Oesophageal injury

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Abstract

Oesophageal injuries are rare, the majority being iatrogenic, spontaneous (Boerhaave's syndrome) or caustic. Partial-thickness injuries are usually not serious, and heal, but, due to its location, full-thickness injuries of the oesophagus can have catastrophic consequences, leading to significant morbidity and sometimes death. Effective early treatment in experienced centres can lead to successful outcomes, although this relies on a low index of suspicion and rapid clinical recognition of this uncommon problem. Discussion with a specialist centre is mandatory, and it is usually appropriate to transfer the patient to such a centre. Computed tomography scan, contrast swallow and selective use of endoscopy by an experienced endoscopist are the investigations of choice, and a multidisciplinary approach to the management involving interventional radiology, interventional endoscopy and surgery will lead to the best outcomes.

Keywords Boerhaave; caustic injury; oesophageal perforation

Pathophysiology

Oesophageal injury can be defined as partial or full-thickness wall disruption. Partial-thickness injuries involve the mucosa and can be associated with pain and bleeding but generally heal. Full-thickness injuries lead to perforation, para-oesophageal contamination and often severe sepsis.

A recent review of hospital episode statistics for 'oesophageal perforation' diagnosis codes revealed an annual UK incidence of about 400 cases with a mortality of 35%. For thoracic perforations (the most common), bacterial contamination of the mediastinum by luminal contents is a life-threatening situation owing to the numerous sensitive adjacent structures. Additionally the negative pressure generated by breathing facilitates the spread of contamination through the chest. Parietal pleural breach on either side will seed pleural space collections, compounding sepsis and gas exchange, and potentially leading to empyema. Sepsis syndromes and organ failure will become established, eventually leading to death. If the initial septic episode is weathered, these patients often follow a protracted recovery punctuated by relapsing chest sepsis, nutrition problems and other complications.

Iatrogenic injury

Oesophageal injury during oesophagogastroduodenoscopy (OGD) is rare. However, thousands of these procedures are performed by individual NHS trusts per annum, and therefore it is a common mechanism of oesophageal injury. Typically, superficial partial-thickness lacerations may cause pain or self-limiting bleeding. Rarely, a full-thickness perforation may occur. The incidence changes with the nature of the technique and pathology of the patient; the British Society of Gastroenterologists quote incidences of 0.03% (diagnostic), 2% (haemorrhage control), 2% (benign dilatation), 4–6% (malignant dilation), and 5% (stenting). Particular risk situations are malignancy in the lower oesophagus and pharyngeal pouches in the elderly.

Numerous catheter-based procedures can also cause oesophageal injury; these include the insertion of nasogastric or nasojejunal tubes, the use of oesophageal monitoring equipment such as Doppler probes, and rarely oesophageal diagnostics equipment. Cardiac catherization can also lead to oesophageal injury; in particular atrio-oesophageal fistula after radiofrequency ablation of arrhythmogenic foci. Trans-oesophageal echocardiography carries a 1:500 risk of perforation.

Direct oesophageal injury can complicate a variety of operations, including cervical spine surgery, mediastinoscopy, lung transplantation, and thoracic aortic surgery. However, the most common surgical cause of oesophageal injury is during hiatal surgery, such as Nissen's fundoplication, Heller's cardiomyotomy and in para-oesophageal hernia repair.

Spontaneous injury

Spontaneous oesophageal injury can occur due to pressures generated by emesis against a closed cricopharyngeus. Simple mucosal (Mallory-Weiss) tears can give rise to bleeding and pain and typically settle early. Full-thickness injuries (Boerhaave's syndrome) are classically described as severe pain and subcutaneous emphysema after explosive vomiting, although in practice this triad is typically incomplete (0–26%). The perforation usually occurs in the distal oesophagus where the muscularis fibres taper towards the stomach. This is an uncommon pathology; a UK tertiary centre will see 3–6 cases per year.

Mechanical injury

An impacted oesophageal food bolus is a relatively common condition which fortunately resolves spontaneously in most cases. Of the 20% that require intervention, the vast majority will be cured with flexible endoscopy; a minority will require rigid oesophagoscopy or open surgery to alleviate tissue necrosis from local trauma. Penetrating food boluses such as fish bones are often to blame in cases requiring intervention. Sites of injury follow the anatomical impingements of the oesophagus (e.g. cricopharyngeus, cervical osteophytes, aortic arch, left main bronchus, and gastro-oesophageal junction).

Extrinsic penetrating oesophageal injuries are generally caused by stabbings or gunshot wounds, and are uncommonly incurred compared to adjacent structures (<6% of penetrating neck trauma, <1% of penetrating thoracic trauma). Oesophageal disruption due to blunt trauma such as road traffic accidents is rare, and typically occurs in the neck.

Caustic injury

The severity of oesophageal caustic tissue necrosis depends on the volume, pH, temperature and contact time of the ingested

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material. The initial tissue necrosis sloughs off in the first 48 hours, exposing deeper layers to the luminal stream. The depth of injury is proportional to the likelihood of perforation, and also the likelihood of further scarring and stenosis (late strictures approach 100% in full thickness necrosis). There are two UK caustic injury demographic groups: accidental caustic ingestion in 2–6-year olds (about 80%), and suicide attempts in 30–50-year olds. The lower oesophagus is often the worse affected region owing to the brief hold-up of the caustic bolus prior to LOS relaxation, which thus increases mucosal contact time. All caustic injuries are graded by depth of tissue necrosis from 1 (superficial, rarely requires surgery) to 3 (full thickness for lengthy segments, always requires surgery).

