Oesophageal trauma

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Abstract

Trauma to the oesophagus is encountered rarely, but these injuries represent life-threatening emergencies. The majority are perforations secondary to therapeutic upper gastrointestinal endoscopy and to a lesser extent, diagnostic endoscopy, probably as a result of the dramatic increase in its use. The principles of investigation and management are similar for all causes of oesophageal trauma and outcomes are more dependent on the severity of the injury than the cause. These remain difficult conditions to manage as the diagnosis is frequently overlooked and through the lack of exposure and experience amongst clinicians in dealing with the spectrum of oesophageal trauma. A high index of suspicion is therefore required. Investigation with chest radiography or contrast swallow is standard, but CT and endoscopic assessment are used increasingly to make the diagnosis and to monitor the status of the injury or its repair. Non-operative management may be possible in carefully selected patients, but should be viewed as the 'radical' choice. The majority of patients require surgery and this should be performed in a specialist centre with experience in dealing with these patients.

Keywords Boerhaave's syndrome; caustic injuries; endoscopic perforation; iatrogenic perforation; spontaneous oesophageal perforation

Introduction

The oesophagus lies in the mediastinum which protects it from most external trauma other than extreme blunt or penetrating injuries. However, the escalating availability and use of upper gastrointestinal endoscopy and associated instrumentation of the oesophagus have resulted in an increase in iatrogenic damage which accounts for the majority of injuries. Oesophageal perforation carries high morbidity and mortality due to the difficulty of access to the oesophagus, the unusual blood supply, the lack of a strong serosal layer and the proximity of vital structures. Added to this, clinicians gain limited exposure to these cases due to their rarity.

Oesophageal perforation

Pathophysiology

Anatomical factors predispose the oesophagus to injury which may then lead to perforation. Full-thickness injury with gross food contamination rapidly leads to mediastinal and pleural soiling as acidic gastric secretions and bacteria are disseminated by negative intrathoracic pressure following breach of the mediastinal pleura. Resulting untreated sepsis from bacterial mediastinitis will swiftly progress to a profound systemic inflammatory response, multi-organ dysfunction syndrome and death. The degree and extent of contamination, the patient's underlying physical fitness and associated co-morbidities are important factors that determine outcome.

Aetiology

Iatrogenic perforation of the oesophagus: the overall risk of oesophageal perforation from a routine diagnostic endoscopy is very low (overall perforation rate: 0.03%). The majority of perforations occur in the distal oesophagus (90%) often in the presence of an underlying abnormality such as an oesophageal stricture. Proximal perforation is rare usually only occurring in the presence of an oesophageal diverticulum, pharyngeal pouch or endoscope-related shearing trauma with sharp cervical osteophytes.

Therapeutic upper gastrointestinal endoscopy procedures include those performed for benign disease such as balloon dilatation for achalasia and for malignancy, such as palliative endoscopic stent placement. These procedures carry a risk of perforation of about 5%, elevated in patients who have received prior chemotherapy or radiotherapy. Rigid oesophagoscopy remains a technique still used by ENT or thoracic surgeons and is associated with greater risk of perforation, similar to that of therapeutic intervention.

Non-endoscopic instrumentation of the oesophagus such as trans-oesophageal echocardiography can result in direct trauma or pressure necrosis as these monitoring devices are left in situ for prolonged periods of time.

Trauma to the oesophagus from the outside applies to open and laparoscopic approaches close to the oesophagus, for example during Nissen's fundoplication particularly when revisional anti-reflux surgery is being performed. Oeosphageal trauma has also been described following spinal, thoracic and head and neck surgery. Awareness of this risk is essential as a general/upper gastrointestinal surgeon may be asked into any of these operations if perforation is suspected.

Iatrogenic perforation is classically associated with less contamination as these frequently occur in fasted patients and the injury is more rapidly diagnosed.

Spontaneous perforation: the rare, eponymous Boerhaave's syndrome is defined as complete disruption of the oesophageal wall occurring in the absence of pre-existing pathology and is characterized by barogenic oesophageal injury leading to immediate and gross gastric content contamination of the pleural cavity, with rapid and catastrophic onset of chemical and bacterial mediastinitis.

