Case Report

Spontaneous rupture of oesophagus: Boerhaave syndrome

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Introduction

Boerhaave syndrome consists of spontaneous longitudinal transmural rupture of the oesophagus.[1] In comparison with iatrogenic rupture, which may develop during diagnostic or therapeutic endoscopic procedures, traumas or various oesophageal diseases, spontaneous rupture most commonly develops during or after persistent vomiting, as a consequence of a sudden increase in intraluminal oesophageal pressure. Spontaneous rupture encompasses 15% of all oesophageal ruptures[2] and has the highest mortality & is seen most frequently among patients aged 50-70 years.[1]

The clinical manifestation of depends on the rupture location. In 50% of the cases, it is manifested by Mackler's triad: vomiting, lower thoracic pain and subcutaneous emphysema.[3,4] If the diagnosis is not established in time and if appropriate therapeutic measures are not undertaken, serious complications can develop and this may lead to a poor outcome.

Case report

54 year old male weighing 80 kg admitted to the emergency medical services of hospital with complains of breathlessness, abdominal pain, suffocation and exhaustion. Patient had prior history of alcohol consumption followed by retching and vomiting, breathlessness and unable to lie flat.

On physical examination patient had dull toxic look, tachycardia and dyspneic, oxygen saturation of 91% on room air. His blood pressure was 100/60 mm hg. Auscultation of lungs showed decreased air entry on left side. Abdomen was tense and tenderness in the epigastric region. The liver and spleen were of normal size.

Patient was started on oxygen supplementation and 1000 ml of normal saline was given rapidly. ECG showed sinus tachycardia. Blood gas analysis showed hypoxemia with metabolic acidosis. Chest X-ray done showed pneumomediastinum and left sided pneumothorax with bilateral blunting of cardio phrenic angles and subcutaneous emphysema as well. Immediately left sided intercostal drain was inserted with under water seal .The intercostal drain showed initially 100 cc of gastric fluid drain followed by air column movement. Appropriate antibiotics, analgesic and intravenous pantoprazole were started.

CT scan of abdomen showed presence of pneumomediastinum, periesophageal air collection, hydropneumothorax on left side, intercostal drain in situ. Following oral contrast studies showed presence of leak from the lower oesophagus at the level of D6-D7 vertebral level (Figure 1). Laboratory investigations showed hemoglobin of 80g/dl, leucocytosis of 11,000. Coagulation parameters were within normal limits.

Patient underwent emergency laparoscopic repair of oesophageal tear with omentopexy. Patient tolerated the procedure well. Barium swallows done subsequently showed smooth passage of dye to stomach and duodenum. The patient eventually was discharged from ICU after 3 days and had normal postoperative course with no further complications (Figure 2).

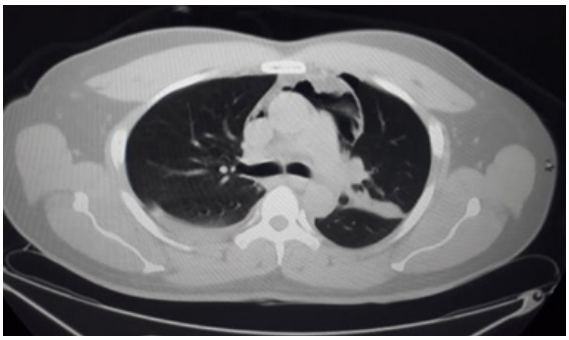

Figure 1 Also mild hepatomegaly with fatty infiltration.

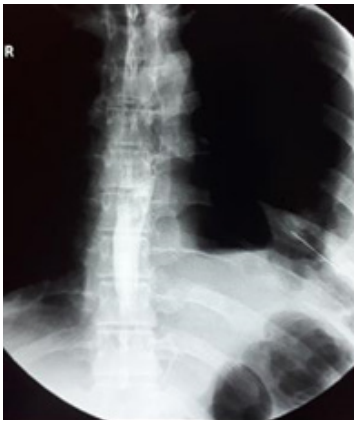

Figure 2 Barium post-surgery.

Discussion

Spontaneous rupture of the oesophagus is a rare clinical entity with a high mortality rate.[5,6] The pathophysiology of Boerhaave syndrome involves a sudden rise in intraluminal oesophageal pressure, thereby forcing the gastric contents against a tight cricopharyngeus muscle.[3,6]

It most often develops during or after intense vomiting caused by excessive eating or drinking alcohol.[7] Perforations are usually longitudinal (0.6-8.9 cm long), with the left side more commonly affected than the right (90%). This is probably due to an anatomical weakness of the left posterolateral aspect of the oesophagus just above the diaphragm. Spontaneous rupture is rare below the diaphragm or in the thoracic part of the esophagus.[3,7]

The clinical manifestation depends on the location of the rupture and the time between its development and examination. Considering that spontaneous rupture most often happens in the distal oesophagus, the majority of patients have Mackler's triad of symptoms and signs: vomiting, lower thoracic pain and subcutaneous emphysema.[3,4] During physical examination of patients, subcutaneous emphysema is observed in 28%-66% within the first 24 hours. Besides typical symptoms, atypical symptoms such as hypotension, tachycardia, tachypnoea, feverishness and cyanosis may also be present.[1,7] Pneumomediastinum (Hamman's sign) is present in around 20% of the cases.[7]

Oesophageal rupture may be followed by serious complications, of which the most important ones are mediastinitis and multiple organ dysfunction. Sepsis may develop within a few hours.[6,7]

Laboratory findings are not specific for diagnosing spontaneous oesophageal rupture. Serum albumin is normal but may be low, while the globulin fraction may be normal or slightly elevated.[7] Radiography of the heart and lungs is valuable for the diagnosis. Radiographs usually show signs of pneumomediastinum or pneumothorax or hydropneumothorax if pleural effusion is concurrent.[3,12] In cases of perforation of the middle third of the oesophagus, pleural effusion is present on the right side, while in cases of rupture of the distal oesophagus, pleural effusion is present on the left side.[5] Diagnostic thoracentesis shows the presence of food remnants, increased amylase and pH below 6. The presence of pneumomediastinum with data including vomiting and chest pain are almost definite signs of Boerhaave syndrome. Overall, 10% of chest radiographs are normal.[7,10]

Oesophagography is an important for confirming the diagnosis and the location of perforation because it shows extravasation of contrast into the pleural space. Thoracic computed tomography imaging is indicated for making the diagnosis in patients who do not tolerate oesophagography which may show localized fluid collection is observed, as well as periesophageal air collection.[1,11]

The treatment for Boerhaave syndrome is both conservative and surgical. The goals of pharmacotherapy are to reduce morbidity and to prevent complications. Surgical management is generally required for both spontaneous rupture and traumatic perforation.[12] Endoscopic stent insertion offers a promising alternative. The mortality rate varies depending on the time that has elapsed since development of the rupture and its recognition and treatment. If treatment is not started within 24 hours from the onset of symptoms, the mortality rate is 25%; after 24 hours, it is 65%; and after 48 hours, it is 75%-89%.[12]

Conclusion

Boerhaave syndrome should be considered in all patients with a combination of gastrointestinal symptoms (epigastric pain and vomiting) and pulmonary symptoms (suffocation), even when all the signs and symptoms (lower thoracic pain and subcutaneous emphysema) of this disease are absent. Early clinical suspicion will lead to timely diagnosis and maximize the survival chances for the patient.

Acknowledgments

None.

Conflicts of interest

None.

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