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Spontaneous oesophageal rupture

Introduction

Oesophageal rupture is a serious and life-threatening condition. It usually presents to non-specialist medical and surgical staff through the emergency department. The clinical features are non-specific and can be easily confused with more common conditions. However, once considered the diagnosis is easily made. Untreated, the condition is rapidly fatal and delay in diagnosis rapidly adds to problems of treatment, and is associated with escalating morbidity and mortality risk. This article will focus on the presentation, investigation and initial management of 'spontaneous' rupture of the oesophagus. The management options that are available in specialist centres will then be considered in brief.

Nomenclature

'Boerhaave's syndrome' and 'spontaneous perforation' have been used to describe oesophageal rupture that commonly follows emesis. 'Barogenic rupture' is probably a better term as emesis is not the only described cause of rupture resulting from raised intra-oesophageal pressure.

Barogenic rupture

Historical note

At midnight on 29 October 1723 the Professor of Medicine at Leyden University, Hermann Boerhaave, attended Baron Jan von Wassener, the High Admiral of the Dutch Fleet. He reported the events the next year (Boerhaave, 1724). Following a large meal of roast duck and beer the Admiral had taken olive oil as an emetic. On vomiting he immediately experienced severe epigastric and chest pain, complaining to his servants that his stomach was torn. Examination of the abdomen revealed no abnormalities but the Admiral died within 18 hours. At post-mortem examination there was emphysema of the chest and abdominal wall, a tear in the left posterolateral wall of the oesophagus 3 inches above the diaphragm and gastric contents in the left pleural cavity.

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Causes

Jones and Ginsberg (1992), in their review of 511 reported cases of perforated oesophagus, found 16% were of barogenic origin, 43% had been caused by upper gastrointestinal endoscopy, 19% were the result of trauma, and the rest resulted from the ingestion of foreign bodies, were caused by surgical injury or were associated with tumours. Ruptured oesophagus has been reported following other causes of barotrauma such as defecation, child birth, blunt trauma to chest or abdomen, and epileptic seizures. Cadaveric studies suggest an intraluminal pressure of 5 pounds per square inch is necessary to tear an otherwise normal oesophagus (Mackler, 1952).

During emesis, as the intra-abdominal pressure rises because of the descent of the diaphragm and contraction of the anterior abdominal wall, the oesophageal sphincters relax to allow egress of intestinal contents. Failure of the upper oesophageal sphincter to relax or a spasm lower in the oesophagus, made more likely by sedation, alcohol ingestion, repeated vomiting or oesophageal disease, causes a rise in intraluminal oesophageal pressure and rupture can occur.

Pathophysiology

Barogenic rupture usually results in a longitudinal tear in the left posterolateral wall of the oesophagus just above the diaphragm. The left side is torn in 80% of cases and it is believed to be a result of weakness where there is splaying of the muscle fibres of the oesophageal wall at the entry point of blood vessels. There is no serosal covering of the intrathoracic oesophagus and following perforation the mediastinum is flooded with oral flora and gastric contents, causing a mixed microbial and chemical mediastinitis which leads to haemorrhagic necrosis.

If the overlying parietal pleura has not also been ruptured by the explosive force of the vomiting it may temporarily contain the contamination. Eventually, however, this membrane does rupture allowing

pleural contamination. The patient develops septic shock, arrhythmias and, without intervention, death quickly follows.

Clinical

History

The dominant symptom is tearing pain, usually retrosternal, sometimes epigastric and even hypochondrial. This may be experienced over the front or back of the chest or abdomen, and can radiate as high as the shoulder. The onset of pain immediately follows the barometric injury in half of the patients (Michel et al, 1981). It is this temporal association, pain that was not present before vomiting but occurred immediately after vomiting, that should raise a suspicion of this injury. Pleural effusion may be associated with dyspnoea.

The patient will be sweaty and distressed with symptoms that are disproportionately severe compared with the clinical signs. Haematemesis is rare. The differential diagnosis includes perforated peptic ulcer, pancreatitis, dissecting aortic aneurysm and myocardial infarction. Diagnosis is difficult and in Michel et al's (1981) series 15% of perforations were first diagnosed at post mortem.

Examination

Pyrexia develops within a few hours (Sawyers et al, 1975). Initially there may only be mild epigastric tenderness. Subcutaneous emphysema can be detected over the chest or in the neck in a fifth of patients (Sawyers et al, 1975). This important sign should be checked routinely as it is pathognomonic of this injury. With delay, mediastinitis develops and the patient becomes increasingly cardiovascularly unstable with arrhythmias and septic shock.

