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An Overview of Gastric Perforation

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Abstract

Gastric perforations may be spontaneous or traumatic and the majority of spontaneous perforation is due to peptic ulcer disease. Improved medical management of peptic ulceration has reduced the incidence of perforation, but still remains a common cause of peritonitis. Sub-diaphragmatic air on chest X-ray may be absent and computed tomography scan is a more sensitive investigation in the stable patient. Perforated peptic ulcer is an indication for operation in nearly all cases except when patient is unfit for surgery. Non-operative management has a high incidence of intra-abdominal abscesses and sepsis. The management of traumatic perforation follows the Advanced Trauma Life Support (ATLS) principles.

Keywords: Gastric; Perforation; Aetiology; Management; Operative; Non-operative

Introduction

Gastric perforation may be spontaneous or traumatic. The causes are listed in the majority is from spontaneous perforation due to Peptic Ulcer Disease (PUD) although there are more unusual causes. The two main factors implicated in the aetiology are Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) and Helicobacter pylori (H. pylori). Other factors include smoking, chronic liver disease, chronic renal failure, especially during dialysis and transplantation, and hyperparathyroidism. Perforated peptic ulcer despite antiulcer medication and H. pylori eradication is still the most common indication for emergency gastric surgery associated with high morbidity and mortality.

Aetiology

Benign gastric ulcers occur predominantly in the elderly, on the lesser curve. Ulcers on the greater curve, fundus and in the antrum are more commonly malignant. Gastric ulcers are less common than duodenal ulcers before age 40 years, but become more common in the elderly. Interestingly, in this era of effective treatment of PUD with H. pylori eradication and proton pump inhibitors, gastric cancer remains main cause of gastric outlet obstruction as opposed to peptic ulcer disease. Most ulcers that perforate are sited on the anterior wall of the duodenum or stomach. The release of food and digestive enzymes into the peritoneal cavity initially causes a chemical peritonitis. Secondary bacterial peritonitis evolves later, and as with bleeding ulcers 10% of these patients will die]. The lifetime risk of benign gastroduodenal perforation is 10% in untreated PUD and, 30-50% of ulcer perforations are associated with NSAIDS. 80% of PUD perforations are H. pylori positive and thus eradication is essential. First line triple therapy (Table 1) with PPI, and two antimicrobial agents (Amoxicillin 1g or Clarithromycin 500mg or metronidazole 500 mg, all given bd for 7-14 days eradicates H. pylori in 85% of cases. Treatment failure indicates antibacterial resistance or poor compliance. Gastric volvulus and strangulated hiatus hernia can lead to perforation if all or part of the stomach wall is rendered ischaemic. Although the stomach has a good blood supply, on occasions severe foregut ischaemia can lead to gastric ischaemia and perforation, although such patients are generally unwell before the perforation is manifest.

Spontaneous	Peptic ulceration
	Perforated carcinoma
	Gastric volvulus
	Strangulated hiatus hernia
	Ischaemic disorders
Traumatic	Surgery
	Endoscopic/ PEG complications
	Ventriculoperitoneal (VP) shunt
	VP shunt complication
	Sharp foreign body

	Erosion by battery
	Stab wound
	Blunt abdominal trauma (rare)

Table 1: Causes of gastric perforation.

Clinical presentation

The presentation of gastric perforation is sudden onset severe epigastric pain, peritonism, a board-like abdominal rigidity caused by spasm of the rectii muscles and sepsis, but, may not be specific in the elderly. The perforation is usually unexpected, with no antecedent history. The peritonitis is associated with varying degrees of shock, and severe peritonitis may induce a generalized ileus.. When posterior wall gastric ulcers perforate, they leak gastric contents into the lesser sac which tends to confine the peritonitis and present with less marked symptoms. There are some instances where patients do not have abdominal symptoms or signs, but chest X-rays taken for other reasons indicate a pneumoperitoneum. Perforated peptic ulcer is a common cause as the perforation is frequently sealed by a plug of omentum or another viscus before significant soiling and peritonitis occurs. If perforation is in the thorax as in the case of strangulated Hiatus Hernia (HH), then the patient is likely to have chest symptoms and general signs of severe sepsis, with little or no evidence of peritonitis. Pneumo-peritoneum on erect chest X-ray is absent in 20-30% of cases with gastric perforation, and if there is generalized peritonitis the diagnosis is confirmed at laparotomy or laparoscopy. In a relatively well patient with a sealed perforation and uncertain diagnosis, a Computed Tomography scan (CT) is useful.

