Single Procedure Esophageal Bypass: A Valid Option In Delayed Surgical Intervention For Complicated Esophageal Perforation

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Citation

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Abstract

We present a case report of Boerhaave syndrome or spontaneous post emetic rupture of esophagus complicated with esophagopleural fistula whose diagnosis was missed at the initial presentation in the emergency. Surgical intervention was delayed for more than two weeks until the esophageal perforation was recognized. The patient was optimized for surgery and a single stage esophageal bypass surgery was done where the esophagus was left in situ and the stomach was pulled up through the sub sternal route and a cervical esophagogastric anastomosis was made to restore the gastrointestinal continuity; thus avoiding the morbidity associated with an esophagostomy and a second procedure for delayed reconstruction.

INTRODUCTION

Boerhaave's syndrome or post emetic rupture of esophagus described by Hermann Boerhaave in 1724¹ is a rare but serious entity that is difficult to diagnose because often no classical symptoms are present and delay in seeking medical care is common. Accurate diagnosis and effective treatment depend upon early recognition of clinical features and accurate interpretation of diagnostic imaging. Outcome is determined by the cause and location of the injury, the presence of concomitant esophageal disease, and the interval between perforation and initiation of therapy².

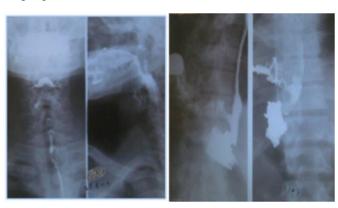
CASE REPORT

A 26 year old man presented to the medical emergency department of Mayo Hospital Lahore with complaint of severe chest pain for the last 12 hours. The patient after having a large meal about 12 hours before the onset of pain had had episodes of forceful vomiting one of which contained blood in it. On presentation the patient's pulse was 124/min, respiratory rate 38/min, and blood pressure of 90/50mmHg, with shifting of trachea to the right and absent breath sounds on the left side but no subcutaneous emphysema was appreciated. Arterial blood gas analysis showed oxygen saturation of 86% with decreased pO₂ levels. Full blood count showed leukocytosis with predominance of neutrophils and a normal platelet count; renal function tests,

liver function tests, serum electrolytes, blood glucose level, serum amylase and PT(INR)/APTT were all within normal range. Patient's EKG did not show any ischemic changes as well. However his chest radiograph showed hydropneumothorax with a collapsed lung on the left side. Urgent tube thoracostomy was done upon which about 500mL of yellowish fluid was revealed. The patient stabilized after the intubation with oxygen saturation returning to 97.3% with pO₂ 90.1 mm Hg. The pleural fluid examination revealed a leukocyte count of 52000/dL with 95% polymorphs and protein content of 3.4g/dL. The patient was given intravenous fluids and parentral antibiotics which included ceftriaxone 2g along with 500mg of metronidazole and an infusion of omeprazole. After the initial resuscitative measures the patient was shifted to the medical ward and the empirical antibiotic regimen was continued. The patient remained vitally stable during the next week with occasional spikes of fever. The chest tube reveal however started turning turbid despite the antibiotic therapy which was modified according to the culture and sensitivity report. It was also noted that the color of the reveal changed when the patient took colored fluids. This finding prompted to get a gastrograffin study of the esophagus and make the patient NPO, after about two weeks of the initial presentation. The contrast study outlined a fistulous tract along the left lateral wall of the distal one third of the esophagus communicating with the left pleural cavity. The contrast however passed

freely into the stomach.

Figure 1Contrast study shows leak of dye from the lower part of esophagus.



The patient was then shifted to the department of surgery with a diagnosis of lower esophagopleural fistula as a complication of post emetic spontaneous rupture of esophagus. The patients NPO regimen was maintained, nasogastric tube was passed, keeping it above the perforation and he was given broad spectrum antibiotics, blood transfusions and total parenteral nutrition (with about 3500 Kcal and 8g of nitrogen per day) through a central line to improve his general condition. Within a week the patient was optimized and he underwent esophageal bypass surgery; a midline abdominal incision was made and the left lobe of liver and stomach was mobilized together with the esophagogastric junction after ligating the short gastric, left gastric and left gastroepiploic vessels. The stomach was divided from the abdominal esophagus and closed with vicryl 3/0 in two layers. The esophageal end was left open. Left sided oblique neck incision was made to expose the cervical part of esophagus. The esophagus was mobilized and stomach pulled up through the sub sternal route based on the right gastric and right gastroepiploic arteries. Cervical part of esophagus was divided and gastroesophageal anastomosis was done with prolene 4/0 interrupted sutures in single layer. Distal end of esophagus was left as such. A 32F tube drain was placed in the abdomen near the lower end of the esophagus and a suction drain was placed in the neck. The patient was mechanically ventilated for 7 days post operatively on SIMV mode, during which time he remained vitally stable.

Figure 2Picture 1: Stomach mobilized based upon the right gastric and right gastroepiploic arteries.

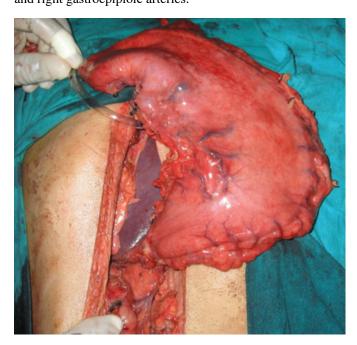


Figure 3 Picture 2: Space created in the sub-sternal plane.