Acid corrosion causes coagulative necrosis through rapid protein denaturation. The formed coagulum is protective for deeper layers, and so a relatively superficial injury occurs. Additionally, they are repulsive to drink, and typically less volume is consumed. Alkalis cause liquefactive necrosis through the solubilization of lipids and proteins and saponification of fats, and so produce a relatively deeper injury. The lower pH of the stomach neutralizes the ingested bolus to a degree, and may be relatively spared.

Presentation

Clinical features will depend on the size and position of injury, and the time to clinical review. Oesophageal injury in the neck may give rise to pain, hoarseness, pharyngeal dysphagia, painful neck movement and neck spasms. Injuries to the thoracic oesophagus may cause pain, dysphagia, vomiting, haematemesis and shortness of breath. Abdominal oesophageal injuries may give abdominal pain and vomiting.

Clinical signs are proportional to the extent and duration of injury, and how established infection has become. The classic sign of surgical emphysema is often absent although occasionally may be florid, for example in the missed perforation during lengthy endoscopy. Respiratory compromise may be evident with tachypnoea and reduced saturations. Haemodynamic instability, arrhythmias and acute kidney injury may follow pyrexia, hypotension and other features of sepsis.

The presenting features are often non-specific; differential diagnoses include pneumonia, upper GI bleeding, oesophagitis, myocardial infarction, aortic dissection, perforated abdominal viscus, and pancreatitis. One study found that only 37% of spontaneous oesophageal injuries were correctly diagnosed at presentation. Therefore, the single most important clinical feature for diagnosis is the circumstance of the onset of symptoms. The ‘high index of suspicion’ for oesophageal injury is crucial in any unwell patient who has recently experienced: (i) an endoscopy or other thoracic instrumentation; (ii) protracted vomiting; (iii) unexplained weight loss and recent swallowing problems; (iv) known oesophageal stricture; or (v) thoracic symptoms in a self-harming patient. Urgent investigations to rule out oesophageal injury are mandatory.

Investigations

Ward-based tests

When oesophageal injury is suspected, basic investigations should be undertaken to establish the extent of sepsis, rule out differential diagnoses and prepare the patient for potential surgery. In particular, samples for a full blood count, renal function, clotting, group and screen blood type, should be sent off urgently, together with any necessary exclusion tests (e.g. amylase, liver function tests, calcium, cardiac enzymes). An arterial blood sample should be taken to assess respiratory physiology and acid-base balance. A 12-lead electrocardiogram will diagnose synchronous arrhythmia and assess myocardial ischaemia from the perspective of anaesthesia. A plain chest X-ray may show a widened mediastinum, pneumomediastinum, surgical emphysema, pneumothorax, pleural effusions, and consolidation, although often very little in the early phase.

Computed tomography imaging

Cross sectional imaging by computed tomography (CT) is essential for the assessment of full thickness injuries: (i) it is typically available at all hours; (ii) it offers sensitive and quantifiable detection of leakage of air and fluid outside the oesophagus; (iii) with oral contrast it is sensitive for minor oesophageal leak; and (iv) it is otherwise non-invasive and does not have the potential to make the injury worse. Classical features (Figure 1) are similar as for plain chest radiographs, although the precise spatial resolution enormously increases sensitivity. CT scans are insensitive for partial thickness injuries or caustic injuries, although they usefully exclude extra-oesophageal involvement in suspected partial injury.

Dynamic contrast studies

Dynamic contrast studies (DCS) are older techniques that can detect oesophageal perforations, although they are limited to the conscious patient and do not provide information about extra-oesophageal complications and adjacent soft tissue anatomy. There is debate regarding choice of agent. Barium is very slowly absorbed, can cause mediastinitis and may impair future studies, but its higher density improves its sensitivity for smaller perforations and may be useful to confirm a diagnosis prior to surgery if

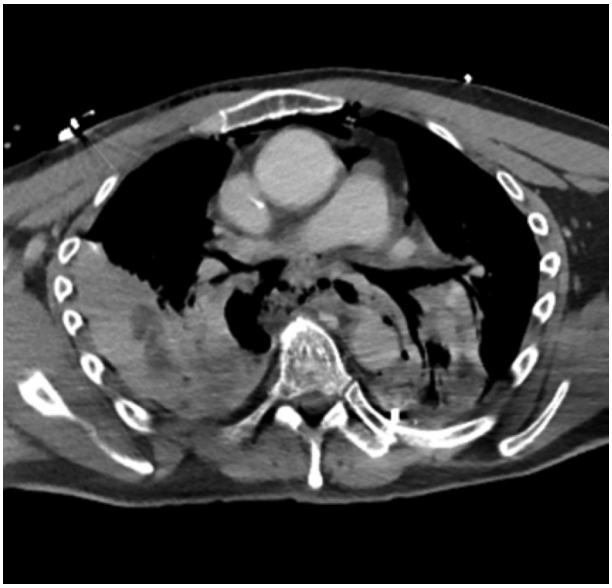

Figure 1 Axial computed tomography image of an acute Boerhaave’s syndrome, with subcutaneous emphysema, widespread pneumomediastinum and mediastinitis, and bilateral pleural effusions.

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