Case report

A 40 year old man arrived to Emergency Department by ambulance with severe epigastric pain and shortness of breath. The pain began suddenly 14 hours before presentation to the hospital, after one single episode of vomiting. The pain was severe, sharp, and constant without any radiation. The pain was worsened by movement and was partially relieved by sitting up. Associated symptoms were shortness of breath and difficulty swallowing.

Five hours after the onset of symptoms the patient developed notable swelling of the neck and face.

The past medical history was significant for a longstanding lasting GORD (gastro-oesophageal reflux). The patient was not on any regular medications. He was a heavy smoker (20 pack-years) and heavy drinker. (Revista de Medicinã de Urgenþã, Vol. 4, Nr. 1: 13-15)

Physical Examination

The patient’s vital signs were temperature 37,8°C, respirations 33 minute, pulse 120 beats per minute, and blood pressure 90/60 mm Hg. His SpO2 was 90% on 15 L/minute of oxygen. On inspection the patient was severely ill-appearing, sitting leaning forward with a grey bluish discoloration to his torso and face. He was conscious, in obvious distress, and unable to complete full sentences.

Physical examination was significant for severe respiratory distress with obvious accessory muscle. Central cyanosis was noted as well as moderate bilateral cervical swelling and swelling over the left maxilla. On palpation, subcutaneous emphysema was present from the base of the neck to his left maxilla with no tracheal deviation. On percussion there was stony dullness in both lung bases. Auscultation revealed decreased breath sounds and coarse crackles in the right and left mid lung fields. A pleural rub was noted bilaterally.

Cardiovascular examination was notable only for tachycardia. Examination of the abdomen revealed diffuse rigidity abdomen with severe tenderness in the epigastrium. The patient refused digital rectal examination.

Investigations

An initial upright chest x-ray showed an enlarged mediastinum with bilateral pleural effusions. Subcutaneous emphysema was readily noted. Subsequent upper gastrointestinal investigation with a water-soluble contrast revealed oesophageal rupture typical of Boerhaave’s syndrome.

Fig. 1. Chest x-ray on presentation showing pneumomediastinum and bilateral pleural effusions

Fig. 2. Fluoroscopy – water soluble contrast study showing typical distal left lateral leak

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Management

The patient was managed in the resuscitation area with continuous cardiac, respiratory, and blood pressure monitoring. High flow O₂ via face-mask with reservoir was provided and two large-bore intravenous lines were placed. One litre of Hartmann’s solution was infused as a bolus and additional fluids given as needed. Analgesia in the form of intravenous morphine was provided. Empiric treatment with two intravenous antibiotics was started as per hospital protocol in patients with suspected sepsis.

The patient was transferred to the operating theatre after initial resuscitation.

Surgery involved radical mediastinal debridement via bilateral thoracotomies. There was extensive soft tissue necrosis of the mediastional tissues bilaterally. The debridement resulted in skeletonisation of the thoracic oesophagus on the right, with preservation of the short oesophageal vessels and excision of all mediastinal pleura to expose the full length of the trachea. On the left, the thoracic aorta was exposed and cleaned from the descending arch to the diaphragm. The oesophageal perforation was identified low on the left. The margins of the perforation were necrotic and were excised. A T-tube was placed in the perforation with closure of the oesophagus around the tube. Bilateral mediastinal and pleural drains were inserted. Subsequent management while the patient was admitted to the Intensive Therapy Unit (ITU) included drainage under CT guidance and subsequent repositioning of drains in the operating theatre. After two months in ITU there was no leak around the T-tube and it was removed. The tract closed quickly thereafter. The patient survived and he was discharged home.

Boerhaave’s Syndrome – literature review

Introduction

The syndrome first described by Herman Boerhaave in 1723 involved a patient, Baron Jan von Wassenaer, Grand Admiral of the Dutch Fleet and Prefect of Rhineland, who vomited after a duck meal, subsequently developed left-sided chest pain, and died 18 hours later. At autopsy, a tear of the left posterior wall of the oesophagus five cm above the diaphragm was discovered after the meal was observed in the left pleural space. Today the syndrome is typically seen in middle-aged alcoholic men but can occur in any patient with severe vomiting.

The cause of rupture is an acute pressure rise in the esophagus from forceful vomiting against a closed glottis. In most cases the rupture occurs at the left aspect of the gastro-oesophageal junction – specifically the left posterolateral wall – with leakage of the acidic gastric contents into the mediastinum and pleural spaces (left pleural space if a
lower oesophageal rupture occurs and right pleura if the rupture is more proximal).

**Diagnosis**

The diagnosis should be suspected in anyone with severe lower chest pain and dyspnoea following vomiting. Associated findings include surgical emphysema due to pneumo-mediastinum and sepsis induced by the subsequent mediastinitis with a sepsis-type picture.

Mackler’s triad consists of chest pain, dyspnoea and subcutaneous emphysema [1]. Chest films frequently show pneumo-mediastinum, pleural effusions, and pneumothoraces. Water soluble contrast studies may confirm the diagnosis and localize the site of rupture. If negative, and the suspicion of Boerhaave’s syndrome is high, endoscopy may prove diagnostic [2].

**Management**

There are two possible treatment approaches to oesophageal perforations due to either spontaneous (Boerhaave’s) or post-traumatic (including following instrumentation) perforation.

Intervention with oesophageal repair has its advocates. There are reports of very good results with primary closure even in late presentations [3, 4]. Some authors advocating primary closure reserve a more conservative approach for those cases where major surgery itself or general anaesthetic are severe risks due to previous debilitating conditions or poor clinical status [2]. Others suggest oesophageal resection as a valid option in those with pre-existing oesophageal pathology with excellent survival rates [5].

Supporters of a more conservative approach report good outcomes by aggressively treating sepsis and any existing or subsequent leaks with pleural drainage rather than with major surgery [6]. There are also reports that suggest a conservative approach in children with oesophageal perforation (none of those involved actually having Boerhaave’s syndrome) has excellent results compared with the adult population [7].

Both approaches use drainage of pleural/mediastinal spaces, T-tubes, draining gastrostomy, and mini-toracotomies. CT and contrast studies are essential for follow up monitoring of healing and also to identify and treat fluid collections.

Management depends upon surgical experience and preference, the type of perforation, and the presence of associated sepsis, effusions, premorbid conditions, patient age, and the time of presentation after the onset of symptoms.

**Conclusions**

Boerhaave’s syndrome is a major emergency, which can present in a variety of ways. In this case, the patient was septic, in respiratory failure and complaining of epigastric pain.

Boerhaave’s syndrome should be suspected in acutely ill patients who present after even minor episodes of vomiting and complain of dyspnoea, lower chest pain, or upper abdominal pain. Surgical emphysema is an invaluable sign which may guide diagnosis.

As with any major emergency, Boerhaave’s syndrome requires prompt recognition and treatment. Major surgery with primary closure or esophagectomy followed by reconstruction, as well as possible conservative approaches should be promptly discussed with the responsible upper GI surgeon.

**References**