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
Continuing Education in Anaesthesia, Critical Care & Pain | 2014

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

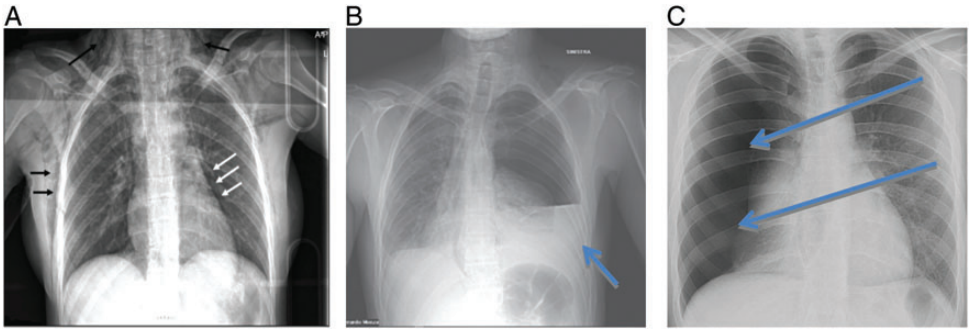

Fig 1 Radiographic presentations of oesophageal injury shown on PA chest radiographs: (A) subcutaneous emphysema (black arrows) and pneumomediastinum (white arrows); (B) left-sided pleural effusion (arrows); and (C) right-sided pneumothorax (arrows).

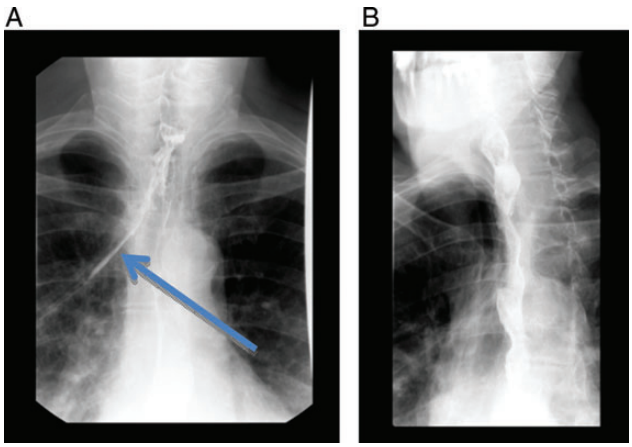

Fig 2 Example of PA chest Gastrografin studies (A) 10 days after operation, demonstrating contrast in the oesophagus and T-tube (arrow), and (B) 3 weeks after operation.

contrast or where there is a high index of suspicion with negative imaging. In many centres, it remains a key part of the routine work-up of these patients, permitting direct visualization of the exact site and size of the perforation and also an assessment of mucosal viability.

Pleural fluid

Pleural fluid obtained from thoracocentesis may indicate oesophageal perforation if testing reveals a pH<6, raised salivary amylase levels, or indeed if undigested food is present.

Immediate management

Successful initial resuscitation, rapid diagnosis, and management in a tertiary referral centre with experience in the management of oesophageal injuries improve mortality (Fig. 4). As overwhelming bacterial mediastinitis may rapidly cause multiorgan failure, patients with an oesophageal injury should be considered as being critically ill and require an aggressive approach to early resuscitation and management.[9] The principles of initial management are to treat infection, prevent continuing septic contamination, provide nutritional support,

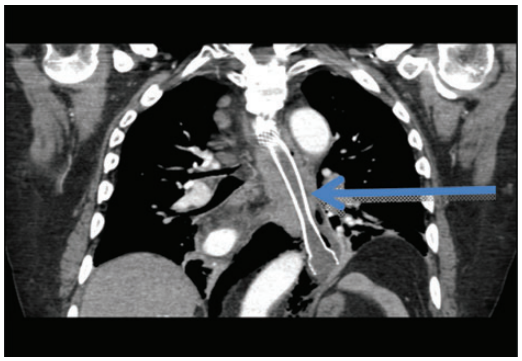

Fig 3 CT image illustrating oesophageal stent *in situ* (arrow).

