

Case Report

Conservative management of Boerhaave's syndrome: a case report

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ABSTRACT

The management of esophageal perforation has evolved. The underlying principles of drainage of sepsis, adequate nutrition and repair, or defunctioning of the esophagus remain. We are presenting a case of Boerhaave syndrome, where we have successfully managed conservatively with endoscopic stenting. Endoscopic therapy and VATS are used with increasing frequency and in appropriately selected patients with good results. Careful selection of patients for such procedures are important. Implantation of self-expanding metallic stent in patients with esophageal leak or perforation is a safe and feasible alternative to operative treatment and can lower the interventional morbidity rate.

Keywords: Esophageal perforation, Endoscopic stenting, Leak

INTRODUCTION

Boerhaave's syndrome, also known as effort rupture of oesophagus, is defined as the spontaneous perforation of oesophagus due to sudden increase in intraesophageal pressure in association with negative intrathoracic pressure. This may occur in the setting of severe straining or vomiting. Oesophageal perforations are rare but are associated with high morbidity and mortality in the absence of timely intervention.³ Boerhaave's syndrome may occur even in a normal oesophagus, but predilection is more if there is presence of eosinophilic oesophagitis, drug induced oesophagitis, ulcer disease or Barrets oesophagus. The tear is most commonly seen in the left postero-lateral aspect of distal oesophagus. A similar condition known as Mallory Weiss syndrome involves the tear of mucosa alone, whereas in Boerhaave's syndrome, there is complete full thickness tear. The triad of vomiting, chest pain and subcutaneous emphysema is known as the Mackler triad.⁶ Endoscopic therapy for the management of esophageal leaks and perforations is a rapidly evolving field and of considerable therapeutic benefit in selected patients.² The aim of the study is to communicate our

experience in management of Boerhaave syndrome by endoscopic stenting.

CASE REPORT

A 70-year-old male patient presented with chief complaints of progressive burning type of abdomen pain over epigastric region-7 days, chest pain and breathlessness for past 3 days. There was history of 3 episodes of vomiting, non-projectile, non-bilious or non-blood stained, 2 days back. There was no history of fever/constipation/diarrhoea/recent trauma. On admission patient was conscious, oriented, afebrile, dyspnoeic and tachypnoeic.

His vitals were blood pressure (BP): 150/90 mmHg, pulse rate (PR): 124/min, respiratory rate (RR): 32/min, and SpO₂: 94%. On examining the respiratory system, air entry was reduced on the left hemithorax, and B/L diffuse crepitations were heard. Cardiovascular system –S1S2 heard, examination of abdomen revealed abdomen distension with tenderness over epigastrium and absence of bowel sounds. There was no guarding/rigidity/organomegaly. Blood investigations showed, mild

anaemia, leucocytosis, raised C-reactive protein (CRP). Blood sugar, renal function test (RFT), liver function test (LFT), electrolytes were normal. X ray chest showed B/L pleural effusion (L>R). Ultrasonography (USG) abdomen showed no detectable abnormality.

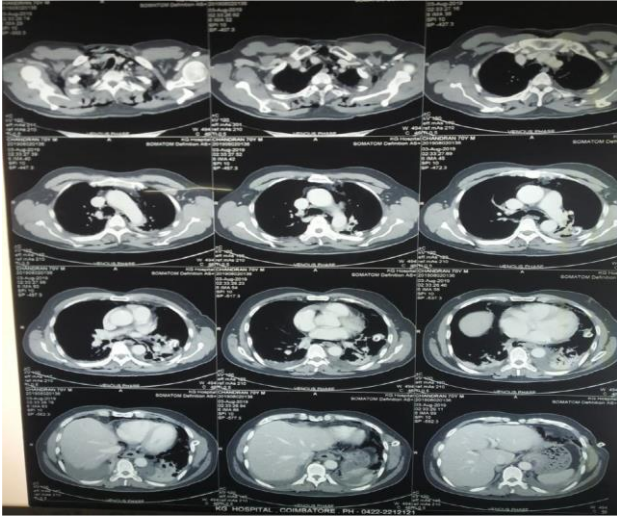


Figure 1: CECT chest and abdomen- shows extensive pneumomediastinum, minimal left pneumothorax, B/L mild pleural effusion with passive atelectasis of adjacent lung parenchyma, active contrast extravasation from left posterolateral wall of lower oesophagus.



