Management of upper gastrointestinal perforations

Emily Moore
Nima Abbassi-Ghadi

Abstract

Perforation of the upper gastrointestinal (UGI) tract is a surgical emergency. Causes of oesophageal perforation include spontaneous (Boerhaave's), iatrogenic or foreign body ingestion. Perforation of the stomach and duodenum is most often caused by peptic ulcer disease. Management involves obtaining an accurate clinical diagnosis, through a combination of patient assessment, imaging and endoscopy. It is important to differentiate intramural from full thickness oesophageal perforations as this will guide the definitive surgical or endoscopic management. Perioperative care of these patients is as important as the definitive management step. This article will summarise an approach to managing perforation of the UGI tract; from initial assessment to postoperative care.

Keywords Boerhaave's; Esosponge; gastroduodenal perforation; oesophageal perforation

Introduction

Perforation of the upper gastrointestinal (UGI) tract includes the oesophagus, stomach and duodenum. This is a surgical emergency associated with high morbidity and mortality but early detection, and initiation of appropriate treatment, improves outcomes for these patients. This article focuses on the principles of management of these conditions.

Types of upper gastrointestinal (UGI) perforations

Oesophageal perforation

Iatrogenic: Eighty per cent of oesophageal perforations are iatrogenic, secondary to endoscopic procedures.[1] The incidence is less than 1% in diagnostic flexible endoscopy,[2] occurring mostly at the cervical oesophagus or hypopharynx, most often due to non-identified pharyngeal pouch or presence of cervical osteophytes. Therapeutic endoscopy (e.g. dilatation of strictures, stent placement, endoscopic resection, etc) is the more common cause of oesophageal perforation, with an incidence of approximately 5%.[2] Perforation can also occur as a result of endotracheal intubation, nasogastric (NG) tube insertion or transoesophageal echocardiography.

Boerhaave's syndrome: Spontaneous oesophageal perforation, or Boerhaave's syndrome, accounts for approximately 15% of all oesophageal perforations.[1] Forceful emesis or retching against a closed glottis causes a sudden increase in intraluminal pressure, resulting in full thickness disruption of the oesophageal wall. Anatomically, this usually affects the left posterolateral aspect of the distal oesophagus, owing to a lack of supporting mediastinal structures here. The tear is often located between the clasp and oblique fibres, extending upwards.[3]

Foreign Body Ingestion: Due to their size, rigidity, and pointed edges, dentures frequently impact in the oesophagus but ingestion of any foreign body of size with sharp edges can result in perforation. Impaction of blunt foreign bodies can also cause perforation due to pressure necrosis.

Gastroduodenal perforation

Peptic ulcer disease (PUD) is the leading cause of perforation of the stomach and duodenum, with a lifetime risk of 5% in patients with the condition.[4] Despite the widespread introduction of proton pump inhibitors, PUD continues to be the most common cause of gastroduodenal perforation due to the increasing prevalence of *Helicobacter pylori*, prescribing of NSAIDs, consumption of alcohol and smoking tobacco.

Perforation mostly occurs in the anterior wall of the first part of the duodenum but also at the lesser curvature of the stomach. It is important to remember that *posterior* duodenal ulcers may perforate into the retroperitoneum rather than into the intraperitoneal cavity. Signs and symptoms of a contained perforation into the retroperitoneal space may be more subtle, and a high index of suspicion is required for diagnosis. Different management strategies may be employed for intraperitoneal versus retroperitoneal duodenal perforations.

Management of UGI perforations

Initial management

Patients may present extremely unwell with signs of mediastinitis or peritonitis, sepsis, and shock. Immediate management should focus on adequate resuscitation and stabilization of the patient following an A to E approach. This should include intravenous (IV) fluids and broad-spectrum IV antibiotics with cover for aerobic Gram-negative bacilli and anaerobes. The benefit of administering prophylactic antifungal therapy is debated. In addition, patients should have oral intake limited to sips of water or nil by mouth depending on site and extent of perforation, and given IV proton pump inhibitors (PPIs).

