

Editorial

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Management of Boerhaave's Syndrome

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Boerhaave's syndrome or spontaneous oesophageal perforation is characterised by barogenic oesophageal injury. This leads to contamination of the pleural cavity with enteric contents and various degrees of injury.¹ The syndrome is named after Herman Boerhaave, a Dutch physician, who first described it in 1724. Incidence of spontaneous rupture amongst all oesophageal ruptures varies between 15-38%.^{2,3} Mallory-Weiss tears are assumed to represent part of the spectrum of the spontaneous perforation, but it is likely that these mucosal injuries reflect shearing rather than barogenic trauma.⁴ Spontaneous oesophageal perforations are associated with the highest mortality amongst all gastrointestinal perforations with an overall rate of nearly 30%. Early diagnosis and definitive surgical management as soon as possible after the presentation of symptoms, indisputably within the first 24 hours show the best outcomes. If treatment is started within 24 hours from the onset of symptoms, mortality rate is observed to be below 10%; after 24 hours, it is approximately 65% and after 48 hours, it reaches 75%-89%. If left untreated it rises up to 100%.^{5,6}

Oral, water-soluble contrast medium is the most used and preferred method of investigation for visualisation of the oesophageal leak. Although this method helps in revealing most cases of oesophageal perforation, it may be associated with up to 66% false negative results and up to 90% sensitivity.⁷ Water-soluble contrast should be used instead of barium contrast to prevent barium-related inflammation of the mediastinum if there is any perforation. If the initial contrast-swallowing study is negative, imaging can be repeated after 4-6 hours if the clinical suspicion still remains high. In approximately 90% of the cases, the area of perforation and contrast leak is at the left posterolateral aspect of the distal third of the oesophagus, usually within 2-3 cm above the oesophagogastric junction.⁸ The complication risk of endoscopic assessment (OGD) is minimal and it excludes the diagnosis if normal. It also influences the management if underlying pathology, such as cancer is found and facilitates placement of nasojejunal feeding tube if required. Computed tomography (CT) with oral and intravenous contrast is frequently performed in critically ill patients. Apart from showing a leak, it reveals the degree of contamination and associated extra-oesophageal insult; in addition, it has a significant role in the decision-making process as well as post-operatively in patient assessment.⁹

Management can be conservative, surgical, endoscopic or a combination and hugely depends on the patient condition and timing of presentation. It is clear that surgery remains the main stay of treatment for Boerhaave's syndrome. Management of the condition should preferably be performed in a specialist oesophageal unit with high expertise. Admission to intensive care unit is usually necessary.

Non-operative management may be appropriate in a small number of patients who are diagnosed early, with minimal contamination and no mediastinitis. It can also be reserved for those with delayed diagnosis that have been stable. All patients should be nil by mouth and receive urgent respiratory and cardiovascular support plus opiate based analgesia. Intravenous fluids, urinary catheter and close fluid monitoring should be given. In addition, broad-spectrum antibiotics and antifungal plus intravenous proton pump inhibitors to lower the acid exposure are strongly recommended. Part of the management includes placement of intercostal chest drains

and nasogastric tube, which should only be done under image guidance. Early enteral feeding is advised in all cases. Laparoscopic or open feeding jejunostomy and venting gastrostomy are advised for nutrition and drainage respectively. Although, usage of covered self-expandable stents as primary treatment has recently gained popularity, it still remains limited with sealing leakage failure rates of up to 50%.^{10,11} Non-operative trans-oesophageal debridement with mediastinal irrigation has also been used with acceptable results. Nevertheless, there are limited cases described in the literature to reach any robust conclusions.¹²

Objective of surgery is to restore oesophageal integrity and prevent further soiling. Debridement, drainage and lavage are more important than the type of repair.¹³ The majority of oesophageal perforations present with left chest contamination (90%) and a left posterolateral thoracotomy appropriate in most cases. Longitudinal oesophageal myotomy is advised as the mucosal injury is usually longer than the muscular one and mucosal debridement may additionally be necessary.¹⁴ The controversy is with the late perforations by the time diagnosis is made the wound edges may have become oedematous, stiff or friable rendering primary repair risky due to the high rate of breakdown. Frequently, there is associated mediastinitis and empyema. In these cases, primary repair may not be feasible. Late perforations can therefore be managed with debridement of the pleural cavity and mediastinum, oesophagectomy and placement of feeding gastrostomy.¹⁵ As a general rule, oesophageal resection is reserved for damage to diseased oesophagus, extensive trauma or delayed presentation; clinical judgment is of paramount importance and that is one of the main reasons why management of the condition in a specialist oesophageal unit is required. Immediate primary reconstruction with oesophago-gastric anastomosis in cases with minimal contamination or a delayed one (oesophageal diversion with cervical or tube oesophagostomy and reconstruction at a second stage) in profuse contamination and/or physiologic instability are the options.

Primary closure or closure over a *t*-tube is a suitable option, especially in cases with early presentation.¹⁴ Primary repair is the most common procedure and should always be considered for those with early onset of symptoms. Single or 2 layered closure using absorbable sutures can be performed with an oesophageal bougie rarely utilised now-a-days.¹⁶ Associated leak rate after primary repair performed within the first 24 hours of presentation is in the area of 20% and if treatment is delayed for more than 24 hours it can rise up to 50%.¹⁷ Re-enforcing the repair with a patch of nearby tissue like pericardium, pleura or pericardial fat, may reduce the leak rate.¹⁸ T-tube repair was developed in the 1970's mainly for cases with late presentation and it can work well for most repairs.¹⁹ The concept is based on a controlled oesophago-cutaneous fistula, where a T-tube (6-10 mm in diameter) is placed through the tear with the limbs of the tube beyond the boundaries of the perforation. The oesophageal wall is approximated loosely over the tube with absorbable sutures. The tube is subsequently externalised and secured at the skin. Basal and occasionally apical chest drains are inserted. Oesophageal healing is monitored with contrast studies. T-tube is generally removed between 3-6 weeks, depending on the clinical and radiological progress. Sulpice et al²⁰ in his comparative study of T-tube repair and primary repair, found no difference between the two, even though the time from symptom onset to surgery was longer for the T-tube repair. In the era of endoscopic surgery, video-assisted thoracoscopic surgery (VATS) could not have been unutilised in the treatment of Boerhaave's syndrome. Haveman et al²¹ showed that VATS can be used as the first choice and is a safe approach associated with a lower rate of complications and similar results in comparison with open surgery.²¹ A Korean study, showed similar outcomes of thoracoscopic repair *versus* open repair, even at delayed presentation.²²

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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