Figure 2: X ray chest shows B/L pleural effusion (L>R).

Contrast enhanced computed tomography (CECT) chest and abdomen showed extensive pneumomediastinum, minimal left pneumothorax, B/L mild pleural effusion with passive atelectasis of adjacent lung parenchyma, active contrast extravasation from left posterolateral wall of lower oesophagus (around 5.5 cm proximal to OG junction) suggestive of oesophageal perforation. Patient treated with nasal oxygen, NPO, IV fluids, higher IV antibiotics (aminoglycoside, cefoperazone sulbactam and anaerobic coverage), proton pump inhibitor (PPI), bronchodilators and analgesics. Left ICD insertion done,

about 600 ml of purulent fluids admixed with food particles drained. Pleural fluid analysis showed, sugar-118 mg%, proteins-3.5 g%, lactate dehydrogenase (LDH)-1642 IU/l, amylase-438 IU/l. Pleural fluid C/S showed – *E. coli* growth, which was sensitive to cephalosporins. Patient’s nutritional support was aided with TPN. On 3rd day after admission, patient underwent OGD scopy with caution, which showed full thickness tear in distal esophagus at 35-38 cm for about 3-4 cm. On 10th day OGD guided self-expanding metallic stent (12 cm length, 18 mm diameter) was deployed over 30-42 cm segment of the esophagus encompassing the perforation, and ryles tube inserted, ryles tube feeding started. ICD removed on 13th day. Patient discharged following day.



Figure 3: CECT chest- shows extensive pneumomediastinum.

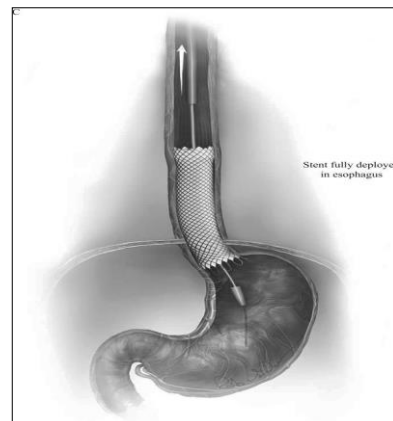


Figure 4: Stent deployment.

DISCUSSION

The management of esophageal perforation has evolved. The underlying principles of drainage of sepsis, adequate nutrition and repair, or defunctioning of the esophagus remain. Conservative treatment of esophageal perforation is accepted in patients that meet the Cameron criteria. Described in 1979, those criteria are: a well circumscribed intramural or transmural perforation, no signs or

symptoms of sepsis, a contained perforation, no distal obstruction or malignant esophageal disease, and perforation that is not located in the abdominal esophagus.⁷

Endoscopic therapy and VATS are used with increasing frequency and in appropriately selected patients with good results. The importance of patient selection is highlighted by Cameron et al.¹ Endoluminal suturing may be used to the close spontaneous perforations and leaks related to esophageal reconstruction. Depending on the size of the perforation, a partially covered stent may also be used. These stents can be sutured in place endoscopically in an attempt to reduce the risk of migration and persistent leak.

Esophageal stenting for leak or perforation is not without risk. An esophageal stent may erode local structures, for example, aorta if left in place for too long, however, not long enough and the perforation or leak would not be healed at the time of stent removal. Stents may also migrate or develop stent leaks. Freeman et al suggest removal of stents for anastomotic leak at ≤ 2 weeks and for esophageal perforation ≤ 4 weeks.² If there is a persistent leak based on endoscopic or radiological examination or both at the time of stent removal another stent can be placed with a different “landing zone” in an attempt to distribute the pressure on the esophageal wall.²

CONCLUSION

The challenge with Boerhaave syndrome is to establish diagnosis early enough for adequate management.⁴ Surgery may be the only option after late diagnosis. However, a recent study found that mortality risk was not related to wait time exceeding 24 hours.⁵ First-line treatment should be conservative. Implantation of self-expanding metallic stent in patients with esophageal leak or perforation is a safe and feasible alternative to operative treatment and can lower the interventional morbidity rate.²

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