Oesophageal injury

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Revalidation FOR ANAESTHETISTS
RCA Revalidation matrix
Matrix reference
2A03, 3A03

Key points

The number of patients sustaining an iatrogenic oesophageal injury has increased.

Clinical features may vary and require a high index of suspicion.

Rapid deterioration may occur if diagnosis and definitive treatment is delayed.

Poor prognostic factors include: <24 h delay before treatment, Boerhaave's syndrome, underlying oesophageal disease, and a thoracic oesophageal perforation.

Outcomes may be improved by rapid referral to a tertiary centre with experience in the management of oesophageal injuries.

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Oesophageal injuries may be due to spontaneous perforations, trauma, or iatrogenic perforations. Despite outcomes improving, the morbidity and mortality for these patients remains high. This review outlines the aetiology and pathophysiology of the injuries, before describing the diagnostic and management strategies used by anaesthetists, intensive care physicians, and surgeons. Management of these high-risk patients relies on a high index of suspicion, early treatment of sepsis and organ failure, followed by an expedited transfer to a unit experienced in dealing with oesophageal injuries.

Aetiology and pathophysiology

With the increased use of endoscopic procedures, the incidence of oesophageal injury has increased and iatrogenic perforations during diagnostic or therapeutic procedures are now responsible for 60% of injuries, Boerhaave's syndrome (spontaneous oesophageal perforation) accounts for 15% of oesophageal injuries, with the remaining injuries attributable to trauma.

The incidence of perforation due to diagnostic flexible oesophagogastroduodenoscopy (OGD) and transoesophageal echocardiography is low.[1] However, therapeutic interventions combined with underlying patient risk factors can increase the incidence to 17% of endoscopic procedures.[2] Risk factors for oesophageal perforation during diagnostic and therapeutic OGD include patient-related factors such as underlying oesophageal pathology (e.g. oesophageal malignancy, oesophageal strictures, tissue damage after oesophageal or mediastinal irradiation, and eosinophilic oesophagitis), systemic disease (e.g. anterior cervical osteophytes, advanced liver cirrhosis, diabetes mellitus, and scleroderma), and advanced age; and factors related to the procedure such as heavy sedation, the level of operator experience, and the complexity of the intervention (e.g. oesophageal stent placement or pneumatic dilatation). During OGD, the most common site of injury is at the level of the cricopharyngeus, followed by the area proximal to the lower oesophageal sphincter. Injuries at this lower site are due to the angulation of the hiatus and the increased incidence of pathology such as oesophageal webs, rings, and strictures. Compared with patients without underlying oesophageal disease, patients with an inflammatory process or malignancy more commonly suffer thoracic perforations.

Over 250 yr ago, Boerhaave described the death of the Grand Admiral of the Dutch Fleet, Baron van Wassenaer, due to a spontaneous oesophageal perforation. Until the first reported repair by Barrett and Olson in 1947, the condition was universally fatal. Despite advances in surgical, medical, and critical care management, the syndrome continues to have a mortality of 20–75%, and left untreated remains near 100%.[3]

Perforations due to foreign body ingestion (most commonly dentures and animal bones), trauma (penetrating or blunt, after road traffic accidents, and the ingestion of caustic substances, particularly in children), operative injury, and tumours (even in the absence of diagnostic or therapeutic interventions) account for the remaining injuries.[4 5]

Oesophageal rupture permits the passage of food, gastric contents, secretions, and air into the mediastinum. The mediastinum can quickly become contaminated, and mediastinal emphysema and inflammation is followed by necrosis. Perforation of the overlying pleura may then occur. Negative intrathoracic pressure causes oesophageal contents to enter the pleural space, causing contamination of the pleural cavity and pleural effusion, most commonly on the left. This is explained by the fact that when perforation occurs proximal to the gastrooesophageal junction, the oesophagus lies adjacent to the left pleura (the middle region bordering the right pleura). Cervical perforations are generally less severe than those occurring more distally, as mediastinal contamination is limited by oesophageal attachments to the prevertebral fascia.

doi:10.1093/bjaceaccp/mku039

The time from injury to the initiation of treatment is a crucial factor in the outcome of these patients. In a large review of 726 patients with oesophageal perforation, the overall mortality in patients with treatment delayed for more than 24 h was 27% compared with 14% in those patients who were treated in <24 h.[6] Patients who survive have prolonged hospital stays and develop multiple postoperative complications. The most common causes of morbidity are pneumothoraces, mediastinitis, and pleural effusions.[7] Of these, mediastinitis is often the most difficult to treat. Direct tissue damage due to acidic enteric contents combined with bacterial contamination of the mediastinal pleura (which has a very poor blood supply) mean that therapeutic levels of systemic antibiotics may not be achieved at the target site.

