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Case Report

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Spontaneous esophageal perforation as a cause of upper gastrointestinal bleed – A Rarity

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Abstract

A 49 year old patient was referred to our emergency department with hematemesis and melena following forceful vomiting. Endoscopy revealed a Mallory Weiss tear and a large ulcer, computed tomography (CT) scan revealed a left-sided esophageal rupture with accompanying pneumomediastinum. Along with conservative management, endoscopic bridging of the tear with a self-expandable metal stent (SEMS) was done after which there was a complete recovery. Boerhaave's syndrome or spontaneous esophageal perforation is a rare but potentially fatal condition characterized by a transmural tear of the distal esophagus. Diagnosis is always challenging and presentation may be atypical. Management is multidisciplinary. We present an atypical presentation of a rare disease with treatment challenges, managed endoscopically with a successful outcome.

Keywords: Boerhaave's syndrome, Esophageal perforation, Hematemesis, Self-expanding metallic stent (SEMS), Mallory Weiss tear

Introduction

Spontaneous esophageal perforation or Boerhaave's syndrome is a rare condition with excessive mortality rate of 20-40%. The Dutch physician Hermann Boerhaave first described it in

1724¹ as an unusual, life-threatening condition requiring prompt diagnosis and treatment. Any delay can lead to potentially lethal complications necessitating a high index of suspicion for timely diagnosis with well-selected radiological and endoscopic investigations.

Case Report

A 49 yr old male, chronic alcohol consumer with no known co-morbidities and h/o binge drinking presented with recurrent vomiting of 01 day duration followed by 03 bouts of hematemesis and melena. Initial clinical evaluation at peripheral centre revealed tachycardia and hypertension. He was managed with IV fluids, Inj Pantoprazole, supportive care and referred to our centre. Evaluation at our centre revealed pain abdomen, giddiness, and persistence of melena with no associated h/o chest pain or breathing difficulty. Repeat clinical evaluation revealed a pulse of 110/ min, blood pressure of 130/90 mm Hg, respiratory rate of 26 / min, oxygen saturation of 100%, tremors, hepatomegaly and epigastric tenderness. Ryle's tube aspirate showed altered and fresh blood. IV fluids, Inj Pantoprazole 80 mg stat IV and 8 mg /hr, Inj Ondansetron 8 mg IV stat/ SOS and supportive care were continued. Urgent esophagogastroduodenoscopy revealed a deep ulcer 3x2 cm with Mallory Weiss (MW) tear on opposite side at GE junction with active ooze from MW tear, which was managed with Inj

Adrenaline (1:10,000) injection into the oozing site and hemostasis was achieved (Figure 1). Other investigations revealed polymorphonuclear leucocytosis (TLC – 13500 /cu.mm) and mild thrombocytopenia (Platelet 109000/ cu.mm). NCCT chest showed esophageal tear and pneumomediastinum (Figure 2). Follow up evaluation showed persistent epigastric tenderness, decreased air entry in the base of left lung. He was initiated on antibiotics (Inj Meropenem, Inj Amikacin and Inj Metronidazole) and a GI surgical consult was taken. Endoscopic management with a 7.5 cm ELLA removable covered self-expanding metal stent (SEMS) placement in the esophagus from 33-41 cm (GE junction at 38 cm) was done (Figure 3). The patient's condition stabilized over the next one week. The antibiotics were continued for 02 weeks and subsequently followed up after 6 weeks. He remained asymptomatic with a normal clinical examination. Investigations and CXR were normal. Follow up UGIE revealed no ulcer /scar, SEMS was not seen in situ (Figure 4). On follow up the patient is asymptomatic.



Figure 1: Deep ulcer at GE junction.