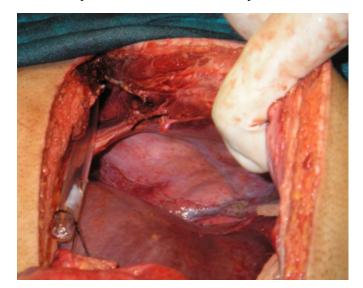


Figure 4Picture 3: Stomach pulled up and cervical part of esophagus mobilized.

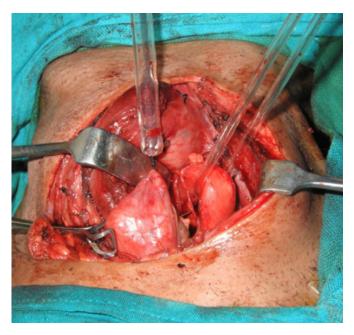


Figure 5Picture 4: Esophagogastric anastomosis made in the neck with single layer prolene 4/0 sutures.



The patient was allowed oral fluids and subsequently solid food after being weaned off from ventilator. The patient's drains were taken out on the 7th post op day. His chest extubation was done on 15th day post op after the sepsis in pleural cavity had settled. Gastrograffin study performed two weeks later showed no extravasation of dye and a patent anastomosis with free passage into the stomach.

DISCUSSION

Boerhaave syndrome is a rare spontaneous, transmural, post emetic perforation of the esophagus, although iatrogenic esophageal perforations during diagnostic or therapeutic procedures are more common. Spontaneous rupture may also occur during childbirth³, defecation⁴ seizures⁵, prolonged coughing or laughing or weightlifting⁶. This syndrome is postulated to be due to neuromuscular in-coordination resulting in failure of cricopharyngeus to relax against the sudden increase in intraluminal esophageal pressure during emesis. Spontaneous rupture is mostly located in the lower part of the esophagus on the posterolateral side next to the cardia and can reach upto 6 cm in length, this site has been implicated because of entry of nerves and vessels, lack of angulation of organ and absence of supporting tissue^{7,8}.

The classical presentation of Boerhaave's syndrome constitutes Mackler's triad which includes vomiting, chest pain and subcutaneous emphysema⁹. There can however be variable presentation and these classical symptoms are sometimes not seen which can pose a diagnostic challenge^{6,10}.

The management of esophageal perforation depends upon the delay in presentation and diagnosis, extent of perforation and overall medical condition of the patient. Non operative management of esophageal perforation is advisable in minority of cases and is exceedingly rare in Boerhaave syndrome. Criteria for nonoperative treatment includes the following: disruption contained in the mediastinum or between the mediastinum and visceral lung pleura; drainage of the cavity back into the esophagus; minimal symptoms; and minimal signs of clinical sepsis¹¹. These patients can be managed sometimes just by oral hygiene, cessation of oral intake, antibiotics and nutritional support given parenterally or enterally distal to the site of injury^{12,13,14}. Endoluminal esophageal stent placement with pleural drainage is also an effective treatment of spontaneous esophageal perforations^{15,16,17}. These stents result in rapid leak occlusion, provide the opportunity for early oral nutrition, may significantly reduce hospital length of stay, are removable, and avoid the potential morbidities of operative repair¹⁸.

However the conservative mode of treatment is more applicable to esophageal perforations due to iatrogenic injuries ¹⁹⁻²³. Patients with Boerhaave syndrome often require surgical intervention. The aim of all surgical options to esophageal perforation is drainage of the infected mediastinum and elimination of the ongoing soilage ²⁴. Transthoracic primary repair is the gold standard if the patient is diagnosed within 24 hours of the spontaneous perforation ²⁵; it was however not recommended if this time limit exceeded ²⁶⁻²⁹. With delayed diagnosis or presentation,

thorough evaluation of the rupture size, the amount of contamination, and the extent of necrosis and edema are essential in determining a therapeutic course³⁰. Recent evidence shows that primary surgical repair with or without reinforcement is successful in treating esophageal perforation even after 24 hours 28,29,31-38. In cases where primary repair is deemed not to be possible successful management with esophageal T tube has been advocated^{7,39-41}. The patients who present or are diagnosed very late may have systemic sepsis with mediastinal contamination from esophageal secretions and gastroesophageal reflux. Here, the esophageal tissue may be devitalised to the extent that repair is not possible. Several exclusion and diversion techniques have been reported for the treatment of esophageal perforation following late diagnosis and development of extensive contamination^{28,39,42-48}

The ongoing presence of the original septic focus and potentially incomplete control of soilage within the mediastinum have limited the success of exclusion and diversion techniques²⁴. In such cases, either transthoracic and transhiatal esophagectomy or closure of the cardia with formation of cervical esophagostomy and delayed reconstruction have been advocated in various studies and reports⁴⁹⁻⁵⁷.

In our case the patient had presented very late with mediastinal sepsis and the formation of an esophagopleural fistula resulting in an empyema. Primary repair in this scenario was not possible. Intense sepsis and fibrosis in the esophageal bed did not allow for the esophagus to be mobilized and resected so it was left in situ and a surgical by pass was made by stomach pull up through the sub- sternal route and a cervical esophagogastric anastomosis. This avoided the morbidity associated with an esophagostomy and a two stage procedure with delayed reconstruction. Mediastinal sepsis and the empyema settled subsequently. The tailored surgical management for this patient proved to be a success as the patient recovered completely. We recommend this one stage procedure in cases where the presentation of esophageal perforation is very late prohibiting the popular course of treatment to be followed.

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