EMERGENCY CASEBOOK

Walked in with Boerhaave's...

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Boerhaave's syndrome is a transmural rupture of the oesophagus. It is a rarer, and less well described complication of forceful emesis. The more common complication being a non-transmural Mallory-Weiss tear. Boerhaave's is the most lethal perforation of the GI tract and has a mortality rate between 10 and 50%.

It most commonly occurs after indulgence in food or alcohol, particularly in males aged 50–70 years. The well described presentation is of a middle aged man with a sudden onset of severe chest pain in the lower thorax/upper abdomen following repeated retching or vomiting induced by excessive dietary and alcohol intake. However, atypical presentations are common. Presented here is the case of a 26-year-old man who attended accident and emergency department complaining of chest pain. Initial examination was normal. He was subsequently diagnosed with Boerhaave's syndrome. This case highlights the varied presentation of this potentially fatal condition.

A 26-year-old man went out with friends and drank two pints of Guinness. On returning home, he started to retch and after about 10 min vomited once. After 2 days, he presented to the accident and emergency department complaining of left-sided chest pain that had started shortly after the vomiting. The pain was mild and was described more as a discomfort, worse on movement, inspiration and coughing. It was relieved by a muscle rub. There was no cardiac nature to the pain, and he had no cardiac risk factors or risk factors for a pulmonary embolism.

His only medical history was of a traumatic subarachnoid haemorrhage 2 years previously, which had caused him to spend 3 months in hospital, and resulted in memory problems and a facial nerve palsy. He was taking tegretol, carbamazepine and phenytoin, although epilepsy was not confirmed. He also

had had some swallowing difficulties following temporary tracheostomy. However, subsequent barium meal was normal. Social history was unremarkable. His maximum alcohol intake per week was 8 units, with a maximum of 2 pints per night.

On examination, he was found to be alert and orientated. He was haemodynamically stable, with a blood pressure of 104/63 mm Hg and a heart rate of 80/min. Oxygen saturation was 99% on air, with a respiratory rate of 24/min. Jugular venous pressure was not increased, and auscultation of the heart revealed normal heart sounds with no murmurs. Auscultation of the chest revealed no abnormality. Abdominal examination was unremarkable. ECG was in sinus rhythm.

Initial impression was musculoskeletal pain, with a differential of left-sided aspiration pneumonia. Chest x ray (CXR) showed a small left-sided pleural effusion, with a small pneumothorax. Mediastinal air adjacent to the aortic arch and Boerhaave's syndrome were suspected.

CT confirmed the presence of mediastinal air on the medial aspect of the upper oesophagus, as well as the left-sided pneumothorax and pleural effusion. This was highly suggestive of a ruptured oesophagus (fig 1). Although gastrograffin contrast study failed to confirm an obvious leak, clinical suspicion of Boerhaave's syndrome remained high, and the patient was referred to the surgical assessment unit and subsequently to the upper gastrointestinal surgical team. A chest drain was inserted, and the patient was managed conservatively with intravenous antibiotics and kept nil by mouth. He was discharged home 9 days later.

Boerhaave's syndrome is a transmural rupture of the oesophagus. It is a rare and less well described complication of forceful emesis, the more common complication being a non-transmural Mallory-Weiss tear.

Boerhaave's syndrome is the most lethal perforation of the gastrointestinal tract.¹ It has a mortality rate between 10% and 50%. This is a reflection of diagnostic difficulties, delays in presentation, and complications such as mediastinitis, sepsis and shock.

The pathophysiology is thought to result from neuromuscular incoordination and a failure of the cricopharyngeal muscle to relax during vomiting, thus causing a sudden rise in intraluminal oesophageal pressure.¹ It occurs most commonly after indulgence in food or alcohol, particularly in men aged 50–70 years. The well-described presentation is of a middle-aged man with a sudden onset of severe chest pain in the lower thorax/upper abdomen after repeated retching or vomiting induced by excessive dietary and alcohol intake. Indeed, Boerhaave himself first reported the syndrome in 1724 on Baron Wassenaar, Grand Admiral of the Dutch Fleet, a glutton who practised the Roman habit of autoemesis.² The Mackler triad describes the classic presentation of vomiting, lower thoracic pain and subcutaneous emphysema.³ The pain may radiate to the back, and may be aggravated on swallowing. However, atypical presentations occur in about a third of patients. A retrospective analysis of 34 patients found pain and

Figure 1 CT showing the presence of mediastinal air on the medial aspect of the upper oesophagus, as well as the left-sided pneumothorax and pleural effusion, suggesting a ruptured oesophagus.

Abbreviation: CXR, chest x ray

vomiting to be the only common historical events suggesting the diagnosis.⁴

An abnormality will be shown in 90% of CXRs, the most common finding being left-sided effusion, as most tears are at the left posterolateral wall of the lower oesophagus, 2–3 cm proximal to the gastro-oesophageal junction, and pneumothorax. The sensitivity of gastrograffin swallow is reported to be between 75% and 95%.¹ Barium oesophagography and non-contrast CT are more sensitive. Management involves a combination of conservative—intravenous fluid resuscitation and broad-spectrum antibiotics, nasogastric suction—and surgical interventions. The approach used depends on time delay to presentation, extent of perforation and overall medical condition of the patient. Diagnosis and surgery within 24 h gives a 75% survival rate.¹

Over 1000 cases of Boerhaave's syndrome have been reported in the literature, including many cases occurring after post-op vomiting, and several cases associated with peptic ulcer disease. Other atypical presentations include those during childbirth, weight lifting and asthma attacks. In all case reports, studied patients presented either acutely unwell, with severe pain, vomiting or shortness of breath or they had evidence of an acute illness by way of a pyrexia or haemodynamic instability. No report was found of a patient presenting as the patient was in our case. Few occurred after single episodes of vomiting.

This case highlights the importance of the clinical history in a patient presenting with chest pain, and the importance of a

CXR to back up clinical examination. Those working in the accident and emergency department should always consider the possibility of Boerhaave's syndrome in patients presenting with chest pain after vomiting, as this potentially fatal condition can present in such a variety of ways.

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