and restore digestive tract continuity.[5] Individualized i.v. fluid therapy and appropriate analgesia should be instigated. Broad-spectrum prophylactic antibiotics, providing cover against aerobic gram-negative bacilli and anaerobes, should be given empirically. Such cover can be provided by regimens that include an extended generation cephalosporin in combination with metronidazole or clindamycin, or as single-drug regimens, for example, piperacillin–tazobactam or carbapenem.[10] Subsequent antibiotic regimens should be prescribed in consultation with a microbiologist. Patients must be kept strictly fasted and a proton pump inhibitor given. Total parenteral nutrition should be commenced.

Definitive management

Optimal care remains contentious, and the emphasis must be on timely transfer to a tertiary referral centre for appropriate management by an experienced multidisciplinary team.

Factors determining the most appropriate treatment strategy include the aetiology and size of perforation, and also patient co-morbidities and physiological reserve. Treatment options include medical, minimally invasive, and surgical management.

Medical management

Conservative treatment may be suitable for patients with limited oesophageal injury and contained leakage. Such patients include those

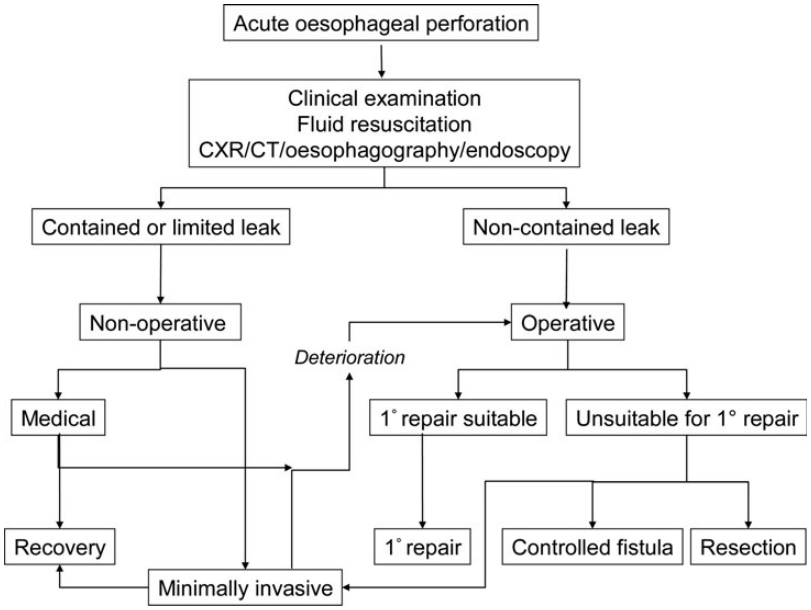

Fig 4 Overview of management options for acute oesophageal injury.

suffering endoscopic iatrogenic perforation, as the patient is likely to be fasted and the diagnosis made promptly. They must remain nil by mouth, with appropriate antibiotic cover, and proton pump inhibitor therapy, total parenteral nutrition, and continued observation. Similarly, medical treatment might be suitable for cases of inoperable malignant stricture, that is, palliation.

Minimally invasive management

There is an increasing trend towards endoscopic stent placement, particularly in cases of contained iatrogenic perforation with minimal contamination, and no evidence of sepsis. Vogel and colleagues[11] demonstrated a 100% survival rate in the 34 (of a total of 47 patients) treated with aggressive, conservative management. A covered stent is placed over the defect in a sedated patient using endoscopy and radiological screening, preventing further contamination while permitting early resumption of oral intake and drainage. The stent is then usually removed ~6–12 weeks later, once the defect has healed. Stent technology continues to evolve with metal, plastic, and biodegradable options available to the endoscopist. Self-expandable stents may be either fully or partially covered. Fully covered stents are more easily removed, but have an increased incidence of migration compared with partially covered stents, which are uncovered at their ends allowing them to embed into the oesophageal tissue. Stent migration can be a significant problem with symptoms of dysphagia, nausea, vomiting, and pain being suggestive. Meticulous ongoing observation is crucial, with repeated contrast oesophagography in case subsequent deterioration requires further intervention. These patients often undergo several other procedures, which may require a general anaesthetic, including tube thoracostomy, drainage gastrostomy, or feeding jejunostomy.