Investigations

Leucocytosis develops rapidly (Michel et al, 1981). Plain radiology is initially normal in up to a tenth of cases (Han et al, 1985). Chest X-ray may show a widened mediastinum; cervical or mediastinal air (41%); a pleural effusion (60%) (*Figures 1* and *2*) or pneumothorax (26%) (Michel et al, 1981). In practice it is rare for the radiograph to be entirely normal. However, if a careful history leaves one with a high index of suspicion further investigation is warranted.

Diagnosis is confirmed in most cases by a contrast swallow using a water-soluble contrast agent such as Gastromiro (Bracco UK, High Wycombe) (*Figure 3*) (Sawyers et al, 1975; Michel et al, 1981; Gouge et al, 1989). This is preferable to Gastrografin (Schering AG, Berlin, Germany) which can cause severe pneumonitis if aspirated into the airways. If the water-soluble swallow is not diagnostic one should not hesitate to repeat the study using thin barium. This provides better contrast, and can outline the rupture in greater detail but can be associated with a granulomatous reaction if debridement of the mediastinal contamination is delayed. Concomitant oesophageal disease may also be seen and further clarified by computed tomography. Aspiration of pleural effusion may be consistent with a reactive effusion and, if the pleura is ruptured, the aspirate may be acidic, bile stained or even contain food fragments which makes diagnosis easy. In the rare case in which the diagnosis remains in doubt after contrast studies oesophagoscopy should be undertaken.

Figure 1. Anteroposterior chest X-ray showing a right hydropneumothorax. The visceral pleura is thickened preventing expansion of the right lower lobe when the effusion was drained.

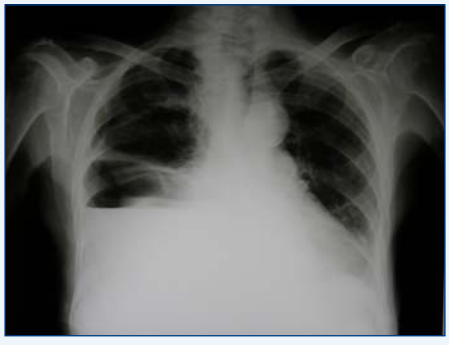
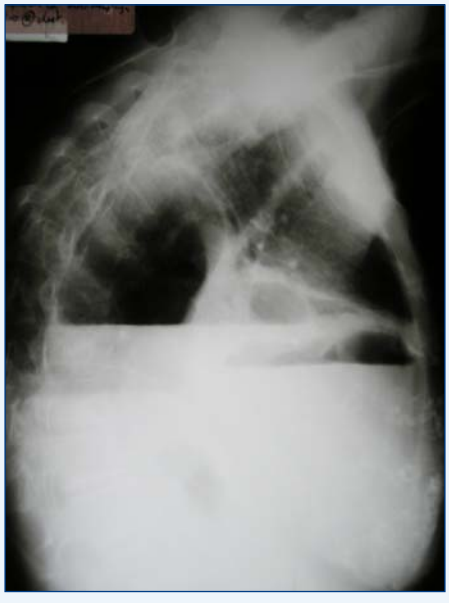


Figure 2. Lateral chest X-ray of the same patient showing the hydropneumothorax lies posteriorly.

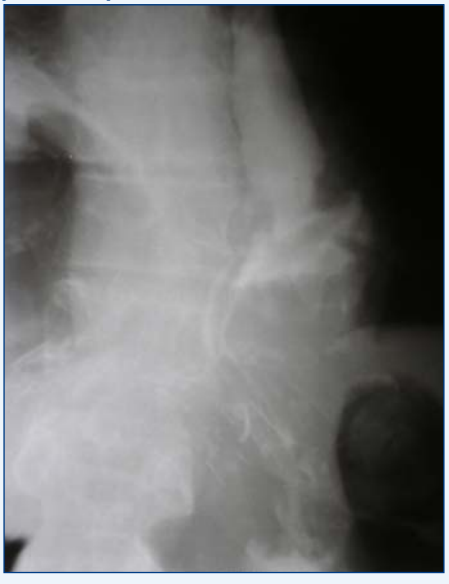


Treatment

Principles

- Time from perforation to diagnosis and treatment determines the management options and mortality
- Patients with oesophageal rupture should be discussed early with the appropriate surgical unit (Muir et al, 2003)
- Ventilatory, circulatory and volume support are provided as necessary
- The patient should receive broad spectrum, parenteral antibiotics with cover for anaerobic organisms
- Oral intake is prohibited. Once a treatment plan is formulated the appropriate route of nutritional support (e.g. feeding jejunostomy or total parenteral nutrition) is instituted
- Contamination of the pleura and mediastinum must be evacuated
- The oesophageal leak should be repaired, with maintenance of alimentary continuity if possible
- If the inflammatory process has allowed a cortex to develop on the underlying lung, restricting its expansion, this is removed by decortication (*Figures 1* and *2*)
- Surgical strategy may have to deal with any pre-existing oesophageal disease.

