Case Report

A successful nonsurgical management of Boerhaave's syndrome

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Abstract

Boerhaave's syndrome consists of barogenic rupture of the esophagus, in a previously healthy esophagus. This is a rare, serious, and rapidly lethal perforation of the gastrointestinal tract. The outcome essentially depends on early diagnosis and prompt intervention. Due to the scarcity of case reports, no clear consensus exists regarding the best therapeutic approach. Surgical intervention has been mainstay of management, but with advances in therapeutic endoscopy, there has been an increasing interest in nonsurgical options. Here, we report a case of Boerhaave's syndrome in a 40-year-old male patient, diagnosed early and successfully treated nonsurgically with temporary placement of fully covered esophageal stent.

Keywords: Boerhaave's syndrome, esophageal perforation, esophageal stenting

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INTRODUCTION

Boerhaave's syndrome is characterized by transmural tear of the esophagus induced by a sudden increase in the intraluminal pressure. The rupture usually occurs in the distal third of the esophagus. Boerhaave's syndrome is one of the most lethal diseases of the gastrointestinal (GI) tract, with a mortality rate up to 40%.[1] The classic triad or Mackler's triad seen in Boerhaave's syndrome are vomiting, abdominal or chest pain, and subcutaneous emphysema. However, classic triad seen in 50% of cases and masquerades many clinical conditions such as acute myocardial infarction, tension pneumothorax, and pneumomediastinum.[2] Diagnosis is challenging and requires a high index of suspicion. Delay in diagnosis and treatment may cause potentially lethal complications such as mediastinitis, pleural empyema, septic shock, and even multiorgan failure.[3] Until recently, surgical intervention has been the mainstay of management plans, but nonsurgical options such as endoscopic esophageal stenting have a promising role in nonsurgical management.

CASE REPORT

A 40-year-old, male was brought to emergency department with complaints of pain in the abdomen and chest with swelling over the chest, neck extending into the face up to eyes on the right side, and shortness of breath. The patient gives a history of alcohol binge and heavy meal the night before. After which he had severe abdominal pain followed by two episodes of forceful vomiting and developed swelling over the chest, neck extending into the

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face up to eyes on the right side. Initial examination of the patient showed crepitus over the right chest wall, neck, and face extending up to the right eye, in favor of subcutaneous emphysema. The patient had tachypnea and tachycardia with reduced bilateral air entry, raising the suspicion of esophageal tear and airway injury.

The patient was subjected to contrast-enhanced computed tomography of the abdomen and chest with oral and intravenous (IV) contrast, as a diagnostic evaluation. He was found to have right hydropneumothorax, pneumomediastinum, and left pleural effusion and confirmed tear in the lower esophageal wall with contrast leak into periesophageal tissues. These findings confirmed the diagnosis of Boerhaave's syndrome [Figure 1a and b].

In view of tachypnea, right hydropneumothorax, and left pleural effusion, immediately the patient was intubated and bilateral intercostal drainage (ICD) tube was placed as shown in chest X-ray [Figure 2a]. Pleural fluid was sent for the analysis, which showed, amylase >1000 U/L, lactate dehydrogenase >1000 U/L, adenosine deaminase – 41.90 U/L, sugar – 123 mg/dl, and protein – 3.9 g/dl, suggestive of exudative, amylase rich effusion secondary to esophageal perforation.

Considering poor general condition of the patient and esophageal tear with free mediastinal communication, endoscopic esophageal stenting was performed within 24 h of admission. Upper GI endoscopy (Olympus GIF-HQ 190 Gastroscope) was done using minimal carbon-di-oxide insufflation instead of air. A linear tear about 2 cm was noted in the distal esophagus, just above the Z line. The margins of the tear were edematous and unhealthy looking and there was edema of the surrounding mucosa. There was no hiatus hernia or any other upper GI pathology. The guidewire was placed into the stomach and external radiopaque markers were placed to mark the desired position for stent placement. Endoscope was withdrawn and a fully covered self expanding metallic stent (Niti-S fully covered stent, length 10 cm, diameter 20 mm, Manufacturer-Taewoong medical, South Korea) was placed under fluoroscopic guidance so that the area of perforation was completely covered. Prior to deployment of the stent, two long threads (Ordinary twine thread) were tied to the proximal end of the stent. After deployment of the stent, these threads were brought out through the nostrils and tied, so that the stent was held in place and distal migration was prevented. This was necessary as the stent was fully covered and distal migration is a commonly reported complication. Mechanical ventilation was continued, the patient was kept nil per orally after the procedure and parenteral nutrition was started.