Surgical management

Primary repair is considered the gold standard operative approach, irrespective of time to diagnosis. It allows for optimal visualization of the perforation, and assessment of tissue damage, particularly in patients with extensive mediastinal contamination and devitalized oesophageal borders, as might occur in Boerhaave's syndrome. Repair at the cervical level is likely to involve a cervical incision, with drainage. Mid-thoracic perforations may require a left- or right-sided thoracoscopic or open approach. The majority of Boerhaave-type ruptures occur above the gastrooesophageal junction on the left side of the oesophagus, thus determining the approach for these cases. A midline abdominal incision, or laparoscopic approach, is reserved for intra-abdominal perforations. After lavage, and debridement of non-viable mediastinal and oesophageal tissue, primary repair is undertaken. This repair is frequently buttressed with a vascularized pedicle flap from intercostal, serratus, or latissimus dorsi muscle, pleura, or omentum in order to reduce fistula formation.

Surgical technique is best tailored to the individual case, and may constitute a hybrid approach including aggressive debridement, drainage, and stent insertion. Primary repair is difficult with Boerhaave-type ruptures due to high failure and leak rates. Therefore, if primary repair is deemed unsuitable, closure might be performed over a T-tube (promoting healing without contamination as an oesophago-cutaneous fistula forms). Additionally, placement of drains, anti-reflux procedures, or oesophageal resection with cervical oesophagostomy and distal feeding tube placement may be performed. In cases of obstructive pathology, including malignancy, end-stage achalasia, or refractory stricture, oesophagectomy is preferred. However, in an unstable patient, diversion may be performed without oesophageal resection. This involves construction of a cervical oesophagostomy permitting

adequate drainage, and a distal feeding tube. Definitive management can then occur at a later date.

Anaesthetic considerations

After appropriate resuscitative measures, the perioperative phase must be considered. Standard preoperative assessment and risk stratification should be undertaken, and communicated with the team and the patient. Physical examination in conjunction with radiographic findings may alert the anaesthetist to the presence of respiratory sequelae such as pleural effusion or atelectasis.

Induction and securing the airway

The conduct of anaesthesia will vary depending on the clinical state of the patient and the procedure performed. Patients might well be in the early stages of septic shock, and choice of induction agent should be considered carefully, with vasopressors/inotropes readily available in the event of hypotension. Patients with oesophageal rupture remain at risk of aspiration, and a rapid sequence induction should be performed, with care taken to minimize the risk of exacerbating the injury further (coughing, straining). Cricoid pressure remains controversial, and the risk of exacerbating contamination through the perforation should be weighed up against soiling the lungs in an already compromised patient. If it is deemed necessary to secure the airway awake, then care must be taken to avoid coughing. Once the airway is secured, an NG tube should be placed in the proximal oesophagus, above the injury. During the operative procedure, the surgeon will then position the NG tube beyond the repaired oesophagus to ensure that the stomach remains decompressed. The NG tube is not used for enteral nutrition, which can be achieved via a jejunostomy formed at the time of surgery.

