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Boerhaave's Syndrome: A Forgotten Fatality

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Abstract

Transmural tears of the oesophageal wall following sudden forceful vomiting is often associated with complications like hydro-pneumothorax, pneumomediastinum, surgical emphysema, mediastenitis, sepsis, shock and death if emergent surgery is not done. Errors in diagnosis are usually caused by unawareness of its varied and atypical presentations or failure to consider its possibility in acute cardiothoracic and upper gastrointestinal conditions. We report a case where the patient presented with 5 days of persistent vomiting, diagnosed as a case of Boerhaave's syndrome within 12 hours, operated within 24 hours but died on 5th Post Operative Day. The principal cause of fatality was delayed presentation to the tertiary care hospital leading to severe mediastinitis and sepsis.

Keywords: Mediastinitis, Mackler's triad, post-emetic perforation

Case Report

A 46 years old male who had a feast for lunch followed by a heavy dinner presented with persistent vomiting which continued for 5 days, inability to take food orally for 4 days, chest discomfort & difficulty in breathing for 2 days. He was neither an alcoholic, smoker nor a tobacco chewer. Treatment history revealed that he was managed as gastritis by a local physician on 1st day and as pneumonia for next 2 days in secondary health care facility. As symptoms worsened he went back to some country medicine for 2 days before reaching our hospital.

On examination he was conscious, alert, oriented with a GCS of 15/15, having respiratory distress. He had pallor, mild icterus, with temperature 99.7˚F. His pulse was 132/ minute, BP was 110/70 mmHg, and respiratory rate was 34/minute with SpO2 78% at room air. Chest examination revealed trachea deviated to the right and hyper-resonant note over and above left 2nd intercostal space and dull note in the left 3rd intercostals space and below. Auscultation revealed diminished air entry on the left mammary area and absent breath sounds in all other areas over left hemithorax. There were no added sounds. Other systemic examinations were normal.

Investigations revealed a Hemoglobin of 11.7gm%, Total leucocyte count of 21,440 cells/mm^3 with a neutrophil count of 96%, total platelet count of 1.9 lakhs, Liver function tests were normal except for Total Bilirubin 3.7 mg/dl and direct bilirubin 2.1mg/dl. Urine routine examination and renal function test were normal. RBS was 314 mg/dl, HBA1C 8.6%, CRP was 21.95mg/dl; Serum procalcitonin was 5.2 ng/dl (in the range of severe sepsis). ECG was normal.

X-ray chest showed a left hydro pneumothorax with multiple air fluid levels in the mediastinum [figure-1]. CT scan of Chest with oral contrast revealed multiple foci of air in the soft tissues of neck and surrounding trachea suggestive of subcutaneous emphysema [figure-2] [figure-3]. CT chest also showed a perforation of the left lateral wall of oesophagus in the distal 1/3rd with extravasation of oral contrast from the site of tear and pooling in the collections in the mediastinum [figure-4].

A diagnosis of Boerhaave syndrome was made within 10 hours of admission and patient was kept nil per orally and an inter-costal drainage was done. Gastrosurgeon and CTVS surgeon operated within 24 hrs of admission. Open trans-thoracic oesophagectomy with cervical oesophagogastrostomy was done. Patient was shifted to ICU post procedure and was on ventilator and continued with broad spectrum antibiotics as the patient was in septicemia with acute mediastenitis. Post-operative chest x-ray PA view showed resolution with no mediastinal air fluid levels [Figure-5].

By 2nd post-operative day (POD), with patient not recovering from septicemia and in view of falling blood pressure, vasopressor drugs were started and titrated accordingly and continued on ventilator. By POD 5, the patient developed persistent hypotension, cardiopulmonary arrest and could not be revived.

Discussion

Boerhaave's syndrome, also called "barogenic perforation" and "postemetic perforation" was first described by a Dutch physician, Hermann Boerhaave in 1724.[1] Other causes of sudden rise in intra-oesophageal pressure such as weight lifting, laughing, hyperemesis gravidarum, and seizures can also result in spontaneous oesophageal rupture. Boerhaave's syndrome classically presents with a background of alcoholism or dietary excess. Mackler's triad of vomiting, lower thoracic pain and subcutaneous emphysema with chest X-ray findings of pneumomediastinum and hydropneumothorax are pathognomonic.[2]. Males are affected more often than females in variable ratios in different reviews. The 40 to 60 years age group has the highest incidence.