In patients who have a hydropneumothorax, an intercostal drain (ICD) may be required during resuscitation to allow for lung expansion, to preserve respiratory function and to ensure stable transfer of the patient. ICDs also have a role in the diagnostic work up; drain insertion yielding bile, or raised amylase, supports a diagnosis of oesophageal perforation.

Centralization of care

Patients with oesophageal perforations should be managed in a specialist tertiary centre where possible. If there is no oesophagogastric team on site, patients should be promptly discussed with

Emily Moore *MBChB MRCS is a Core Surgical Trainee at the Royal Surrey County Hospital, Guildford, UK. Conflicts of interest: none declared.*

Nima Abbassi-Ghadi *PhD FRCS is a Consultant Oesophago-Gastric Surgeon at the Royal Surrey County Hospital, Guildford, UK and Honorary Senior Clinical Lecturer at the University of Surrey, Guildford, UK. Conflicts of interest: none declared.*

and transferred to a hospital where this is available. This centralization of care is fundamental, as management of perforations in centres with experience of managing oesophageal injury directly reduces mortality.[5]

Perforated peptic ulcers are a surgical emergency managed by most general surgeons. They do not usually require specialist input.

Definitive management

Oesophageal perforations

Management of oesophageal perforations is patient specific and is guided by a combination of factors including the aetiology, size of perforation and degree of contamination, as well as patient comorbidities and underlying physiological reserve.

Mortality of oesophageal perforation can be up to 50% if initiation of treatment is delayed beyond 24 hours,[6] so early diagnosis and *decision* for management is essential. Options include conservative/medical management, surgical intervention, or endoscopic therapies.

Steps of Surgical/Endoscopic Management

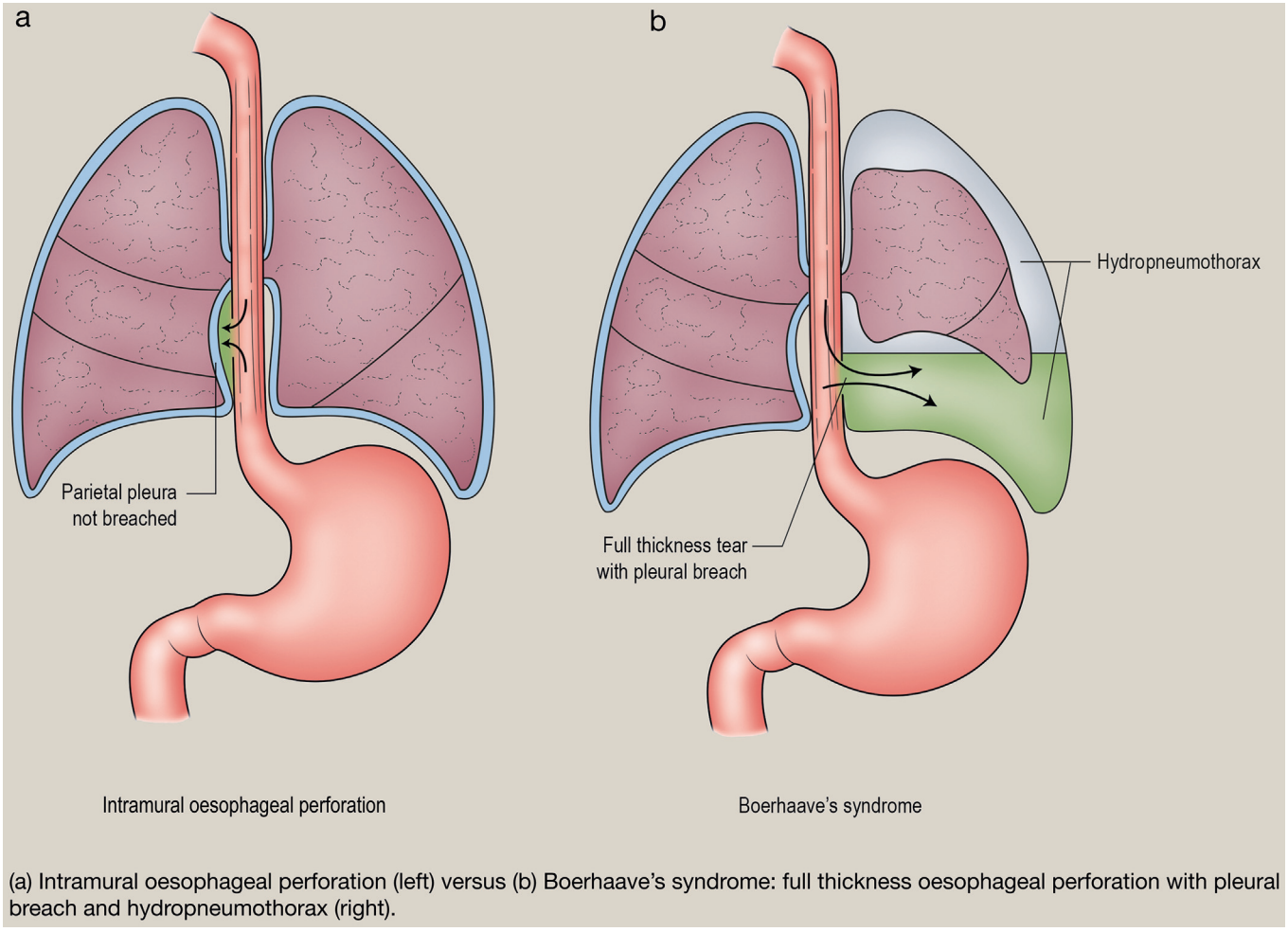
1. Defining anatomy and extent of contamination:
   It is important to identify the location, extent, and cause of the perforation correctly for appropriate management and surgical planning. Diagnosis is made using a combination of computed tomography (CT) imaging and upper GI endoscopy.
   a. CT with oral contrast has a sensitivity of 92%−100% in the detection of oesophageal perforation.[1] It is used for the following reasons:
      i. Determining presence of contrast leak and into which cavity.
      ii. Differentiating pleural/peritoneal contamination from perforation just isolated to the mediastinum.
      iii. Identifying other pathology, for example, malignancy.