Figure 3. Water-soluble contrast study of the oesophagus showing leak of contrast into the right pleural cavity.



Methods of surgical treatment

Primary closure

This is the preferred method of surgical treatment. It allows thorough evacuation of the pleural and mediastinal contaminants, repair of the rupture and maintenance of alimentary continuity. Although this is easiest if performed early after the rupture it has been used successfully more than 24 hours after the event (Wright et al, 1995).

The rupture is approached through whichever incision gives better access, commonly a left thoracotomy for ruptures of the lower third of the oesophagus or a right thoracotomy for the less common tears of the middle third. This usually coincides with the side of the tear and also the side of the contaminated pleural space. The pleural space is evacuated and the mediastinal pleura opened widely. The defect in the muscular layer of the oesophagus is extended until the full extent of the defect in the mucosa can be assessed. The mucosa and muscle layers are closed separately. The authors prefer continuous closure using a non-absorbable monofilament suture such as polypropylene. Following closure the mediastinum and pleural space are lavaged extensively to remove any residual food fragments. If possible, the pleura can then be repaired. One or two pleural drains are inserted.

A number of authors recommend buttressing the repair. Parietal pleura, intercostal muscle, diaphragm, pericardium, omentum or the fundus of the stomach have all been used (Jones and Ginsberg, 1992). It is inadvisable to use any foreign material to buttress the repair in a heavily contaminated environment. Grillo and Wilkins (1975) described using a parietal pleural flap to wrap the repair. Non-randomized studies suggest buttressing probably reduces the postoperative leak rate compared to simple repair alone from 24% (Michel et al, 1981) to 14% (Gouge et al, 1989). The presence of systemic sepsis is associated with a significantly increased leak rate following simple repair and buttressing should be considered in all such cases (Wright et al, 1995).

Oesophageal resection

Rupture of the oesophagus in the presence of a resectable oesophageal carcinoma or massive oesophageal necrosis will necessitate a more extensive procedure and is the commonest situation in which oesophagectomy is undertaken for an oesophageal rupture (Orringer and Stirling, 1990). Salvage oesophagectomy may also be applied when the initial management plan has failed. Immediate reconstruction, using a gastric tube, is preferred but delay may be necessary if more complex conduits are needed. In this situation the cervical oesophagus is exteriorized, as an end oesophagostomy, a jejunostomy or gastrostomy is inserted and the distal oesophagus closed (Orringer and Stirling, 1990; Altorjay et al, 1998).

In reported series oesophagectomy as the primary treatment was applied in 10–20% of patients (Michel et al, 1981; Attar et al, 1990; Muir et al, 2003). Mortality ranges from 3.7% to 36% (Griffin et al, 1990; Orringer and Stirling, 1990; Altorjay et al, 1998; Muir et al, 2003). Three quarters of those discharged from hospital following oesophagectomy for oesophageal rupture have no swallowing problems (Altorjay et al, 1998).

T-tube drainage

If there is delay in diagnosis and an attempt at repair is jeopardized by intense inflammation or fibrosis around the tear, T-tube drainage can be an option to produce a controlled fistula. The edges of the tear are freshened and, once the T-tube has been inserted into the oesophagus, the tear is sutured over as much of its length as feasible. The T-tube is fashioned from an intercostal drain which has been incised along both aspects of its distal 10 cm. The short limbs of the T-tube are inserted into the proximal and distal segments of the oesophagus and the long limb is brought out across the pleural space and through the chest wall.

The T-tube is removed after 2–3 weeks once contrast swallows have confirmed healing around the fistulous tract. This closes spontaneously over the next 48 hours. Although used sparingly, in three of 66 patients in one study (Bufkin et al, 1996), this technique can allow oesophagectomy to be avoided in circumstances where the oesophagus is unsuited to primary repair.

Exclusion and diversion

This has already been discussed as an option when immediate reconstruction is considered unwise after oesophagectomy. It has been advocated by others as an option when the ruptured oesophagus is isolated and allowed to heal spontaneously. It is not an attractive option as subsequent restoration of swallowing will involve complex surgery. It has been reported as an occasional necessity in some series (Jones and Ginsberg, 1992) while other surgeons believe it has no place in the management of oesophageal rupture (Gouge et al, 1989). It has been reported as a salvage procedure following failure of more conservative surgery (Kotsis et al, 1997).