First successful closure was done by Barrett in 1947, more than 200 years after the original description[3]. Even after a good surgery, mortality remained very high due to serious cardio-respiratory embarrassment with shock-like condition and sepsis due to fulminating mediastinitis secondary to accumulation of corrosive gastric juices, enzymes, food and bacteria [4]. The mediastinal pleura ruptures with the initial insult or it is digested at a later stage by gastric acid and ezymes which are then drawn into the pleural space by the negative intra-thoracic pressure [5]. The length of the tear varies from 0.6 to 8.9 cm, the average being 2.24 cm. The tear in the mucosa is usually longer than the muscle tear [6].

Pain abdomen or chest pain was the complaint in all cases whereas only 3 out of 14 patients complained of dyspnoea in a study by Walker et al [7]. Subcutaneous emphysema varies from 28% to 66% in various reports. Pate et al [8] in their series found that the initial chest roentgenogram was abnormal in 97% of the patients but was interpreted as compatible with perforation of the oesophagus in only 27%. If pneumomediastinum is present, mediastinal

cracking coincident with each heart beat known as Hamman's crunch may be heard in 20% of cases with a stethoscope and can be mistaken for a friction rub of pericarditis.[9]

The differential diagnosis of Boerhaave's syndrome includes Aspiration pneumonia, Dissecting aneurysm, Appendicitis, Oesophagitis, Lung abscess, Mesenteric thrombosis, Myocardial infarction, Pancreatitis, Pneumonia, Pneumoperitoneum, Pneumothorax, Pulmonary embolism, Ruptured subphrenic abscess, Oesophageal or gastric tear, Splenic bleeding, Diaphragmatic hernia, Pneumomediastinum [10]. A delay of 12 hours or more between symptom onset and surgery is associated with a 36% mortality while a delay of 24 hours increases the mortality to 64% [10]. Conservative management without surgery leads to 100% mortality within 7 days.

Conclusion

Spontaneous rupture of the oesophagus is a well documented surgical condition which is always a diagnostic and therapeutic challenge. Treatment becomes complicated when diagnosis is delayed. Sepsis and pulmonary complications contribute substantially to poor outcome. Elective mechanical ventilation in modern intensive care units have improved the outcome. Aim of reporting this case is to make the emergency room residents suspect this condition at the earliest, establish diagnosis as quickly as possible and take action for urgent surgical intervention which would help in reducing mortality.

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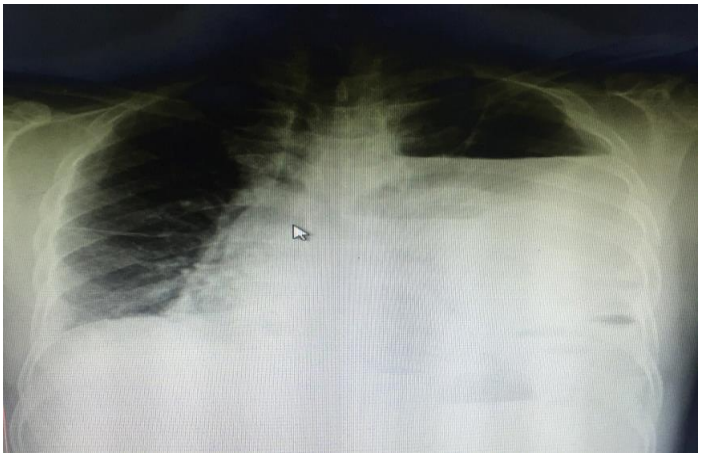

Figure 1: X-Ray Chest PA view showing a left hydro-pneumothorax with multiple air fluid levels in the mediastinum

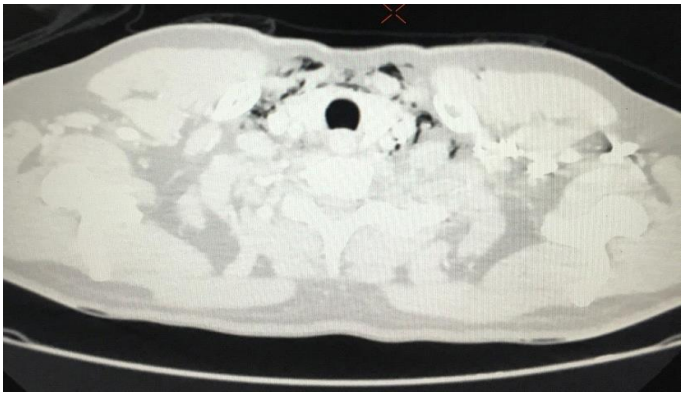

Figure 2 – CT Scan of Thorax showing multiple foci of air in the soft tissues of neck and surrounding trachea suggestive of subcutaneous emphysema