ICD output decreased and subcutaneous emphysema gradually resolved by end of 1 week. Repeat computed tomography (CT) chest with IV and oral contrast was performed on day 11, which showed moderate pleural effusion with underlying collapse consolidation. Esophageal stent was *in situ* and there was no extravasation of orally administered contrast, demonstrating adequate sealing of perforation by the stent [Figure 2b]. The patient gradually weaned off from ventilator support and was extubated on day 13. Gradually, ICD and right-sided

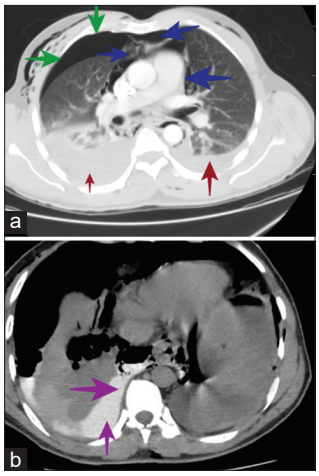

Figure 1: Axial lung window computed tomography chest images. (a) Axial lung window computed tomography chest image showing right pneumothorax (green arrow), extensive pneumomediastinum (blue arrow), and bilateral pleural effusions (red). (b) Axial computed tomography chest with oral contrast showing leakage of contrast to right pleural space (pink arrow) 0 suggestive of the esophageal tear

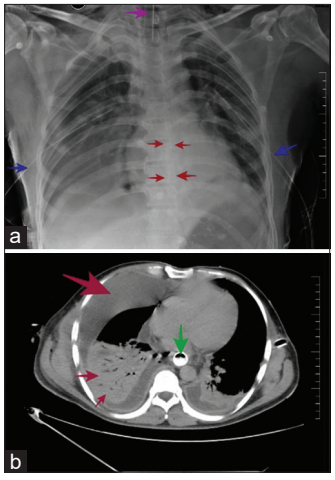

Figure 2: (a) Chest X-ray showing the endotracheal tube (pink arrow), bilateral intercostal drainage (blue arrow), and esophageal stent (red arrow). (b) Axial computed tomography chest showing esophageal stent with oral contrast (green arrow) and no contrast leak into pleural space with right lung consolidation and pleural effusion (red arrow)

chest drains were removed on day 18. The patient was hemodynamically stable and maintained off oxygen support. The patient was discharged after 3 weeks of admission and after 6 weeks follow-up chest X-rays show radiological recovery of perforation and the esophageal stent was removed [Figure 3].

The patient was asymptomatic at 8 weeks of follow-up, able to tolerate solids orally, and leading a normal life.

DISCUSSION

Boerhaave's syndrome is also known as spontaneous esophageal rupture or effort rupture of the esophagus. Hermann Boerhaave, a Dutch physician and professor of clinical medicine, first described spontaneous rupture of the esophagus, which typically occurs after forceful emesis.[4] It accounts for approximately 15% of all cases of spontaneous rupture of the esophagus and has a mortality rate up to 40%. The condition is highly associated with alcohol ingestion and excessive indulgence in food. Although vomiting is thought to be the most common cause, other rare causes include weightlifting, defecation, epileptic seizures, abdominal trauma, compressed air injury, and childbirth.[5] In our case, the diagnosis was not challenging as the patient presented with classic Mackler's triad of vomiting, abdominal pain, chest pain, and subcutaneous emphysema.

