

Left Sided Chest Pain: A Case Report of Boerhaave Syndrome Mimicking Acute Coronary Syndrome

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Abstract

The Boerhaave syndrome (Boerhaave 1742) or spontaneous rupture of the oesophagus is an unusual condition, which is rarely encountered in clinical practice. It is crucial to establish the diagnosis of this condition promptly. The mortality and morbidity is directly proportional to the delay in diagnosis and the severity of mediastinitis due to mediastinal contamination. Although its clinical features had been well described, an occasional case do present with some diagnostic difficulties unless there is a high index of suspicion. Here, we report a patient who presented with left sided chest pain that mimics acute coronary syndrome and diagnosed as Boerhaave syndrome.

Key words: Boerhaave syndrome, spontaneous rupture of the oesophagus, chest pain, acute coronary syndrome

Introduction

Boerhaave (1724) described the first case of spontaneous rupture of the oesophagus in Baron van Wassenaer, the Grand Admiral of the Fleet of Holland.¹ Although several well documented reviews had been published since then, this condition is still very rarely seen by an individual clinician, or even in a single institution. The mortality rate of this condition is high if not treated appropriately and promptly. We describe a patient who was diagnosed as Boerhaave syndrome with clinical presentations mimicking acute coronary syndrome.

Case Report

61-year-old gentleman with history of hypertension presented with sudden onset of left sided chest pain that radiating to jaw and left shoulder. The chest pain was associated with nausea, sweating and breathlessness. One day prior to admission, he experienced several episodes of diarrhoea. The frequency of diarrhoea increased over the rest of the day and he developed vomiting later in the afternoon. He consulted general practitioner and he was given antiemetic. Over the next few hours, the vomiting became more frequent. The left sided chest pain was immediately preceded by the vomiting.

In the emergency room, his blood pressure was 90/50 mmHg, oxygen saturation on room air was 89%, pulse rate was 110 beats per minute and respiratory rate was 40 breaths per minute. On examination, the breath sounds were diminished from left midzone to lower zone as well as in right lower zone. He appeared to be diaphoretic. He was diagnosed as acute coronary syndrome with cardiogenic shock and was treated with aspirin, clopidogrel, morphine, and statin. Electrocardiography at that time revealed sinus tachycardia and T inversion about 1 mm at V5 and V6. Chest radiograph demonstrated the presence of left pleural effusion. He was subsequently referred to a cardiologist. Cardiac enzymes such as Troponin T and creatine kinase MB (CK-MB) levels were within normal range.

Thoracic computed tomography (CT) was performed in order to exclude aortic dissection. It confirmed the presence of moderate left hydropneumothorax with pneumomediastinum extending up to the neck. (Figure 1) A chest tube was inserted immediately and drained 1 litre of coffee-ground material. The diagnosis of Boerhaave syndrome was then made. He was resuscitated until the vital parameters were stabilised. Thereafter, a left thoracotomy and primary oesophageal repair was performedurgently.

Postoperatively, he developed aspiration pneumonia and mediastinitis. An oesophagogram by using Gastrografin showed about 3 mm leakage near the oesophagogastric junction on postoperative day 7. He was treated conservatively and repeated Gastrografin study on day 14 showed no leakage. Chest tube was continuously draining about 50 to 100 cc purulent fluid per day for about 4 weeks. The chest tube was converted to silicon percutaneous endoscopic gastrostomy (PEG) tube (modified T tube conversion) later, with the aim to create anoesophagocutaneous fistula. He was discharged with the PEG tube and there were continuously purulent fluid draining about 40 to 50 cc per day. Repeated chest radiography on Week 6 revealed no evidence of pneumomediastinum and recurrent left pleural effusion. The PEG tube was then replaced by colostomy bag and attached to the opening of the fistula. (Figure 2) The drainage of purulent fluid finally stopped at week 8. He was well since then.

Discussion

Diagnosis of Boerhaave syndrome is often missed or delayed as a result of its heterogeneity and rarity in clinical presentation. The classical presentation of this disease is namedMackler's triad (vomiting, chest pain and subcutaneous emphysema)which is not regularly seen. This typical presentation was reported only 14% in patients with Boerhaave syndrome.² This condition was

often wrongly diagnosed as perforated ulcer, myocardial infarction, pulmonary embolism, dissecting aneurysm and pancreatitis. The diagnostic error rate is relatively high. According to Keighley et al.³ 1 in every 2 patients was misdiagnosed. The mortality rate increases more than 50% if intervention delayed longer than 24 hours, and it is even higher (almost 90%) after 48 hours. Conversely, the mortality rate falls to 10-20% with early diagnosis and appropriate intervention within 12 hours.⁴⁻⁶