Figure 2: NCCT Chest with pneumomediastinum



Figure 3: SEMS placement beyond to GE junction



Figure 4: Follow up endoscopy shows normal esophagus

Discussion

Boerhaave's syndrome is an emergency wherein there is a spontaneous rupture of the esophagus, in the lower third of the left postero-lateral wall (90% cases). Its pathophysiology is characterized by a rapid rise in intra-luminal pressure resulting in a barogenic rupture of an anatomically weak region causing a tear 3–6 cm above the diaphragm (average 2 cm). It is an extremely uncommon cause of substantial haemorrhage¹. The classical history is of food or drink overindulgence resulting in severe or frequent vomiting followed by severe chest pain, dyspnoea and presence of subcutaneous emphysema and shock (Macklers triad)¹⁻³. This classical triad is rarely encountered; even our patient had only two features of this triad. Decreased breath sounds on the side of perforation, and presence of Hamman's sign (mediastinal crackling accompanying every heart beat) in left lateral decubitus position usually indicates mediastinal contamination/emphysema¹⁻³. Both these signs were absent in our patient, possibly due to a early diagnosis.

Differential diagnosis is confounding as various entities like perforated ulcer, myocardial infarction, pulmonary embolism, aortic emergency, dissecting aneurysm and pancreatitis may present in a similar manner^{1,2}.

On radiological evaluation, abnormalities such as mediastinal, free peritoneal air, pre-vertebral or subcutaneous air may be present, depending on the location of perforation⁴. An inverted V sign may be noted occasionally, which is a radiolucent streak of air dissecting the retrocardiac fascial planes⁵. In up to 15% of patients' chest x-ray may be normal; in fact, diagnosis would have been missed in our patient, if only CXR had been done. Contrast enhanced CT scan of the chest and upper abdomen is the preferred sensitive examination modality that shows esophageal wall edema, extra-esophageal air, peri-esophageal fluid collections, pneumothorax, pleural effusion and pneumo retroperitoneum although the site of the perforation may not always be localized⁶. Lastly, cautious endoscopy enables direct visualization of the location and extent of the perforation.

Boerhaave's syndrome can be managed conservatively, endoscopically and by surgery^{5, 6}. Patients with small or well-contained perforation are managed conservatively with cessation of oral intake, fluid administration, parenteral nutrition, broad-spectrum antibiotics, proton pump inhibitors with mediastinal, pleural, or abscess drainage⁶. Endoluminal placement of a self-expandable metallic stent (SEMS) to bridge an esophageal tear has shown encouraging results in various case reports^{7, 8}, and Persson et al (2014), in their case series, have successfully managed patients with SEMS⁹. In a systematic review of stent placement for esophageal leaks by Boeckel et al (2011)¹⁰, including 46 patients of Boerhaave's with complete follow up, only 7 patients required a subsequent surgical procedure with sealing rates ranging from 75 to 100% and migration rates ranging from 9 to 57% (more for plastic stents). In another meta-analysis by Biancari et al (2013)¹¹, of the pooled mortality of 11.9% for esophageal perforations, 14.8% mortality was noted for spontaneous esophageal perforations and stent-grafting was associated with somewhat lower mortality rates, although bias was a confounding factor. Nevertheless, endoscopic stenting is operator dependent, and can be associated with worsening mediastinal or pleural contamination, accidental stent migration or even increased mortality, as noted in a recent study by Schweigert et al (2013)^{1, 12} in which 11 of 13 patients in the stent group mandated an operative intervention and three of 13 required repeated surgery. Therefore, need for minimal insufflation and utmost care is the norm.

In patients with sepsis, large uncontained leaks, and extensive contamination early in the disease course, De Schipper et al (2009) and Abbas et al (2009) advocate primary surgical repair^{1, 13}. The success rates of open thoracotomy and video-assisted thoracoscopic surgery (VATS) were found to be similar by Haveman et al. (2011)¹⁴. Success in our case was related to the early detection and immediate stenting that prevented contamination.

Conclusion

An ideal approach for managing a case of Boerhaave's syndrome requires having a high index of suspicion in patients presenting with forceful vomiting and hematemesis. Initial assessment (clinical and endoscopy) followed by close observation and the use of CT scan for prompt diagnosis of the leak as well as the complications is of paramount importance. Subsequently, early therapy as mandated, endoscopic in our case, can be vital in decreasing morbidity and mortality.

Conflicts of interest: Nil

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