It is usually essential that the lungs can be isolated. According to operator skill and preference, either a single-lumen tracheal tube with bronchial blocker or a double-lumen tube should be inserted, thus permitting ventilation of either one or both lungs. Malposition of a double-lumen tube is not uncommon, and fibreoptic confirmation of its position should be made after intubation. The physiology and conduct of one-lung ventilation has previously been described.[12]

Anaesthetic conduct and positioning

A balanced anaesthetic technique usually involves maintenance with a volatile agent and good analgesia. Normothermia should be maintained using a forced air device, and warmed i.v. fluids. The patient will be required to be in differing positions, according to the pathology and chosen surgical approach. Primary repair, thoracoscopy, or open thoracotomy will necessitate a lateral position. The upper arm will need to be abducted for surgical access, ensuring that there is not excessive stretch on the brachial plexus. Care must be taken to avoid corneal abrasions.

It is likely that the patient will need to be moved intraoperatively, frequently more than once. Meticulous attention should be paid to pressure points, it is important to recheck tube position after any repositioning, as turning the patient to a lateral position can result in proximal movement of the tube. Further detailed intraoperative management can be found in the *CEACCP* article on minimally invasive oesophagectomy.[13]

Fluid therapy

If not already inserted, invasive arterial pressure monitoring, and central venous access should be instituted. In our institution, we utilize cardiac output monitoring via pulse contour analysis (oesophageal Doppler technology is clearly impractical), and implement individualized goal-directed fluid therapy throughout both the peri- and postoperative phases. Fluid management in the elective patient undergoing elective oesophageal surgery is a balance between avoiding excessive fluid, which may exacerbate acute lung injury after operation, and causing postoperative hypovolaemia, hypoperfusion, and acute kidney injury due to restrictive fluid regimens.[14] In contrast, patients who have sustained an oesophageal injury are often exhibiting overt signs of multiorgan failure with haemodynamic instability and leaky capillaries. We aim to maximize stroke volume, while avoiding fluid overload. The definitive goal is tissue oxygenation, and central venous oxygen saturation can be used as a surrogate for mixed venous oxygen saturation. Blood gas analysis is performed at intervals, and hourly urine measurements taken.

Analgesia

Adequate postoperative pain control is essential, in order to prevent delay in extubation or mobilization. In the absence of systemic sepsis, a thoracic epidural may be inserted before operation. However, if a neuraxial technique is contra-indicated, or the patient refuses, we utilize a remifentanil infusion perioperatively. Paravertebral blocks and wound catheters may also be used, depending on the condition of the patient and the surgical approach. A multimodal approach is then adopted once the patient is awake, including regular paracetamol and i.v. morphine patient-controlled analgesia.

Postoperative care

Patients are transferred to the intensive care unit sedated and ventilated. They receive a maintenance balanced crystalloid infusion of 1 ml kg^{-1} h^{-1} after operation, with further goal-directed fluid therapy being guided by cardiac output monitoring. In selected patients, with minimal organ dysfunction, there is an emphasis on early extubation, enteral nutrition (jejunal), and mobilization, which mirrors the enhanced recovery pathway developed for our elective oesophageal resections.[15] Patients with sepsis and organ dysfunction at presentation often require prolonged organ support. Postoperative management also includes broad-spectrum antibiotics, and close observation for signs of sepsis. Possible collections or leakage can be evaluated using ultrasound or CT. A Gastrografin contrast study may be performed when there is no ongoing sepsis or mediastinitis, often at 2–3 weeks after repair of the injury. If a T-tube has been left *in situ*, a contrast swallow is performed to look for evidence of a leak around the

repair. If there is no evidence of a leak, then the patient is allowed to gradually build up to a normal diet, and the T-tube may be removed at 8–10 weeks after operation. This involves an overnight stay.

Conclusions

Oesophageal injury, either spontaneous or iatrogenic, remains a devastating condition if not recognized and treated rapidly. The incidence is increasing in line with the increased utilization of endoscopic interventions. The difficulty in diagnosing the condition requires a high index of suspicion. Outcomes are improved by early management in a tertiary centre experienced in the management of oesophageal injuries.

Declaration of interest

None declared.

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