condition is essential to ensure appropriate treatment. We present the treatment of Boerhaave syndrome in a 78-year-old man who was admitted to the emergency department with a vomiting complaint. *(JAEM 2015; 14: 88-90)*

Keywords: Emesis, esophagus, Boerhaave's

Introduction

Spontaneous esophageal rupture, which was first described by Boerhaave in 1724, is a rarely seen clinical condition following a forceful emesis. It is a life-threatening syndrome; therefore, it requires early diagnosis and management. Boerhaave's syndrome is different from Mallory-Weiss syndrome, which occurs following any forceful emesis and lacks transmural esophageal tears. It is the result of vertical and transmural ruptures at the cardioesophageal junction after a sudden increase in intraluminal pressure in the distal esophagus following forceful retching and vomiting. It is taken as spontaneous ruptures to be differentiated from iatrogenic perforations, which constitute most esophageal ruptures. Delays and difficulties may be seen in making a diagnosis because its non-specific symptoms can easily mimic other diseases. Vomiting and retching are prominent in the etiology. The classical presentation consists of subcutaneous emphysema, chest, and back pain after emesis. Its delayed diagnosis is usual because its incorrect diagnostic rate is higher (1). Boerhaave's syndrome often presents a diagnostic challenge. We present a patient who was admitted to the emergency department with chest pain and dyspnea and was lately diagnosed of Boerhaave's syndrome.

Case Presentation

A 78-year-old man, without any known disease, was admitted to the emergency department with chest pain and shortness of breath occurring after vomiting and nausea after drinking too much mineral water and eating a heavy meal. His medical findings on admission were blood pressure 150/80 mmHg, heart rate 114/min, respiratory rate 24/min, temperature 38°C, and oxygen saturation 96%. Sinus rhythm was seen on his electrocardiogram. He was conscious, cooperative, and oriented, and his general condition seemed good. Nothing abnormal was detected on examination. His cardiovascular system was found normal except his heart rate. Respiratory sounds were decreased in the left side, and some rales existed at the basal areas. Some tenderness was found on abdominal examination. No significant finding was found on his chest X-ray, and abdominal ultrasonography did not reveal any pathology. Minimal pleural effusion was present in his radiological study (Figure 1). His blood test results were within normal limits. He was discharged with symptomatic treatment. He was admitted to the emergency department again in the morning of the next day because of his enduring complaints. Subcutaneous emphysema was noted on physical examination. Computed tomography of the thorax was ordered. The tomography scans revealed normal tracheal and bronchial structures, prominent consolidated areas at the bases at the left side, hiatal hernia, and the break-down of esophageal integrity superior to the esophagogastric junction (Figure 2). A perforation area of 1 cm distal to the "Z" line in the esophagus was seen on upper gastrointestinal endoscopy. The patient underwent immediate thoracotomy. The defect was primarily repaired after the debridement of infected tissues at the proximity. He could not be extubated postoperatively. Renal failure developed after one week, and the patient was in sepsis on day 10. He died on day 14.

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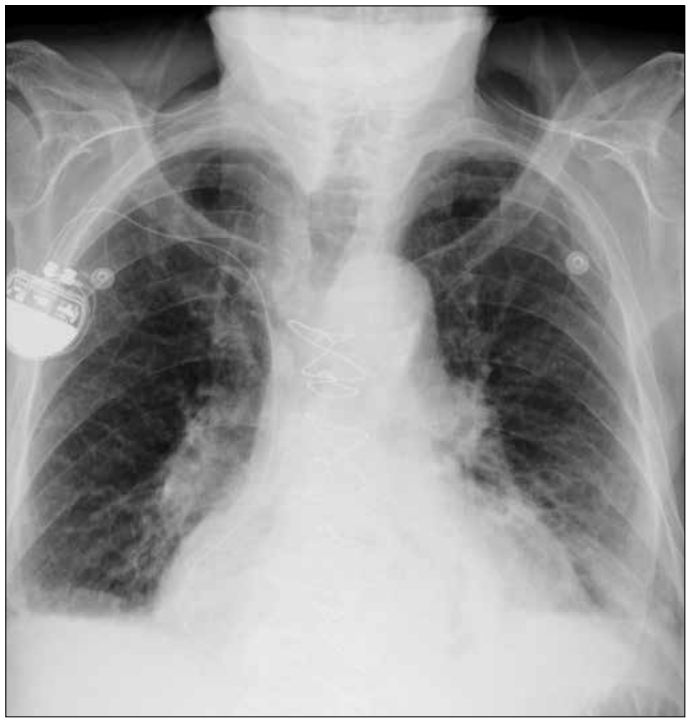

