Boerhaave's syndrome: a case report.

**KEYWORDS:** Boerhaave's syndrome, chest drainage, enterostomy, total parenteral nutrition.

**ABSTRACT**
Boerhaave's syndrome or spontaneous oesophageal perforation, is a potentially lethal and frequently elusive medical condition which presents not only a diagnostic but also a therapeutic challenge. It is insufficiently considered in diagnostic hypotheses, yet may be confirmed or excluded by simple methods such as an erect chest film and a contrast study of the oesophagus. 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. Early aggressive surgical intervention in the form of open and wide mediastinal and chest drainage, with or without oesophageal repair, resection or exclusion, offers the patient the best chance of survival against this otherwise invariably fatal event. Nonoperative therapy consisting of antibiotics, nil oral regimen, nasogastric tube suction, pleural drainage, H2 receptor blockers and either a feeding enterostomy or total parenteral nutrition, may also be appropriate in selected patients. It is probable that the condition is more common than is generally supposed. All clinicians need to be aware of this lethal disease, its frequently unusual presentations and the importance of early diagnosis.

**Introduction**
Boerhaave's syndrome is rupture of the oesophageal wall. 56% of oesophageal perforations are iatrogenic, usually due to medical instrumentation such as an endoscopy or paraoesophageal surgery. In contrast, the term Boerhaave's syndrome is reserved for the 10% of oesophageal perforations which occur due to vomiting. Spontaneous perforation of the oesophagus most commonly results from a sudden increase in intrathoracic pressure combined with relatively negative intrathoracic pressure caused by straining or vomiting (effort rupture of the oesophagus or Boerhaave's syndrome). Other causes of spontaneous perforation include caustic ingestion, pill oesophagitis, Barrett's oesophagus, infectious ulcers in patients with AIDS, and following dilation of oesophageal strictures.

In most cases of Boerhaave's syndrome, the tear occurs at the left postero-lateral aspect of the distal oesophagus and extends for several centimeters. The condition is associated with high morbidity and mortality and is fatal in the absence of therapy. The occasionally nonspecific nature of the symptoms may contribute to a delay in diagnosis and a poor outcome. Spontaneous effort rupture of the cervical oesophagus, leading to localized cervical perforation, may be more common than previously recognized and has a generally benign course. Preexisting oesophageal disease is not a prerequisite for esophageal perforation but it contributes to increased mortality. This condition was first documented by the 18th-century physician Herman Boerhaave, after whom it is named.

**Case Details**
A 18 year male presented to casualty with chief complaints of acute onset of breathlessness and chest pain following a bout of alcohol. He was transferred from primary centre with preliminary treatment of ICD insertion.

**fig 1:** CXR showing pneumothorax with subcutaneous emphysema.

**fig-2:** CXR post ICD insertion.

On presentation, the patient was in mild respiratory distress with a respiratory rate of 30 breaths/min, a heart rate of 90 beats/min, and an oxygen saturation measured via pulse oximetry of 93% while breathing room air. A respiratory examination revealed the left-sided findings of reduced chest expansion, hyper resonance to percussion, and reduced air entry. Patient was referred from primary centre with preliminary treatment of ICD insertion. A chest radiogram revealed large left hydro pneumothorax left with subcutaneous emphysema in upper thorax, total collapse of left lung, surgical emphysema in neck, mediastinum shift to right, Right lung normal. The ICD was draining serous fluid with no air column movement. Hence a contrast tomography was performed which showed ICD insertion through the 8th intercostal space, peritoneum, diaphragm into
left pleural space. Followed by which an ICD was inserted in left 6th intercostal space. Post ICD insertion radiogram showed expansion of left lung with clear left costo-phrenic angle. Cardiac shadows and mediastinum appears normal and blunting of right costo-phrenic angle.

Patient was stabilized and an Oesophagagastroscopy was performed which showed full thickness mallory weiss tear extending 1 inch proximally from cardioesophageal junction with devitalised mucosa and margins with gap of 1 mm with no active bleed from the tear. Patient was then managed by NPO, Byles Tube insertion Gastrostomy and feeding jejunostomy. During the course a right ICD was inserted for right pleural effusion.

Methylene Blue dye (diluted) was given per orally to determine the patency of oesophagus at regular intervals which showed dye in left under water seal bag which eventually stopped. Confirmation in form of gastrograffin studies were done which showed normal patency of oesophagus. Patient tolerated per orally with removal of gastrostomy and jejunostomy.

After some days patient developed signs of intestinal obstruction for which diagnostic laproscopy was performed which showed adhesion between anterior abdominal wall and loop of jejunum resulting in kinking of the jejunal loop. Adhesion band was cut. Postoperatively the patient made excellent progress and was weaned to a normal diet over a period of a month.

Discussion
The Spontaneous oesophageal rupture was originally described by Herman Boerhaave in 1724. It typically occurs in men aged 30-40 years after over indulgence in alcohol or food; but may result from any emetogenic pathology. It must be distinguished from the more common traumatic oesophageal rupture and pathological oesophageal rupture. In Boerhaave's syndrome, the oesophagus undergoes barotrauma when increased intra-gastric pressure is transmitted to the oesophagus against a closed glottis. The classical presentation is Mackler's triad, which comprises thoraco-abdominal pain, vomiting and surgical emphysema. Diagnosis of Boerhaave's syndrome can often be a clinical challenge. The single most important factor is a high index of clinical suspicion. Blood investigations are of limited diagnostic use. A convincing history, suggestive clinical findings and radiological imaging were sufficient to diagnose Boerhaave in this case.

fig 3: Gastrograffin Study

fig 4: Diagnostic laproscopy finding-adhesion

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