   Radiological features suggestive of perforation include extraluminal free air, luminal contrast leak or evidence of collections in the mediastinum and in the neck. Whilst useful for detecting the presence of a perforation and its extent, CT is not a good modality for localizing the site of perforation and this may be difficult to see radiologically. This is therefore achieved using endoscopy.

   b. Oesophagogastroduodenoscopy (OGD) allows direct visualization of the GI tract to ascertain the exact anatomical site and size (length) of the perforation. The position and laterality of the defect relative to the gastro-oesophageal junction (GOJ) can be noted. It also has a role in identifying other mucosal pathology, for example, oesophageal cancer or diverticulum.

   An important distinction is that of intramural oesophageal perforation as opposed to a full thickness oesophageal perforation. The layers of the oesophagus from innermost to outermost, are mucosa, submucosa, muscularis propria and serosa. However, in the thoracic oesophagus, the serosal layer is replaced by parietal pleura which isolates the mediastinum. A full thickness perforation, therefore, is one which breaches the parietal pleura. This manifests itself as a pleural effusion or as a hydropneumothorax, which may be detected clinically or radiologically. Boerhaave's syndrome is a type of full thickness oesophageal perforation (Figure 1 a and b).

2. Categorizing and treating:
   Once this information has been gained, we can categorise and treat accordingly. The principles of surgical treatment include debridement or lavage of mediastinal/pleural contamination and closure of oesophageal perforation.
   a. Intramural oesophageal perforation (oesophageal perforation with mediastinal contamination and no parietal pleural breach)
      i. Large defect with *significant* mediastinal contamination is best treated with Esosponge (Figure 2a−c). Esosponge is an endoluminal VAC (E-VAC) therapy designed specifically for the management of perforations and anastomotic leaks.[7] The sponge is placed endoscopically to sit within the lumen of the oesophagus adjacent to the perforation or within the perforation cavity itself, and is connected to a negative pressure vacuum pump. This drains infection, promotes granulation of healthy tissue and therefore healing of the oesophageal wall; thus, reducing the overall size of the cavity over time. It is replaced at intervals of approximately 72 hours until endoscopic visualization of the oesophagus demonstrates sufficient healing. Of note, a nasojejunal (NJ) catheter does not position well adjacent to the sponge and therefore these patients require total parenteral nutrition (TPN).
      ii. Small defect with *minimal* mediastinal contamination − an NJ tube for feeding (to allow for diversion beyond the healing oesophagus) +/− NG tube for gastric content drainage (to prevent further contamination) may be required if the perforation crosses the GOJ or there is a non-functioning lower oesophageal sphincter (e.g. hiatus hernia).
   b. Full thickness perforation (Figure 3)
      i. This requires pleural toilet/decontamination and repair of the oesophageal perforation. It can be achieved via thoracotomy or thoracoscopy, with the laterality and exact approach determined by the anatomical findings. The principles of this are as follows:
         - Debridement
           Removing all necrotic and non-viable tissue of the pleural cavity and mediastinum is essential.
         - Primary closure
           This should be the aim in all patients who do not have underlying disease of the oesophagus. This is performed by a 2-layer closure of the mucosa and surrounding muscularis layer, or where this is not possible, by creating a vascularized local flap for a buttress repair, sourced from the intercostal, serratus or latissimus dorsi muscle, omentum or pleura (Figure 4). A buttress repair has been shown to decrease fistula formation and overall mortality. Drains are often placed adjacent to the closure in case of leakage.
         - T-tube
           Closure can be performed over a T-tube, creating a controlled oesophageal−cutaneous fistula to allow for continuous drainage of infection, and healing. This is useful when sepsis and



(a) Intramural oesophageal perforation (left) versus (b) Boerhaave's syndrome: full thickness oesophageal perforation with pleural breach and hydropneumothorax (right).

Figure 1

inflammation are severe, creating an unfavourable environment for healing of the oesophagus after direct closure. Drains are often placed adjacent to closure in case of leakage around the T-tube. They will then need to be pulled back gradually to allow pleural and mediastinal structures to isolate the T-tube and create a fistula tract.

- Resection
  This may be indicated if the extent of damage is beyond repair. Oesophageal resection is therefore appropriate when the oesophagus is diseased or obstructed, as in malignancy, stenosis, or reflux-related strictures. Oesophageal malignancy may be treated with oesophagectomy and reconstruction

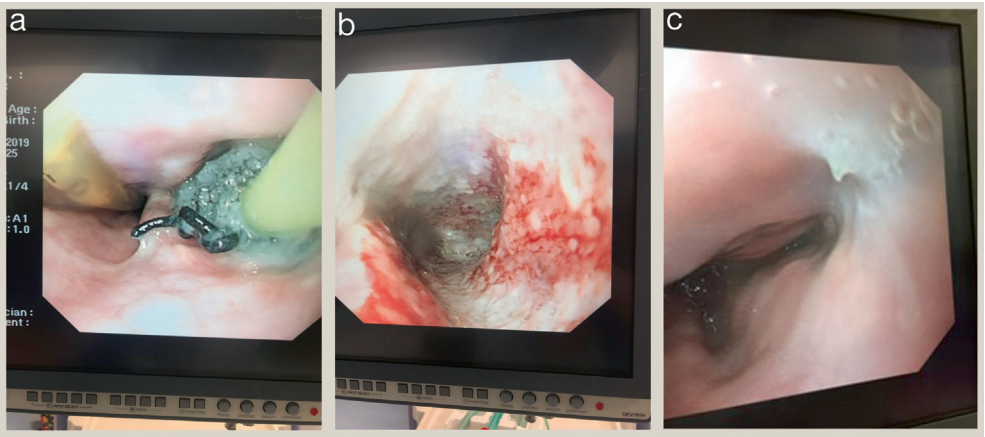


Figure 2 Esosponge for management of oesophageal perforation. (**a**) Day 0 - placement of Esosponge for Boerhaave perforation, (**b**) Day 3 – developing granulation tissue, (**c**) Day 30 – sealed perforation with mucosal scarring.