The differential diagnosis of Boerhaave's syndrome includes a variety of acute thoracic and abdominal conditions such as perforated peptic ulcer, Mallory–Weiss syndrome, pancreatitis, myocardial infarction, pulmonary embolus, ruptured aortic aneurysm, pneumonia, and spontaneous pneumothorax.[6] The laboratory tests are used only to excludes these common differential conditions. Pleural fluid with low pH, high amylase, and the presence of food particles are highly suggestive of esophageal perforation. In our case, pleural fluid had high amylase levels and low pH. Chest radiograph may be normal in 15% of cases and only helps to know related findings such as mediastinal emphysema, mediastinal widening, and pleural effusion. The diagnostic tool of choice is CT scan due to its high sensitivity and more detail assessment of the involved organs.[7]

The treatment options available are conservative, endoscopic, and surgical. As per study (J. P de Schipper *et al.*), conservative includes volume replacement and broad-spectrum antibiotic coverage. Surgical intervention includes primary esophageal tear repair through open thoracotomy. However, endoscopic treatment includes

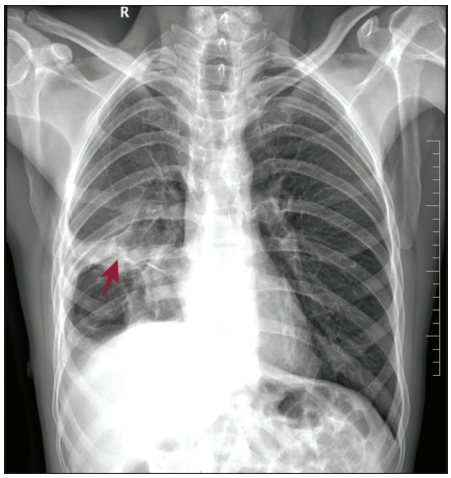

Figure 3: Follow-up chest radiograph showing complete resolution of pneumothorax and pneumomediastinum with small residual fibrotic consolidation in the right lower lobe

endoscopic esophageal stenting which is minimal invasive preferred in patients with delayed diagnosis (>48 h) and those with early diagnosis (<48 h) without widespread contamination or sepsis.[8] In our case, the patient diagnosed <48 h, without sepsis, so preferred for endoscopic stent placement and broad-spectrum antibiotics (Piperacillin + Tazobactum) 4.5 g IV 8th h for 10 days, along with Metronidazole 500 mg IV 8th h for 10 days.

The prognosis depends on the time of diagnosis. Delayed diagnosis and treatment usually are associated with poor outcomes. Despite all these, mortality as high as 20%–40% in treated and 100% in untreated cases.[9]

In conclusion, the endoscopic treatment seems to be an effective management strategy in patients with Boerhaave's syndrome. This case report could serve to implement nonsurgical modality of management and satisfactory outcomes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Turner AR, Turner SD. Boerhaave Syndrome. In: StatPearls (FL): StatPearls Publishing; 2019.
2. Spapen J, de Regt J, Nieboer K, Verfaillie G, Honoré PM, Spapen H. Boerhaave's syndrome: Still a diagnostic and therapeutic challenge in the 21st century. Case Rep Crit Care 2013;2013.
3. Han D, Huang Z, Xiang J, Li H, Hang J. "The Role of Operation in the Treatment of Boerhaave's Syndrome." BioMed Res Int 2018; 2018;5.
4. Janjua KJ. Boerhaave's syndrome. Postgrad Med J 1997;73:265-70.
5. Kumar TR, SourindraNath B, Arnab R, Anand Kumar P, Kaliprasanna C, Pratik B. Boerhaave's syndrome presented with right-sided hydropneumothorax and hoarseness of voice. Case Rep 2015;8:300-3.
6. Brauer RB, Liebermann-Meffert D, Stein HJ, Bartels H, Siewert JR. Boerhaave's syndrome: Analysis of the literature and report of 18 new cases. Dis Esophagus 1997;10:64-8.
7. Backer CL, LoCicero J 3rd, Hartz RS, Donaldson JS, Shields T. Computed tomography in patients with esophageal perforation. Chest 1990;98:1078-80.
8. Roh JL, Park CI. Spontaneous pharyngeal perforation after forceful vomiting: The difference from classic Boerhaave's syndrome. Clin Exp Otorhinolaryngol 2008;1:174-6.
9. Curci JJ, Horman MJ. Boerhaave's syndrome: The importance of early diagnosis and treatment. Ann Surg 1976;183:401-8.