Penetrating injuries of the oesophagus: sharp, penetrating injuries of the oesophagus are most common as it passes superficially through the neck. These injuries often occur in conjunction with other serious injuries to surrounding viscera so are easily missed. Consequent delay greatly increases morbidity and mortality.

Blunt oesophageal trauma: oesophageal trauma as a result of blunt injury is very rare, occurring almost exclusively to the

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thoracic oesophagus with blunt cervical trauma only occurring in high-velocity accidents. Trauma can result from vascular thrombosis due to severe contusion from traction laceration in rapid deceleration events or from barogenic damage.

Clinical presentation

The clinical features of oesophageal perforation depend on the cause, site and delay from injury.

Iatrogenic injuries: tend to be full-thickness and recognized at the time of injury. They may be associated with sudden onset of chest pain, dysphagia, odynophagia and surgical emphysema. The latter tends to be subtle initially and increase with time from the injury. Systemic symptoms are less common as due to pre-procedure fasting there is minimal pleural or mediastinal contamination. Cervical injuries present with neck pain and hoarseness.

Penetrating oesophageal trauma: manifests in a similar fashion, but a high index of suspicion based on the tract of the injury is essential for diagnosis and damage should be suspected in any transcervical or transmediastinal wound, especially when gunshot derived.

Spontaneous perforation: usually presents in a more profound manner with systemic disturbance which may present as septic shock. The classic triad of subcutaneous emphysema, chest pain and vomiting (Mackler's triad) was seen in only 15% of cases in a large national series (Griffin et al). It is essential that oesophageal perforation is included in the differential diagnosis of any patient who presents with acute cardiorespiratory distress of uncertain aetiology. Differential diagnosis for oesophageal perforation is the more commonly occurring myocardial infarction, leaking or dissecting aortic aneurysm, gastroduodenal ulcer perforation and acute pancreatitis. As a result, the majority of spontaneous oesophageal perforations are diagnosed with a significant delay by which stage the patient may have developed multi-organ failure as a consequence of overwhelming bacterial mediastinitis.

Investigations

With the exception of iatrogenic perforation a diagnostic delay of more than 12 hours is seen in **the majority** of cases. A high index of suspicion cannot be emphasized enough as an active approach to making the diagnosis may be life saving.

Following a full history and examination, a full blood count, urea and electrolytes, amylase and C-reactive protein (CRP) alongside arterial blood gases, blood grouping and a 12-lead electrocardiogram should be performed.

Plain radiography: the classical findings of oesophageal perforation on a chest radiograph are pleural effusion, pneumomediastinum, subcutaneous emphysema, hydropneumothorax, pneumothorax and collapse or consolidation. These features may be subtle and easily missed. Care must be taken to examine the subcutaneous tissues on the radiograph for surgical emphysema and for the presence of pneumomediastinum (Figure 1).

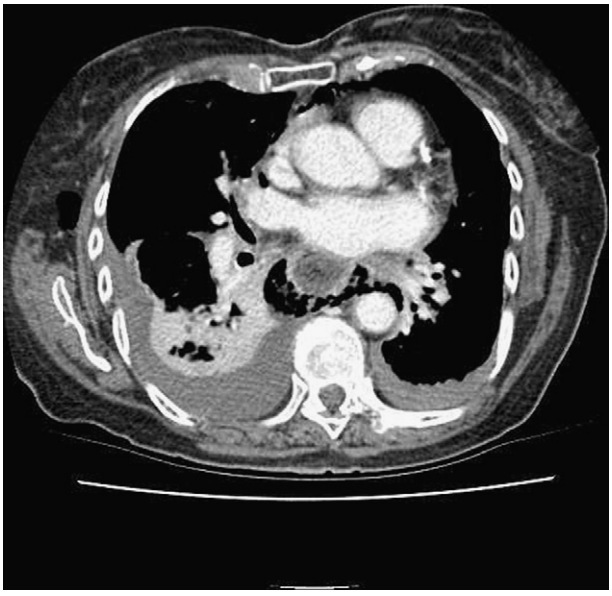

Figure 1 CT of the chest demonstrating pneumomediastinum and bilateral pleural effusions with right basal consolidation and collapse.