Management

The management of gastric perforation may be operative or non-operative. The contributory factors to either of these are the general condition of the patient, poor pre-morbid status, significant co-morbidities, and complicated pathology. Most cases are within the remit of the general surgeon, but perforation due to strangulated HH in chest is best dealt with by a dedicated upper GI surgeon.

Non-operative management

The non-operative management is for (i) the asymptomatic and (ii) the unfit patients. The asymptomatic patients are those whose pneumo-peritoneum has co-incidentally been discovered and the CT is used to investigate the pneumoperitoneum. Early endoscopy is not advisable because of the risk of insufflation disrupting the plug which has sealed any gastroduodenal perforation, but it should be performed at some later stage to exclude malignancy. Treatment with intravenous (IV) Infusion, Naso Gastric Tube (NGT) decompression, broad spectrum antibiotics, analgesia and IV PPIs is instituted, and a Nil By Mouth (NBM) policy is initially adopted. Close observation is important as the development of sepsis or peritonitis may alter treatment radically, CT- guided drainage may be required. Indeed, because of the high incidence of intra-abdominal abscesses and sepsis with non-operative management it has been largely abandoned, even in high risk cases with current advances in anaesthetic approach. This is corroborated by the demonstration that even though small trials show similar results to operative intervention, 30% for whom non-operative treatment was initiated proceeded to surgery, particularly if age is greater than 70. Thus, non-operative treatment is advocated in selected patients who do not have generalized peritonitis or continued duodenal leak, and for those in whom there is an absolute contraindication for surgery. The unfit patients are those with advanced peritonitis and sepsis with significant co-morbidity and / or poor pre-morbid function. They may be deemed unlikely to survive and it is important to discuss the implications with the patient and family. Perforation of an advanced gastric cancer may be another indication for pursuing a conservative course.

Operative management

Operative management entails a quick and easy access via a formal midline laparotomy to identify the site and nature of the pathology. The perforation is usually found on the anterior wall of the duodenum, in proximity to the duodenal bulb. If the perforation is not apparent, mobilization of the duodenum along with inspection of the stomach and jejunum is carried out. With the advent of Proton Pump Inhibitors (PPIs) and peptic ulcer association with H.pylori, definitive ulcer preventing operations, i.e. vagotomy or gastrectomy, have largely been abandoned. Simple closure of perforation by suture plication of ulcer, and reinforcement with an omental patch is the preferred method of dealing with perforation. A routine drain insertion is unproven. Laparoscopic repair using the easily mobilized falciform ligament for patch closure is a reasonable option in selected patients with a history of less than 24 h, no evidence of hypovolaemic shock, and with a perforation of <8-10mm. However, practice depends on expertise and local availability of laparoscopic surger. A meta-analysis showed 85% success in the laparoscopic approach with a reduced wound infection, and pain. However, there was an increase rate of re-operation. The mortality and morbidity is comparable in published series for open vs laparoscopic approach, but there have been no large randomized clinical trials comparing one against the other. Following peritoneal washout with several litres of warm saline to prevent interloop and intra-abdominal abscesses, tissue biopsies from the edge of the gastric ulcer are taken because of the risk of malignancy, even in a benign-looking condition. It may be possible to excise the ulcer, which allows closure of 'healthy' gastric tissue, as well as providing histology, but, a distal gastrectomy should be considered if closure is difficult. The closure with an omental patch as in duodenal perforation is feasible in distal or pre-pyloric ulceration. In the pre-H.Pylori eradication era 80% of patients with simple omental closure alone developed recurrent ulcers. The overall mortality remains between 2 and 8% rising to as high as 30% in the elderly population. The post-operative mortality correlates with pre-operative shock, co-morbidity and perforation>48h, HIV status (CD4 count <200 cells/ul), gastric ulcers. Factors such as shock on admission or delayed surgery were associated with omental patch leakage with increased mortality [41]. Although the best palliation is resection of a perforated gastric tumour at laparotomy, the management at laparotomy is more difficult, especially with regard to decision-making. Even in cases of benign ulceration with perforation where tissue is oedematous and swollen and have appearances of a neoplasm, decision to resect is difficult especially in these usually unstable patients. If any doubt as to how to