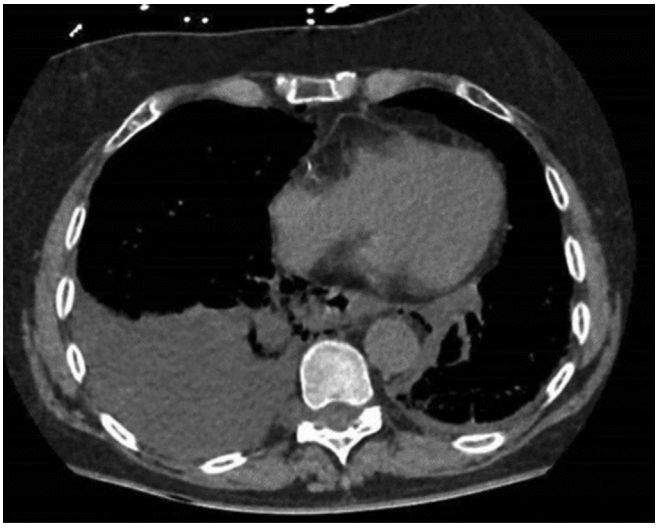


Figure 3 CT showing oesophageal perforation with extensive left pleural collection.

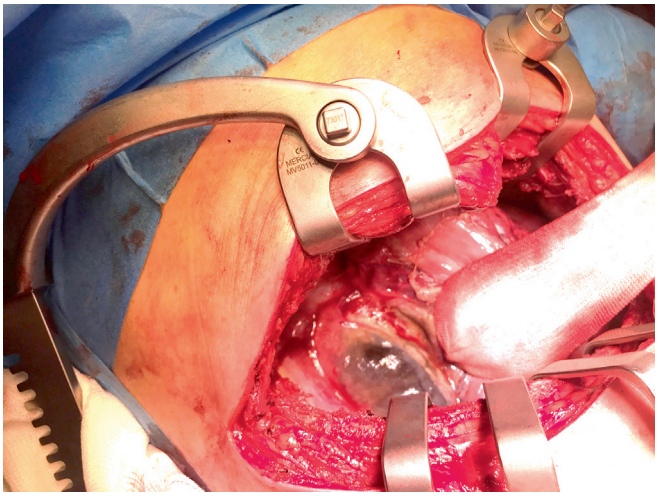


Figure 4 Thoracotomy plus pleural lavage/decortication with primary repair of oesophageal perforation.

in the acute phase, provided the patient would otherwise be suitable to undergo a resection and there is minimal physiological disturbance. However, most patients are not suitable for this approach due to physiological disturbance from the perforation and therefore require a damage limitation approach.

ii. Laparoscopy, or an open midline incision must be used to access intra-abdominal perforations.

iii. Esosponge – in patients too unfit to undergo a major operation, Esosponge is a useful alternative, as outlined above.

Other endoscopic techniques

Endoscopic therapies are reserved for patients in whom there is minimal or no contamination, or who are considered too unfit to undergo an operation.

Options for this include stents, clips and suturing. These therapies may be of benefit in cases where there is no mediastinal/pleural contamination/necrosis, particularly iatrogenic perforation discovered at the time of endoscopy. They should be used with caution as untreated mediastinal or pleural contamination can lead to ongoing sepsis and the potential for abscess formation.

a. **Stents**: Endoscopically placed self-expandable stents, made from metal or plastic, are an option in patients with minimal degree of contamination. Completely uncovered stents run a higher risk of tissue reaction and granulomatous inflammation, and are difficult to remove; thus, they are not often used here as a temporising measure. Fully covered stents are more favourable as they are easily removed. Nonetheless, these stents may slip and erode into surrounding structures so proximal fixation with clips or endoscopic suture may be useful. Early exchange or removal (at approximately 4 weeks) is advocated to prevent tissue ingrowth/embedding or erosion/migration. Stent migration is associated with symptoms of epigastric or chest pain, nausea or vomiting and dysphagia and can therefore be very debilitating for a patient. Close observation of patients with stents is recommended and they may require repeated contrast studies.

b. **Clips**: Defects can be successfully closed through endoscopic clipping[8] (through the scope (TTS) or over the scope (OTSC) clips) though this alone does not deal with mediastinal contamination.

c. **Sutures**: Endoscopic suturing can be performed with the Apollo Overstitch system,[9] allowing for continuous or interrupted sutures for primary closure of the defect.