Computed tomography (CT): contrast-enhanced CT is increasingly used to make the diagnosis and is particularly useful when oesophageal perforation is suspected in a critically ill patient as it can be performed in a ventilated patient or in those in whom alternative differential diagnoses may be possible (e.g. aortic dissection). It is diagnostic, delineates mediastinal or pleural contamination and may allow drainage of any collections.

Contrast radiography (Figure 2**):** oral contrast radiography is the standard investigation to confirm perforation of the oesophagus particularly when iatrogenic injury is suspected, but is rapidly being superseded by CT scanning not least because a contrast swallow is not possible in ventilated patients. Water-

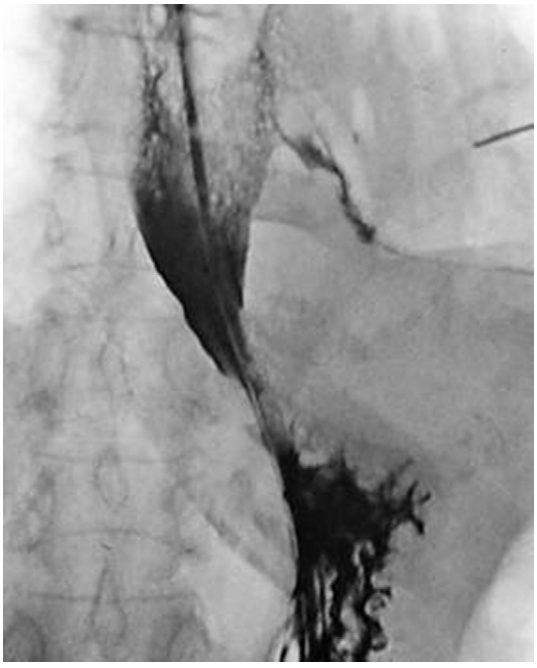

Figure 2 Contrast swallow demonstrating oesophageal leak following achalasia balloon dilatation.

soluble contrast is used initially, but if negative the radiologist may choose to use dilute barium which outlines better detail.

Upper gastrointestinal endoscopy (Figure 3)**:** flexible video-endoscopy allows confirmation of the diagnosis as well as critical assessment of perforation size, any associated pathology. Furthermore it allows placement of a nasogastric tube for drainage and a nasojejunal tube for feeding. Endoscopy is also especially useful in an 'on table' situation where trauma is suspected but where other injuries preclude radiological examination.

Other: drainage of gastric contents on thoracocentesis is diagnostic and may be aided by measurement of pH, amylase or microscopy for squamous cells. Administration of oral dyes, such as methylene blue, in the presence of a communicating drain may also be useful.

Management

Successful management is dependent on a multidisciplinary and active approach to the patient. Full resuscitation and early involvement of a critical care team along with close liaison with a regional oesophagogastric unit is crucial. Survival is reliant on avoiding or controlling mediastinal and pleural contamination with surgery being mandatory when gross contamination is present.

Patients require initial respiratory and cardiovascular assessment and support and opiate-based analgesia whether or not shock, respiratory distress or organ dysfunction is present. Regular re-assessment is obligatory.

Once the patient is stabilized, a decision needs to be made as to whether the patient should be managed operatively or non-operatively. This depends mostly on the degree of contamination.

Non-operative management: a non-operative approach may be considered in patients with minimal contamination, for example iatrogenic perforation or those with a delayed diagnosis who have demonstrated tolerance to the perforation. Criteria for non-operative management have been described (Box 1). This is only possible because of advances in interventional radiology, antibiotics and enteral nutrition. It is crucial to appreciate that non-operative treatment is not a 'conservative' approach but is actually a radical way to manage these patients. A low threshold for aggressive intervention should always be applied (e.g. radiologically guided drainage of collections).

