CASE REPORT

Spontaneous Oesophageal Perforation: A Case Report

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ABSTRACT

A 49-year-old gentleman presented with epigastric pain for one day associated with one episode of vomiting and dyspnoea. Respiratory examination showed reduced breath sound over his left lower zone. He was treated as left spontaneous pneumothorax and left lung empyema requiring left chest tube insertion and intravenous antibiotics. His left pleural fluid biochemistry result was exudative while its centrifuge showed empyema. In ward, we noticed food material draining from his left chest tube during feeding. An urgent contrast enhanced computed tomography (CECT) thorax showed a left oesophageal-pleural fistula with possible broncho-oesophageal fistula. During oesophagogastroduodenoscopy (OGDS), air bubbles were seen in his left under-water chest drainage during air-insufflation of the oesophagus. The revised diagnosis was Boerhaave syndrome. He was treated with an esophageal stent to cover the perforation and a left lung decortication via video assisted thoracoscopic surgery (VATS) for his left empyema. He improved and was discharged well.

Keywords: Boerhaave Syndrome, Spontaneous pneumothorax, Empyema, Oesophageal-pleural fistula.

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INTRODUCTION

Dutch physician Herman Boerhaave was first described in 1724 (1,3). This rare spontaneous longitudinal transmural rupture of the esophagus (most commonly occurs over left postero-lateral wall of the esophagus) develops during or after persistent vomiting due to a sudden increase of intraluminal pressure in the esophagus (incidence rate of approximately 3.1 per 100,000 population)(2). It is the most serious and rapidly lethal perforation of the gastrointestinal tract. The diagnosis can be difficult because few patients present with the classic Mackler's triad of vomiting, chest pain, and subcutaneous emphysema (3). These have lead to a delay in diagnosis and may lead to sepsis or multi-organ failure. There are several case reports emphasizing on identification of the key findings on chest radiographs to resolve this problem in which tension pneumothorax and rapidly developing pleural effusion were among the two most convincing radiographic clues in diagnosing Boerhaave syndrome (3). We report a case of Boerhaave syndrome presenting as spontaneous left pneumothorax which was complicated with pleural empyema.

CASE REPORT

A 49-year-old gentleman presented with severe epigastric pain for one day duration associated with one episode of vomiting. He subsequently experienced dyspnoea with no chest pain. His past medical history was unremarkable. Respiratory examination showed reduced breath sound over his left lower zone. Chest radiograph showed a left hydropneumothorax and subsequently a left chest drain was inserted. Full Blood Count revealed his white cell count of 20,000/mm^3 (Table I). There were brownish sediments draining from his left chest drain. His left pleural fluid biochemistry results showed an exudative picture according to Light's criteria while the centrifugation revealed no clearing of left pleural fluid (Table II). His condition deteriorated inpatient requiring mechanical intubation at day one of admission. The first contrast enhanced computed tomography (CECT) thorax showed bilateral pleural effusion with collapsed consolidation (more prominent on the left than the right side) with air pockets in his left pleural effusion. There was minimal residual left apical pneumothorax and presence of pneumomediastinum. He was given intravenous antibiotics of Co-Amoxiclav and oral Azithromycin which was subsequently escalated to intravenous Piperacillin-Tazobactam for total of six weeks in view of his left pleural fluid culture growth of Pseudomonas aeruginosa. He was extubated

Table I: Blood Investigations

Blood Investigations	Day 1	Day 14
Full Blood count		
White Cell Count	20,000/mm^3	12,900/mm^3
Haemoglobin	15g/L	12.2g/L
Platelets	300,000/mm^3	482,000/mm^3
Blood Culture	No growth	
Inflammatory markers		
Erythrocyte sedimentation rate (ESR)	25mm/Hour	10 mm/Hour
C- Reactive Protein (CRP)	310 mg/L	50 mg/L
Renal Profile		
Urea	5.1 mmol/L	5.1 mmol/L
Sodium	135 mmol/L	134mmol/L
Potassium	3.5 mmol/L	3.5 mmol/L
Creatinine	104 umol/L	74.4umol/L
Liver Function Test		
Total protein	73 g/L	82 g/L
Albumin	28 g/L	28 g/L
Globulin	45 g/L	54 g/L
Total bilirubin	13.5 umol/L	11.7 umol/L
Alkaline phosphatase (ALP)	147 U/L	149 U/L
Amylase	63 U/L	46 U/L
Aspartate transaminase (AST)	27 U/L	41 U/L
Alanine Transaminase (ALT)	32 U/L	190 U/L
Lactate dehydrogenase (LDH)	229 U/L	82 g/L

Table II: Left Pleural Fluid Investigation

Microbiology	
FEME	
Epithelial cell	1+
Gram positive Cocci	NIL
Gram negative cocci	NIL
Gram negative bacilli	NIL
Gram positive bacilli	NIL
Pus cell	3+
Red Blood Cell	1+
Differential Count	
Polymorph	500cells/uL
Lymphocyte	380cells/uL
Appearance	Turbid
Organism	NIL
Culture	Pseudomonas aeruginosa
Acid Fast Bacilli	Negative
Biochemistry	
Random Blood Sugar	0.6mmol/L
Chloride	47.4 mmol/L
Lactate dehydrogenase	4051.5 U/L
Total protein	17.6 g/L
albumin	8.3 g/L
pH	6
Triglyceride	0.17 mmol/L

on day four of admission. After the establishment of oral feeding, we noticed food material draining from his left chest tube during feeding. Another urgent contrast enhanced computed tomography (CECT) thorax showed a left oesophageal-pleural fistula with possible broncho-oesophageal fistula and an unchanged left pleural effusion with progressive collapse consolidation. An oesophagogastroduodenoscopy (OGDS) revealed blood clots with air bubbles from left anterolateral side of lower esophagus (30 cm from incisura) and air bubbles in his left chest tube under-water seal during air-insufflation of the esophagus. The revised diagnosis was Boerhaave syndrome. He was treated conservatively with the insertion of a silicone esophageal stent to cover the perforation by the upper gastrointestinal surgical team. The esophageal stent was removed after two months when the perforation had healed by itself. Subsequently, the thoracic surgeon performed a left lung decortication via video assisted thoracoscopic surgery for his left empyema. He improved and was discharged well.