Perforated peptic ulcer disease

Diagnosis

A perforated peptic ulcer may be suspected if free air underneath the diaphragm is seen on erect chest X-ray. However, most patients with an appropriate history and examination findings would proceed directly to a CT abdomen and pelvis. Findings of peri-gastro-duodenal free fluid, free air and fat stranding are suggestive of perforation. Peri-portal free gas sign (PPFG) is considered to be diagnostic of upper GI pathology, thus helping to differentiate between upper and lower GI tract perforations.[10] Scans using oral contrast may show luminal contrast leak, though the sensitivity of this is disputed.

Surgical management

It is now generally accepted that non-operative management of perforated peptic ulcers is safe only in a select number of cases. For this to be considered, the perforation must be small, the patient stable with normal vital signs and no signs of sepsis, and the ulcer must have been shown to *already* have healed by water contrast study. However, it should be noted that mortality increases with each hour of delay towards surgery, and if there is any uncertainty about suitability for non-operative management, this should be avoided.[11]

Omental patch repair

The primary method for operative management of perforated ulcers is by an omental patch, for which there are various described techniques. This can be done open, via laparotomy, or more increasingly, by laparoscopy; however, laparoscopic repair has been shown to be superior only in reducing postoperative pain.[12]

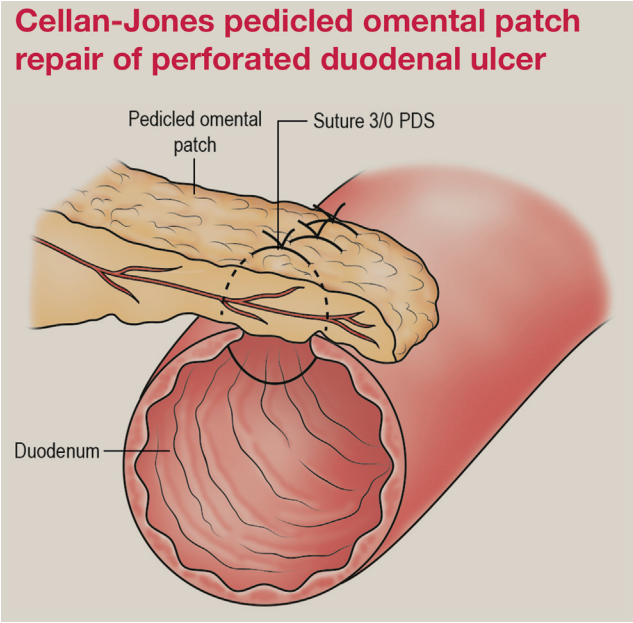


Figure 5

Intra-abdominal contamination firstly requires peritoneal washout to control sepsis and decrease the likelihood of abscess development. The Cellan-Jones technique (Figure 5) is widely accepted as the gold standard approach for peptic ulcer repair and defines a pedicled omental patch repair. The repair is tension free and allows for the formation of an omental plug secured with sutures.[13]

Empirical treatment for *H. pylori* is given (triple therapy consisting of a PPI and two anti-bacterials) to help to prevent recurrent PUD. It must be remembered that approximately 10% of perforated *gastric* ulcers are malignant. Therefore, any gastric ulcer must be biopsied intraoperatively and sent for histology.

Giant duodenal ulcers

Giant duodenal ulcers are those whose size exceeds 3 cm in diameter. These are an increasingly rare occurrence in the developed world due to the improved medical management of PUD but remain an important consideration in the developing world. They represent a surgical challenge as the much larger size of defect results in high postoperative leak rates of up to 10% with traditional repair methods, and patients are often more unwell with haemodynamic instability.[14]

Definitive management would involve a distal gastrectomy but in instances when this is not achievable other techniques can be attempted. Good results have been demonstrated using a triple ostomy approach — primary closure is achieved using an omental patch repair, followed by the formation of a gastrostomy, retrograde duodenostomy and feeding jejunostomy.[14] Others have described creating a jejunal serosal patch, or resecting the diseased portion of duodenum and distal stomach via antrectomy and Billroth 2 (gastrojejunal) reconstruction.[15]

Postoperative management

Oesophageal

The majority of patients with oesophageal perforation will require intensive care in the perioperative period for close monitoring and expectant treatment. They will often develop sepsis, arrhythmias and organ dysfunction due to mediastinal/pleural contamination and systemic inflammatory response syndrome (SIRS).