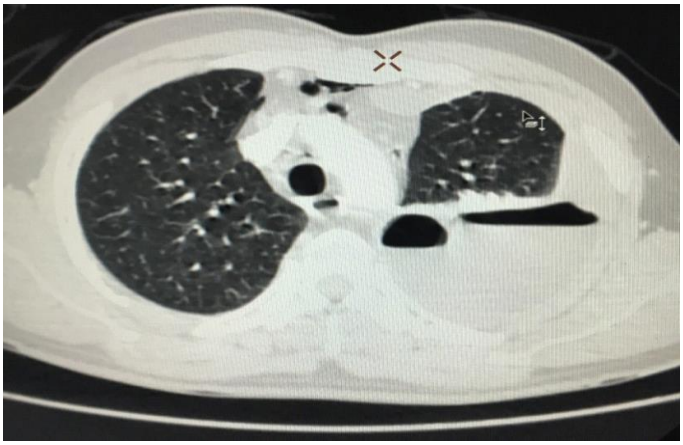

Figure 3 CT scan showin multiple curvilinear foci of air collection in the mediastinum with few of the air pockets showing air fluid levels predominantly in peri-oesophageal location

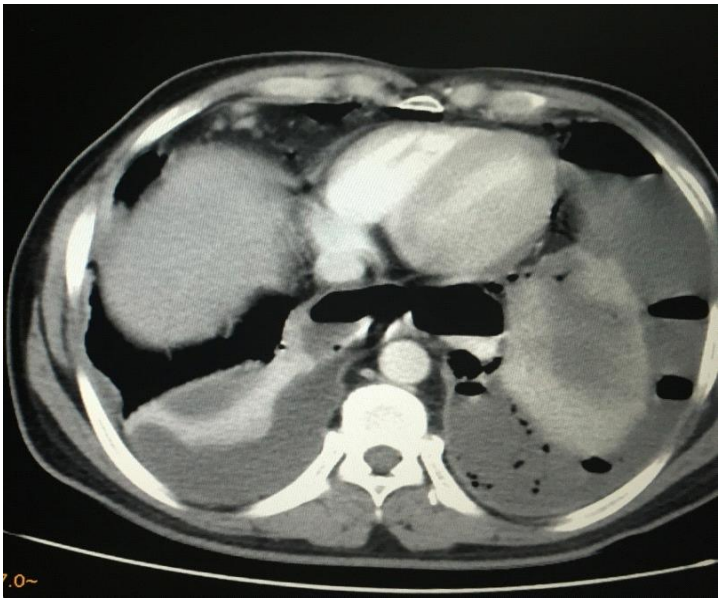

Figure 4- CT scan of thorax with oral contrast shows a perforation of the left lateral wall of oesophagus in the distal 1/3rd with extravasation of oral contrast from the site of tear and pooling in the collections in the mediastinum.

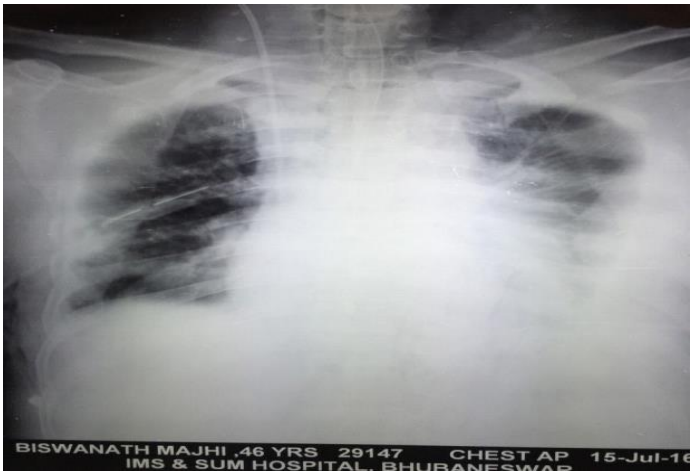

Figure 5- Post op Chest X ray PA view showing resolution with no mediastinal air fluid levels