Long-term quality of life will be determined by the management approach, which in turn is affected by the aetiology of the injury. Patients with a limited injury and contained leak may expect to have a normal quality of life once fully recovered from the acute episode. Patients with more severe injuries, such as those seen in Boerhaave's syndrome, who have undergone emergency oesophagectomy with a cervical oesophagostomy and feeding jejunostomy have reported a poor quality of life.[8]

Presentation and diagnosis

Clinical features vary according to the level of perforation and time interval to presentation. Symptoms may be non-specific, mimicking other diagnoses such as oesophagitis, peptic ulcer disease, myocardial infarction, pneumonia, spontaneous pneumothorax, acute pancreatitis, varices, or aortic dissection. The variety of presenting symptoms highlights the importance of always considering oesophageal rupture as a diagnosis in order to avoid any delay in definitive treatment.

Signs and symptoms

Initial examination may reveal a range of symptoms and signs. Patients will frequently complain of vomiting, dysphagia, and pain, dependent on the perforation site. On inspection, subcutaneous emphysema may be obvious, with neck and chest wall swelling, giving a characteristic crackling sensation on palpation as trapped air moves within the tissue planes. Percussion of the chest wall will be resonant if a pneumothorax is present, or indeed dull if there is lung atelectasis. Reduced air entry on the affected side is likely upon auscultation.

The more frequently occurring cervical perforations present with subcutaneous emphysema and anterior neck pain, exacerbated by movement and palpation, accompanied by dysphonia, dysphagia, or hoarseness.

Thoracic perforations tend to be more difficult to diagnose. Pain is present in 70% of full thickness thoracic oesophageal perforations. Other symptoms are non-specific (vomiting, dyspnoea, dysphagia), explaining the occasional post-mortem diagnosis, or indeed confusion with oesophagitis, myocardial infarction, spontaneous pneumothorax, or pneumonia. Pneumomediastinum can be heard as a cracking sound upon auscultation (the Hamman crunch), and Mackler's Triad, consisting of thoracic pain, vomiting, and subcutaneous emphysema, is highly suggestive, but seen in less than one-third of cases. Peritoneal cavity contamination occurs where a perforation is at the gastrooesophageal junction, and presents with an acute abdomen, epigastric or back pain, and referred shoulder pain. Differential diagnoses include peptic ulcer disease, acute pancreatitis, and aortic dissection, and a high index of suspicion should be maintained.

A systemic inflammatory response usually follows within hours of a thoracic or abdominal perforation, with septic shock and multiorgan failure developing rapidly. It is useful to note that these patients generally do not present with evidence of gastrointestinal bleeding such as haematemesis, or melaena.

Investigations

Blood tests

Blood tests may reveal acute inflammation with a leukocytosis and left shift (immature neutrophils), and also signs of dehydration (haematocrit up to 50%).

Posterior–anterior and lateral chest X-rays

The earliest finding is often cervical or mediastinal emphysema. A left-sided pleural effusion is commonly seen, but along with a widened mediastinum, takes hours to develop. Other findings include pneumothorax and atelectasis, but in many cases, plain film is normal (Fig. 1).

Gastrografin oesophagography

The use of a water-soluble contrast such as Gastrografin will usually reveal a contrast leak if there is a perforation. Barium should not be used as it may worsen mediastinal inflammation and, as it is not readily absorbed, might hinder future assessment of tear resolution. Contrast studies have a false-negative rate of 10%, so if clinical suspicion remains, they are worth repeating after 4–6 h. Such studies are also of great value after oesophageal repair, in order to investigate the possibility of an ongoing leak (Fig. 2).

Computerized tomography

Computerized tomography (CT) may reveal air in the peri-oesophageal tissues or mediastinum, a pneumothorax, pneumopericardium, pneumoperitoneum, or abscess. The addition of oral contrast can reveal a leak, and also identify the site of perforation and extent of contamination, thus guiding treatment. CT also offers the advantage of visualizing other organ pathology, and thus possibly excluding oesophageal perforation as a diagnosis (Fig. 3).

Flexible endoscopy

Although it holds the risk of extending a perforation, flexible endoscopy can prove invaluable when the patient is unable to swallow