Approach to postoperative management can be considered in an A to E format.

A — Patients transferred to ITU postoperatively will remain sedated and ventilated via an endotracheal tube. However, early extubation is advised, and is an important part of pulmonary recovery. If patients develop significant respiratory failure, or prolonged intubation is expected, a tracheostomy may be required.

B — Preservation of lung function as much as possible is essential, and can be enhanced by chest physiotherapy, which should begin on day 1 postoperatively. Non-invasive positive pressure ventilation (NIPPV) is used in the treatment of acute respiratory failure and can help to avoid endotracheal intubation. There is limited data on its safety, however, and this must therefore be used with caution as high pressures can affect the integrity of the anastomosis. Not exceeding pressures of 10 mmHg can help to prevent this.

C — Cardiac monitoring and goal-directed fluid therapy (GDFT) prevents over-resuscitation and the deleterious effects of excess intravenous fluid administration, such as pulmonary oedema.

D — Adequate analgesia is necessary to allow for early mobilization and to facilitate early extubation. A thoracic epidural is contraindicated in the presence of concurrent sepsis. Paravertebral catheters or intercostal blocks placed at the time of thoracotomy are a good alternative option.

E — Drain output and content should be closely monitored, and removal guided by the operating surgeon and team.

F — Patients should be limited to sips of water only unless they are treated with an Esosponge, in which case they are kept nil by mouth. Early introduction of nutrition is essential for healing and options for this therefore include enteral or parenteral nutrition, whilst the oesophagus is healing.

Enteral nutrition should be in the form of NJ tube feeding, or more definitively, feeding jejunostomy. Percutaneous gastrostomy (PEG) is best avoided due to the risks of reflux of stomach contents and contamination of the healing oesophagus. A jejunostomy can be sited intra-operatively, reducing the need for a further general anaesthetic. Practice has moved towards enteral feeding where possible, with supplementation or replacement via parenteral nutrition only if required. Jejunal feeding should begin early, on day 1 postoperatively. Nutritional requirements should be carefully calculated with experienced dietician input, to promote adequate healing whilst avoiding refeeding syndrome. The benefits of enteral over parenteral feeding are summarised in the Box 1.

Total parenteral nutrition (TPN) is delivered intravenously and can be used to supplement enteral feeding. It also has a role in patients treated with Esosponge, for example, where an NJ tube is difficult to site in the oesophagus alongside the sponge. The main risks are outlined in the Box 2.

M — IV antibiotics should continue with targeted therapy being guided by MC&S results and microbiology advice.

Management of healing oesophagus

Oral contrast studies such as CT with oral and enhanced IV contrast, or barium swallow tests, can be performed to check progress of healing, with integrity of the oesophageal wall indicated by no extraluminal contrast leak, and resolution of

Benefits of enteral versus parenteral nutrition

- Maintenance of intestinal mucosa (prevents bacterial translocation)
- Maintenance of gut-associated lymphoid tissue (GALT)
- Avoids TPN-related immunosuppression
- Reduced SIRS response
- Better glycaemic control
- Efficient use of nutrients
- No line-associated complications

Box 1

Risks of total parenteral nutrition

- Hyperglycaemia
- Hepatic steatosis (LFT derangement)
- Hyperchloraemic metabolic acidosis (amino acids = high chloride content)
- Hypercholesterolaemia
- Refeeding syndrome
- Line related – sepsis/occlusion/insertion, e.g. pneumothorax

Box 2

mediastinal free air or fluid. If a T-tube or stent has been placed, removal is considered at approximately 4 weeks (stents) or 6–8 weeks (T-tube) when satisfactory healing has been demonstrated and patients have tolerated progression to a normal diet. T-tubes should only be removed when a clear fistula tract containing the tube is demonstrated on CT scan. The surrounding tissue should be fully collapsed around the tube with no significant cavities communicating or adjacent to it.

Management of perforated peptic ulcer disease

Patients treated for gastroduodenal perforation may not require the same level of invasive monitoring in an intensive care unit and continuing management should therefore be guided by developing clinical status, as with any critically unwell surgical patient. However, standardization of care now dictates that many patients will be managed in a surgical HDU for a period of time postoperatively.