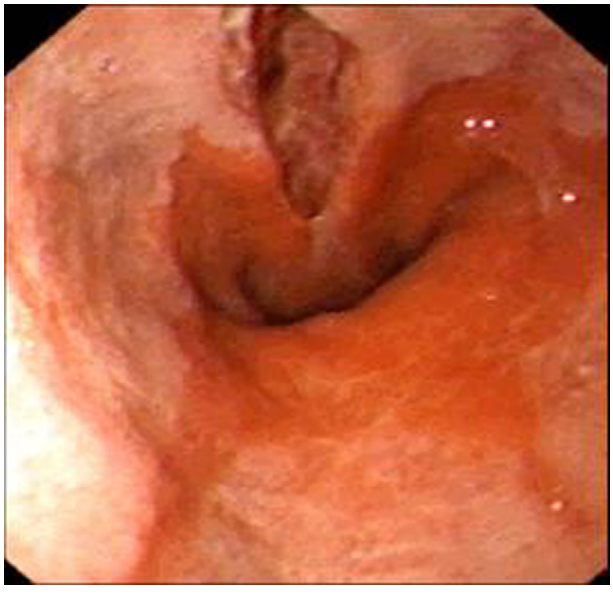

Figure 3 Endoscopic view of spontaneous oesophageal perforation.

Criteria for non-operative management of oesophageal perforation

- Contained perforation
- Free flow of contrast back into oesophagus on contrast swallow
- No symptoms or signs of mediastinitis
- No evidence of solid food contamination of pleural or mediastinal cavities

Other factors to consider

- Perforation is 'controlled'
- No underlying oesophageal disease
- No sepsis
- Availability for intensive observation and access to multidisciplinary care
- Low threshold for intervention
- Enteral feeding established

Box 1

Specific steps of non-operative management include:

- Management in a close observation unit such as high dependency with regular re-assessment by the surgical team.
- Strictly nil orally.
- A nasogastric tube should be placed under endoscopic and/or radiological assistance past the perforation to decompress the stomach and prevent bacteria and acid from escaping through the perforation into the pleura/mediastinum.
- Large-bore chest drains or radiologically targeted drains should be placed where pleural perforation has occurred.
- Contrast radiology, endoscopy and CT should be used to monitor the status of the oesophageal leak and collections. This will require assistance from an experienced radiology team.
- All patients should be given broad-spectrum, intravenous antibiotics, anti-fungal and anti-secretory agents.
- Low threshold for drainage of collections and surgical intervention.
- Enteral feeding may be established using either a feeding jejunostomy paced via mini-laparotomy or endoscopic nasojejunal tube placement.

Removable self-expanding metal stents may be used to seal iatrogenic perforations of malignant tumours if deemed unfit for resection but stent insertion cannot be recommended for perforations within a normal oesophagus, as expansion of the stent can expand the defect and in the presence of sepsis can erode into local structures.

Operative management

Surgery is indicated in a patient with:

- overt signs of sepsis, shock
- gross contamination (e.g. solid food in pleural space)

- presence of underlying pathology or retained foreign body
- failed non-operative management
- significant penetrating injury (e.g. gunshot wounds).

The principles of operative management are to clear the contamination, debride the tissues around the site of perforation and to restore oesophageal integrity whilst preventing further soiling. The main steps are:

- Left-sided (distal oesophagus) or right-sided (for upper or mid oesophagus) postero-lateral thoracotomy – the side and level dependent on the site of the perforation – usually through the fifth or eighth intercostal space.
- Double lung ventilation may be required throughout which can restrict surgical access due to the critically nature of the patients condition.
- Pleural and/or mediastinal collections are drained and specimens sent to microbiology for culture and sensitivity. The cavities are cleaned and lavaged thoroughly, the mediastinal pleura is incised to expose the injury and necrotic, devitalized tissue debrided.
- In a spontaneous perforation the mucosal injury is always longer than the muscular one, so an extending myotomy should be made to ensure repair.
- A feeding jejunostomy should be fashioned for enteral feeding.

Operative approaches

T-tube repair (Figure 4) – closure of the oesophageal defect over a T-tube and formation of a oesophago-cutaneous fistula is a safe approach and avoids the high leak rate associated with primary closure. A large-diameter T-tube (6–10 mm) is placed in the defect with the proximal and distal limbs of the tube well beyond the site of the perforation. The oesophagus is loosely closed around the tube with 3.0 absorbable sutures. The T-tube is exteriorized and secured at the skin. Great care must be taken to ensure that the T-tube is fully secured. The T-tube is left until a defined tract is established with the majority removed at around 6 weeks.