Figure 1. Chest X-ray of the patient on the admission day

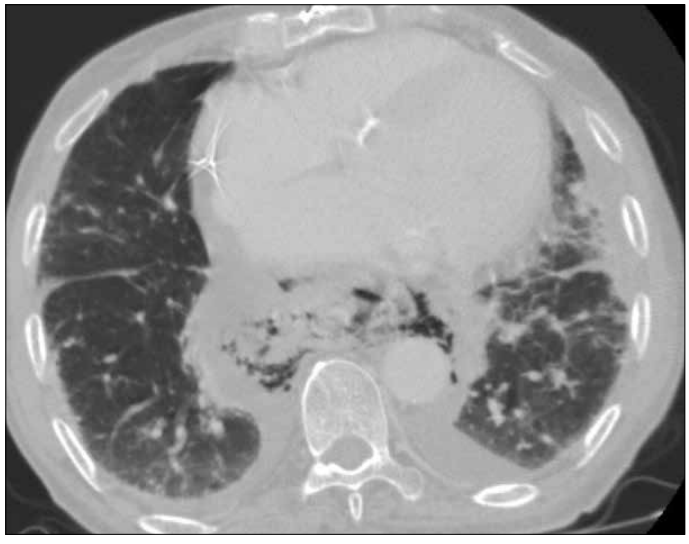

Figure 2. Thorax computed tomography scan of the patient the day after

Discussion

A rupture in the mucosa and submucosa of the esophagus is referred to as Mallory-Weiss syndrome; the rupture is ascribed as Boerhaave's syndrome if the tear occurs in all the esophageal layers. The syndrome was first described by the Dutch physician Boerhaave. It is much more common in men than in women; the rate varies in different communities. It is most commonly encountered between 50 and 70 years of age. Conditions such as vomiting and retching, in which intra-abdominal and intraluminal pressures increase, are common in the etiology. The syndrome can rarely result in straining, delivery, forceful cough, blunt trauma, and epileptic attacks. The syndrome was seen after vomiting and retching following a heavy meal in the presented case.

The esophagus cannot tolerate decreased pressures and getting torn in comparison with other parts of the digestive system because it lacks serosal layers. The lower esophageal diameter increases and cricopharyngeus muscle cannot release as a result of neuromuscular incoordination during emesis. At this moment, the increase in intrathoracic pressure suddenly changes the luminal diameter, and this causes ruptures. Esophageal perforations are most commonly seen at its weakest part, the left posterolateral distal esophagus (1, 2).

The classical findings of Boerhaave's syndrome, known as Mackler triad, include emesis or retching, chest pain, and subcutaneous emphysema. All of them may not be seen in every patient (3, 4). The findings of Mackler triad except subcutaneous emphysema were found in the presented patient on the first examination. Dyspnea, hematemesis, tachycardia, tachypnea, and fever were also found. This syndrome is kept in mind in patients with dyspnea or when pneumomediastinum or left side pleural effusion exists. Findings may be atypical in some patients, and the diagnosis can be overlooked. Diagnosis is rarely made within 24 h of the probable event. In some cases, the diagnosis is made by chance with the aid of tomography. In the presented case, the patient was diagnosed after 24 h on the second admission because the symptoms on the first admission were obscure. Surgical treatment, therefore, was late.

Diseases to be considered in the differential diagnosis cause to omit the diagnosis. These include peptic ulcer perforation, acute myocardial infarction, aortic aneurysm, pulmonary thromboemboli, and pancreatitis. The important diagnostic approach is suspicion., chest X-ray, computed tomography, endoscopy, and esophagography are the diagnostic tools for Boerhaave's syndrome (4-6). Atelectasis, pleural effusion, infiltration, hydropneumothrax, and pneumomediastinum can be found even though some chest X-rays are normal. Periesophageal air collections, pneumomediastinum, subcutaneous emphysema, pleural effusion, infiltration, and atelectasis are easily seen. Periesophageal air collections are shown to extend to the periaortic and intra-abdominal areas in some cases. Initial chest X-ray and later thorax computed tomography can be used to carefully evaluate Boerhaave's syndrome in suspected cases.

Conclusion

Boerhaave's syndrome can be treated medically, endoscopically, or surgically. Its management is controversial because indications vary according to the functional condition of the esophagus. Endoscopic therapy can be conducted up to 48 h of the admission provided that there is no sepsis. Surgical management is inevitable if sepsis is present. Nil by mouth, nasogastric tube placement, and intravenous antibiotherapy are used as the medical treatment (7). First, mediastinitis, empyema, and peritonitis and then sepsis and shock develop with the dissemination of aerobic and anaerobic infections with necrotizing super infection regarding the locations of perforation. Complications due to infections have a profound effect on high morbidity and mortality rates. Increases in complications and mortality are associated with the time to start treatment. The mortality rate is 30% in patients who are immediately urgently treated, and the rate is 100% in patients with the aforementioned complications (2, 8). Therefore, early diagnosis and rapid management are essential. The morbidity and mortality rates begin to increase after 24 h.

The prognosis of Boerhaave's syndrome depends on early diagnosis and prompt treatment. The duration between perforation and surgical intervention should not prejudice the team against primary repair. This syndrome should be suspected in those who have acute chest pain following sudden vomiting and nausea.

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