Conclusion

Perforation of the UGI tract should be categorized into two different groups dependent on anatomical location - oesophageal perforations and gastroduodenal perforations – as the causes, management and prognosis differ significantly. All patients should be adequately resuscitated, with centralization of care if appropriate. Definitive management is highly patient specific and should be guided by the patient's current and premorbid state, after characterizing the clinical picture with a combination of imaging and endoscopy. Adequate postoperative care is fundamental to improving prognosis. ◆

REFERENCES

1 Chirica M, Kelly M, Siboni S, et al. Esophageal emergencies: WSES guidelines. *World J Emerg Surg* 2019; **14**.

2 Quine M, Bell G, McCloy R, et al. Prospective audit of perforation rates following upper gastrointestinal endoscopy in two regions of England. *Br J Surg* 1995; **82:** 530–3.

3 Korn O, Oñate J, López R. Anatomy of the Boerhaave syndrome. *Surgery* 2007; **141:** 222–8.

4 Lau J, Sung J, Hill C, et al. Systematic review of the epidemiology of complicated peptic ulcer disease: incidence, recurrence, risk factors and mortality. *Digestion* 2011; **84:** 102–13.

5 Markar S, Mackenzie H, Wiggins T, et al. Influence of national centralization of oesophagogastric cancer on management and clinical outcome from emergency upper gastrointestinal conditions. *Br J Surg* 2017; **105:** 113–20.

6 Markar S, Mackenzie H, Wiggins T, et al. Management and outcomes of esophageal perforation: a national study of 2,564 patients in england. *Am J Gastroenterol* 2015; **110:** 1559–66.

7 Eso-SPONGE [Internet], Bbraun.co.uk. [cited 31 March 2022]. Available from: https://www.bbraun.co.uk/en/products-and-therapies/wound-closure/endoluminal-vacuum-therapy/eso-sponge.html.

8 Vinnamala S, Menon V, Arasaradnam R, et al. Endoscopic closure of acute Boerhaave's syndrome with an over-the-scope clip. *Endoscopy* 2014; **46:** E481–2.

9 Technology Committee ASGE, Banerjee S, Barth BA, Bhat YM, et al. Endoscopic closure devices. *Gastrointest Endosc* 2012; **76:** 244–51.

10 Cho H, Yoon S, Park S, et al. Distinction between upper and lower gastrointestinal perforation: usefulness of the periportal free air sign on computed tomography. *Clin Imag* 2009; **33:** 332.

11 Tarasconi A, Coccolini F, Biffl W, et al. Perforated and bleeding peptic ulcer: WSES guidelines. *World J Emerg Surg* 2020; **15**.

12 Søreide K, Thorsen K, Harrison E, et al. Perforated peptic ulcer. *Lancet* 2015; **386:** 1288–98.

13 Cellan-Jones CA. Rapid method of treatment in perforated duodenal ulcer. *BMJ* 1929; **1:** 1076–7.

14 Ali W, Ansari M, Rizvi S, et al. Ten-year experience of managing giant duodenal ulcer perforations with triple tube ostomy at tertiary hospital of north India. *Indian J Surg* 2016; **80:** 9–13.

15 Gupta S, Kaushik R, Sharma R, et al. The management of large perforations of duodenal ulcers. *BMC Surg* 2005; **5**.

Practice points

- Morbidity and mortality after oesophageal perforation is reduced with swift diagnosis and transfer to a tertiary oesophago-gastric unit for management.
- The thoracic oesophagus is invested with parietal pleura rather than serosa.
- Boerhaave's syndrome is defined as a spontaneous, full thickness breach of the oesophagus, secondary to a barogenic insult. If the pleura is not breached it would be defined as an intramural oesophageal perforation.
- Endoscopy is the gold standard investigation for diagnosis, anatomical mapping, operative planning, and potential therapeutic interventions for oesophageal perforations.
- Non-operative management of perforated peptic ulcer disease is appropriate only in selected cases. The principal method for operative intervention is by use of a pedicled omental patch repair (Cellan-Jones).