proceed, immediate patient safety must come first, with peritoneal lavage and drainage as priority. Perforated HH or gastric volvulus, when part or all of the stomach is in the chest, present extremely difficult scenarios. Surgery in this situation may require thoracotomy, resection, and then a decision made regarding primary or delayed reconstruction. The influencing factors are the time since presentation, degree of mediastinal and pleural soiling, and the general condition of the patient. Although operative management of a perforated duodenal ulcer (usually anterior D1) is generally straightforward, with an omental patch being fashioned after peritoneal lavage, Kocher's manoeuvre to mobilize the duodenum is performed if access to the duodenum is poor. A large perforation may lead to duodenum appearing to disintegrate and various methods are described to deal with this difficult duodenum. Finney pyloroplasty invoves involves fully Kocherizing the duodenum and opening it longitudinally along most of its length. It is then closed transversely in a similar fashion to simple pyloroplasty. More often, if the duodenal ulcer is too large and/or the tissues are too friable to perform a simple closure, a partial gastrectomy may be required. It may be necessary in some cases to exclude or excise the ulcer, close the duodenum distally, and excise the gastric antrum resulting to a Billroth II resection. If no perforation site is evident on initial laparotomy, the posterior surface of the stomach is exposed in the lesser sac. A perforated gastric ulcer needs careful assessment. A proportion (8%) will be malignant and gastric ulcers are more likely to re-perforate after simple closure. Ulcer excision with post operative PPIs or if the expertise is available a partial gastrectomy is advisable. If it is not available it is reasonable to perform simple closure but biopsies must be taken from all four quadrants of the ulcer and medical therapy started. Perforated stomal ulcers are usually managed with omental patch. The usual anatomy will be distorted by the presence of either an antecolic, retrocolic gastroenterostomy or a Roux-en Y anastomosis. An antecolic gastroenterostomy is relatively easy to find as there will be a loop of small bowel anterior to the transverse colon to the stoma but a retrocolic gastroenterostomy may not be immediately apparent as it lies deep to the transverse colon and omentum. Postoperative complications following repair of gastric ulcer perforation include intraperitoneal abscess in the subphrenic space or pelvis, persistence or recurrence of ulcer symptoms especially if post operative H.pylori eradication was avoided, leakage from oversewn perforation and reperforation, and gastric outlet obstruction from scarring of the duodenum. H. pylori is the most important factor for ulcer recurrence following operative repair of perforated duodenal ulcer and merits eradication along with PPI therapy for about 4-6 weeks. Confirmation of eradication with Urea breath test is recommended in patients with resistant ulcer, MALT lymphoma and previous resection of gastric cancer.

Traumatic Perforation

Traumatic perforation follows major trauma. Gastric injury is suspected following penetrating or blunt abdominal injury. The management is along the lines of the Advanced Trauma Life Support (ATLS) principles in which injuries are managed in the order ABCDE: Airway, Breathing, Circulation, Disability (neurological injury) and Exposure, with priorities given to immediate life-threatening injuries. Gastric injury is likely to require surgery for sepsis source control. It is vital to inspect carefully the anterior and posterior gastric wall, GOJ, lesser sac entered with partial gastric mobilisation, and to look for associated hepatic lacerations. Primary closure is generally feasible, but if not possible as in severe trauma, damage control surgery is aimed at haemorrhage control and limiting the soiling of the peritoneum. It entails the acute resection (stapling off) of damaged tissue, drainage and delayed reconstruction at re-look laparotomy at 48h. This will allow the correction of physiology and avoid the lethal triad of death consisting of hypothermia (temp<34 °C), coagulopathy (PT>16secs) and acidosis (pH<7.2). The correction of physiology takes precedence over anatomical correction in the exsanguinating critically ill patient. It is important to remember that acute gastric dilatation is a common postoperative complication of major upper abdominal surgery although it is commonly seen with the gastric autonomic neuropathy of diabetes mellitus, trauma and post splenectomy. It may even cause gastric perforation. From the author's experience, the subtle presentation of left shoulder tip pain and hiccups may lead to it being unrecognized and untreated with a fatal outcome due to vomiting and aspiration. The correction of biochemical abnormalities, such as potassium is essential. The treatment is by large bore Naso Gastric (NG) tube and regular aspiration.

Highlights

- Gastric perforations may be spontaneous or traumatic
- The majority of spontaneous perforation is due to peptic ulcer disease
- Sub-diaphragmatic air may be absent and CT is more sensitive
- Perforated peptic ulcer is an indication for operation in nearly all cases except when patient is unfit
- High incidence of intra-abdominal abscesses and sepsis with non-operative management
- Management of traumatic perforation follow the ATLS principles

Conclusions

The majority of gastric perforations are spontaneous from peptic ulcer disease. The management is mostly operative with simple closure and omental patch reinforcement. This must be followed by H.pylori eradication therapy to prevent recurrence. Primary closure is achievable in traumatic perforation but with the exsanguinating critically ill patient in severe major trauma, damage limitation surgery to correct physiology prior to a later anatomical reconstruction is the principle of management.

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