DISCUSSION

Mackler's triad of vomiting, chest pain, and subcutaneous emphysema are the classical presentations of Boerhaave syndrome (3). However, recent published case reports of Boerhaave syndrome suggest that there is increased incidence of presentation of hydropneumothorax and rapidly evolving pleural effusion together with gastrointestinal symptoms (3). Our clinical case here presented with a mixture of respiratory and gastrointestinal complaints on arrival i.e. epigastric pain, vomiting and dyspnoea. The Chest radiograph on arrival showed a left hydropneumothorax. However, many attending doctors may miss the hydrothorax or pleural effusion in this radiograph and only focused on the pneumothorax finding. The presence of brownish sediments (likely food material) in the chest drainage and the pleural fluid biochemistry of an exudative picture should have given us a clue that this is not just a simple parapneumonic effusion. Another key finding of gastrointestinal tract perforation is the presence of pneumomediastinum on the first CECT Thorax (Fig. 1). This was further suspected when we noticed coffee draining from his chest tube few seconds after coffee ingestion. Therefore, another urgent CECT Thorax was ordered to look for any oesophageal –pleural fistula and indeed there was fistulous connection in evidenced by leakage of oral contrast from the oesophagus into the left pleural space (Fig. 2). The diagnosis was confirmed by OGDS which revealed air bubbles in chest tube following air-insufflation of the oesophagus. OGDS is the gold standard in identifying the location of oesophageal defect (4). This diagnosis has explained the continuous large amount of chest tube drainage even after one week of chest tube insertion due to the extravasation of food particles or bile from oesophageal lumen into the pleural space (5). Thoracotomy and aggressive lavage

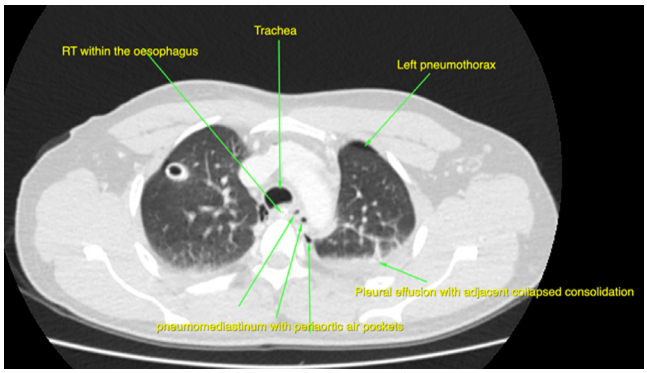

Figure 1: First CECT Thorax lung window. Presence of pneumomediastinum and bilateral pleural effusion with adjacent collapsed consolidation. Residual left apical pneumothorax is also seen.

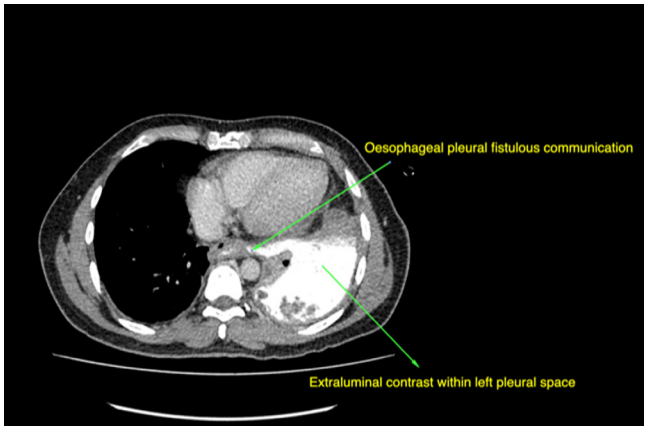

Figure 2: Second CECT Thorax mediastinal window. Demonstration of oesophageal pleural fistulous communication with evidence of extraluminal contrast within pleural space after administration of oral contrast.

and repair of rupture are the preferred treatments for Boarhaave syndrome with empyema. In this case, our upper gastrointestinal team had used a conservative approach for the oesophageal rupture. They inserted an esophageal stent to cover the perforation and allowed the perforation to heal spontaneously. Conservative management is reserved for small or contained ruptures. On the other hand, the cardiothoracic surgeon did left lung decortication via video assisted thoracoscopic surgery for his left empyema. Fortunately, this patient did not present with shock on arrival. He improved and was discharged well despite the diagnosis was not established earlier.

CONCLUSION

Boerhaave syndrome is a rare condition and it poses a great challenge to the emergency and general physician in establishing the diagnosis early in view of the early non-specific signs and symptoms. Hence, clinicians should have a high suspicion level of this diagnosis when a patient presents with a mixture of respiratory and gastrointestinal complaints especially vomiting which is accompanied by hydropneumothorax or empyema. Surgery is the key for most patients while conservative approach may be suitable for some stable patient as shown in this case.

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