Chest radiograph plays a crucial role in diagnosis of Boerhaave syndrome as it commonly reveals left sided pleural effusion, subcutaneous emphysema, air under diaphragm or pneumomediastinum (Naclerio's V-sign).⁷ Water soluble contrast studies (oesophagogram) have offered better accuracy in diagnosing perforation; however, these studies are associated with high false negative results (27-66% cases).⁸ With thoracic CT, Boerhaave syndrome can be confirmed by the findings of paraoesophageal air tracks, pneumomediastinum, pneumothorax, pleural effusions and air-fluid level at the site of perforation.⁹ Some of the literature had suggested thoracocentesis as an alternative method to diagnose Boerhaave syndrome (presence of food particles, gastric juice pH < 6, high amylase content or squamous cells from saliva in the drainage).¹⁰ In last few years, there are some publications reported the use of flexible endoscopy in confirming Boerhaave syndrome. Although this is more specific and sensitive, it is associated with considerable risk especially in an acutely ill patient.¹¹

The ultimate aim of all the interventions are avoidance of further contamination from oesophageal perforation site, debridement of necrotic tissues, eradication of infection, lavage and drainage of the affected mediastinum and pleural cavities. Generally, primary oesophageal repair and reinforcement is commonly practised as 'gold standard' if diagnosis is established within 24 hours. T-tube diversion or oesophagocutaneous fistula creation is recommended in patients with complications of oesophago-pleural communication and sepsis. Oesophagectomy is usually reserved to ill patients with damaged oesophagus resulting from delayed diagnosis; however, this procedure is associated with poor outcome.

Conclusion

This case report highlights typical presentation of acute coronary syndrome (left sided chest pain) could be a presentation of Boerhaave syndrome. Considering the high mortality rate, the diagnosis of Boerhaave syndrome needs be established urgently. A high index of clinical suspicion of the disease leading to early diagnosis and resuscitation remains the cornerstone of the favourable outcome in terms of mortality and morbidity. Hence, the possibility of Boerhaave syndrome should be kept in mind when clinicians encounter a patient presenting with left sided chest pain that mimics acute coronary syndrome, especially if the symptom immediately following vomiting (pain following a sudden raise of oesophageal intraluminal pressure).

Conflict of Interest: None

References

1. Herbella FAM, Matone J, Del Grande JC. Eponyms in esophageal surgery, part 2. *Dis. Esophagus*. 2005;18(1):4–16.
2. Griffin SM, Lamb PJ, Shenfine J, Richardson DL, Karat D, Hayes N. Spontaneous rupture of the oesophagus. *Br. J. Surg*. 2008;95(9):1115–20.
3. Keighley MR, Girdwood RW, Ionescu MI, Wooler GH. Spontaneous rupture of the oesophagus. Avoidance of postoperative morbidity. *Br. J. Surg*. 1972;59(8):649–52.
4. Richardson JD, Martin LF, Borzotta AP, Polk HC. Unifying concepts in treatment of esophageal leaks. *Am. J. Surg*. 1985;149(1):157–62.
5. Henderson JA, Péloquin AJ. Boerhaave revisited: spontaneous esophageal perforation as a diagnostic masquerader. *Am. J. Med*. 1989;86(5):559–67.
6. Brewer LA, Carter R, Mulder GA, Stiles QR. Options in the management of perforations of the esophagus. *Am. J. Surg*. 1986;152(1):62–9.
7. Marinis A, Rizos S. Boerhaave' s Syndrome or Spontaneous Perforation of the Oesophagus. *Hellenic Journal of Surgery*2011:258–262.
8. Buecker A, Wein BB, Neuerburg JM, Guenther RW. Esophageal perforation: comparison of use of aqueous and barium-containing contrast media. *Radiology*. 1997;202(3):683–6.
9. Khan a. Z, Strauss D, Mason RC. Boerhaave's syndrome: Diagnosis and surgical management. *Surg*. 2007;5(1):39–44.
10. Jones WG, Ginsberg RJ. Esophageal perforation: a continuing challenge. *Ann. Thorac. Surg*. 1992;53(3):534–43.
11. Schmidt SC, Strauch S, Rösch T, et al. Management of esophageal perforations. *Surg. Endosc*. 2010;24(11):2809–13.



Figure 1: Thoracic CT showing pneumomediastinum and periaortic air tracks



Figure 2: Colostomy bag (with purulent discharge) attached to the opening of the fistula