Drainage alone

Some authors have restricted their surgical approach to the evacuation of pleural and mediastinal collections, the placement of appropriate drains and establishment of enteral feeding but make no attempt to close the oesophageal rupture. In some series this approach was never thought to be necessary while in others it was the commonest surgical procedure (Muir et al, 2003). It is in accordance with the trend in some centres towards limited initial emergency operative intervention, so-called 'damage control surgery'.

Non-operative treatment

This comprises withholding oral intake, prescribing parenteral antibiotics, establishing gastric drainage and providing nutritional support. Cameron et al (1979) outlined the conditions where non-surgical treatment of oesophageal perforation could be attempted – a contained mediastinal leak with no pleural contamination and minimal systemic symptoms. Usually this is used for instrumental perforations identified at the time of injury and for barogenic ruptures that present late and have therefore been self selected as limited in extent and consequence.

These patients must be closely observed for signs of deterioration in a centre that can provide immediate surgical management if required. Any subsequent operation will be more complex and hazardous because of the delay and deterioration in the patient's condition. The frail patient with co-morbid conditions is often the one least able to tolerate the protracted recovery resulting from such a conservative approach. This is therefore not a substitute

for transfer to a specialist surgical unit, but something that may occasionally be used in such units.

In those series where non-operative treatment has been used it has been applied to between 3 and 23% of patients (Attar et al, 1990; Bufkin et al, 1996; Port et al, 2003; Muir et al, 2003). Mortality varies widely between 0 and 66% (Sawyers et al, 1975; Michel et al, 1981; Port et al, 2003; Muir et al, 2003). Patients treated non-operatively are a heterogeneous group comprising those with minimal consequences from their rupture and those presenting too moribund to undergo surgery.

Outcome

Series give overall mortality rates of 3.8% (Port et al, 2002), 16% (Muir et al, 2003), 17% (Michel et al, 1981), 19% (Kotsis et al 1997), 20% (D'Journo et al, 2006), 24% (Bufkin et al, 1996) and 35% (Sawyers et al, 1975). Mortality is affected by multiple factors, many outside of the surgeon's control. Richardson et al (1985) found those factors associated with increased mortality to be:

Pre-existing oesophageal disease

Confirmed by Michel et al (1981), mortality with a normal pre-perforation oesophagus was 4% compared with 23% where disease was present. Bufkin et al (1996) reported patients with oesophageal varices had a mortality of 83%.

Delay in treatment

Most series show a rise in mortality with delay of surgical intervention more than 24 hours post perforation – 13% *vs* 56% (Sawyers et al, 1975), 11% *vs* 28% (Michel et al, 1981) and 16% *vs* 52% (Attar et al, 1990). In the series by Muir et al (2003) time to diagnosis was the only significant predictor of mortality with immediate diagnosis (<1 hour) associated with a mortality of just 5%; early diagnosis (<24 hours) 14% and late diagnosis (>24 hours) 44%.

Thoracic site

Barometric rupture of the cervical and abdominal oesophagus are rare but are easier to deal with surgically and sepsis develops more slowly.

Following primary repair of barogenic rupture contrast swallow on seven of 10 survivors at a median 13 months following surgery showed no anatomical defect. However, on manometry six (85%) had oesophageal motility disorders and on pH monitoring four (54%) had nocturnal gastro-oesophageal reflux disease (D'Journo et al, 2006).

Conclusions

The importance of early diagnosis and the speedy institution of treatment measures cannot be overstated. This complex problem should be dealt with in specialist units as soon as transfer can be arranged. Data are limited to small retrospective series which show an evolution of treatment strategy over a number of years. Treatment must be individualized and the surgeon must have at his/her disposal a wide armamentarium of operative and non-surgical strategies. Casualty staff can influence the outcome of patients with ruptured oesophagus by considering this possibility whenever the history is suggestive. BJHM

Conflict of interest: none.

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KEY POINTS

- Ruptured oesophagus is a rapidly fatal surgical emergency.
- Early diagnosis is the single most important factor in determining patient outcome.
- All patients with ruptured oesophagus should be managed in a specialist centre.
- Non-operative intervention is applicable in a very small minority of cases and must be accompanied by close observation of the patient.
- There are a wide variety of operative interventions described and each patient's care should be individualized to his/her circumstances.