Boerhaave’s Syndrome

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A 58 year old male was admitted with complain of sudden onset severe central chest pain following a bout of vomiting after lunch. He was having a constricting feeling in the chest and upper abdomen associated with profuse sweating. There was no history of similar episode in the past nor he was having history of hypertension, diabetes mellitus, COPD or ischemic heart disease. He was a non-smoker, non-alcoholic with no history of any known drug allergy. On admission, the patient was dyspnoeic, looking distressed. Respiratory rate was 16/min, pulse 96/min, low volume, regular; blood pressure was 130/80 mm of Hg in the right upper limb in supine position. Auscultation of chest showed bilateral vesicular breath sound with few crepts in left infrascapular region. Cardiovascular and nervous system examination did not reveal any abnormality. There was guarding on per abdominal examination.

Pulse oximetry showed decreased oxygen saturation (SPO₂ 95%) on air. ECG on admission showed RBBB with left axis deviation. A provisional diagnosis of acute coronary syndrome with differential diagnosis of acute pancreatitis, acute cholecystitis, acute severe gastritis, oesophageal rupture (Boerhaave’s syndrome) was kept. Patient was kept on conservative management. Routine blood investigations were sent. Chest X-ray AP View (Figure 1) showed left-sided hydropneumothorax and mediastinal emphysema. Ultrasonography showed moderate

Fig. 1: Chest X-ray showing left-sided hydropneumothorax and mediastinal emphysema

Fig. 2: CT thorax showing pneumomediastinum

References


left-sided pleural effusion with internal echoes along with mild right sided effusion. Patient continued to have chest pain despite conservative symptomatic management and started desaturating, SPO₂ falling to 92% on 10 l/min of oxygen. An intercostal drain was put on the left side with around 600 ml of coffee-coloured fluid with air coming out. Patient was given iv meropenem along with other supportive treatment. A contrast-enhanced CT scan of thorax and abdomen was done which showed air density around the mediastinal structures extending into the subcutaneous plane of the neck, esophageal perforation at D7-D8 level, fluid with residual air in left pleural space with ICD in situ, right-sided pleural effusion (Figures 2, 3, 4). Patient continued deteriorating despite giving inotropic and ventilator support. Another ICD was put on the right side and thoracotomy was planned but unfortunately the patient succumbed to his illness.

Boerhaave first described the spontaneous rupture of the esophagus in 1724. It typically occurs after forceful emesis. Boerhaave syndrome is a transmural perforation of the oesophagus to be distinguished from Mallory-Weiss syndrome, a nontransmural esophageal tear also associated with vomiting. Because it usually is associated with emesis, Boerhaave syndrome usually is not truly spontaneous. However, the term is useful for distinguishing it from iatrogenic perforation, which accounts for 85-90% of cases of esophageal rupture. The most common anatomical location of the tear in Boerhaave syndrome is at the left posterolateral wall of the lower third of the esophagus, 2-3 cm proximal to the gastroesophageal junction, along the longitudinal wall of the esophagus. The second most common site of rupture is in the subdiaphragmatic or upper thoracic area.¹ ²

Prompt recognition of this potentially lethal condition is vital to ensure appropriate treatment. Mediastinitis, sepsis, and shock frequently are seen late in the course of illness.

References