Primary repair – a primary suture repair should only be considered as an option for perforations that present early or with minimal contamination. The leak rate is approximately 50% for those delayed beyond 24 hours and 20% even within 24 hours, therefore primary repair has a limited role. The suture line may be reinforced with omentum, pedicled intercostal muscle flap or pericardium.

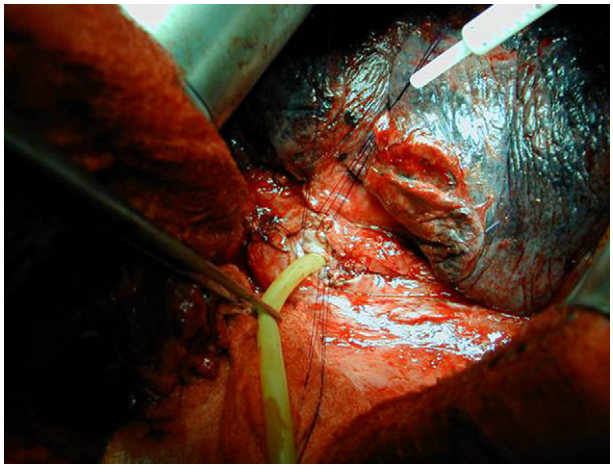

Figure 4 T-tube repair, lung displaced anteriorly.

Exclusion and diversion – exclusion of the contaminated mediastinum and diversion of secretions is complex and achieves no better results than other simpler treatments.

Oesophagectomy – oesophageal resection may be the only option in the presence of oesophageal pathology. Immediate reconstruction should only be considered in the presence of minimal contamination.

Perforation and cancer: perforation of an inoperable malignancy should be managed non-operatively, in this situation a sealing self-expanding metal stent may be appropriate treatment. In patients with less clearly defined operability most authors recommend resection. However, this carries considerable mortality (between 22% and 75%) and this treatment should be considered palliative. As such, every effort should be made to prevent iatrogenic injury during staging procedures.

Cervical perforation: perforations of the cervical oesophagus are often managed non-operatively with percutaneous drainage of collections but when uncontained, primary closure with pre-vertebral lavage and drainage using a left lateral incision anterior to the sternocleidomastoid is recommended and is well tolerated by even critically ill patients.

Caustic injuries

Significant ingestion of a corrosive chemical (predominantly acids and alkalis) is uncommon, but may be devastating both in the short term and long term. The majority of childhood ingestion injuries are accidental whereas in adults these are often as a result of a suicide attempt. The severity of the injury is dependent on the substance ingested, the amount, concentration, viscosity and the duration of contact with oesophageal mucosa. In general terms alkali injuries are often worse as these are 'easier to swallow' and thus are swallowed in greater quantities.

The first priority is establishing a safe airway as oropharyngeal damage by the agent can lead to extensive and life-threatening airway oedema. There is no place for 'neutralizing' the injury with another agent, nor steroids to prevent late strictures.

A chest radiograph should be performed to help rule out aspiration. CT and upper gastrointestinal endoscopy are the best methods of evaluating the trauma sustained. A classification has been developed in similarity to that for skin burns to assess the depth of injury and this helps to determine the best management (Box 2). Patients with minimal burns can be discharged if asymptomatic and there is no oropharyngeal injury. Other patients require in-patient observation, enteral feeding either via a nasojejunal tube or formal surgical feeding jejunostomy and monitoring for perforation. Management of full-thickness injury follows the same course as that for any oesophageal perforation with resection a more common requirement. Reconstruction can be immediate or delayed dependent on the level of contamination. Long-term sequelae are stricture formation, which can be refractory to dilatation, and the development of squamous oesophageal cancer some decades later.

Degrees of caustic injury

First degree: superficial mucosal oedema and hyperaemia
Second degree: transmucosal injury with bleeding, ulceration and exudates

- Airway management
- Analgesia
- Non-operative support

Third degree: full-thickness necrosis

- Operative management

Box 2

Conclusions

Although rarely seen by the majority of surgeons oesophageal trauma remains an important differential diagnosis to consider. Vital to a successful outcome is early liaison with an oesophagogastric unit as well as a high index of suspicion enabling an early diagnosis to be made. Both operative and non-operative management options have been shown to be successful even in frail, elderly patients and this condition should not be assumed to be uniformly fatal. ◆

FURTHER READING

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