Peptic Ulcer Disease

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11.1 Introduction

Peptic ulcer disease (PUD) is a significant health care burden. Its presentations range from being asymptomatic, having mild gastrointestinal type symptoms—dyspepsia and bloating—and succumbing to features of haemorrhage or perforation. Ulcers arise as a result of a breach in the muscularis mucosa of the gastroduodenal wall. The gastric juices exert a deleterious effect to the susceptible mucosa. A strong correlation is seen between PUD and Helicobacter pylori (HP) infection. 95% of duodenal ulcers and 80% of gastric ulcers are seen to harbour the pathogen. There is a younger preponderance in developing countries. This is because most children have HP infection before age 10. The prevalence of HP infection is modified by factors such as the use of non-steroidal anti-inflammatory drugs (NSAID), smoking, socioeconomic conditions and age.

11.2 Presentation

Most patients with PUD may be asymptomatic. Epigastric pain and discomfort with complaints of dyspepsia are the most prevailing symptoms. A myriad of other symptoms includes postprandial belching, fullness, early satiety, fatty food intolerance and nausea. The latter symptoms result in an entity known as silent PUD. This is preferentially seen in the Asian continent and is relatively uncommon. The manifestation of symptoms is the result of pyloric channel ulcers with reduced visceral sensation and resultant gastroduodenal dysmotility. Radiation of pain to the back may occur but pain as the primary isolated symptom is atypical with PUD.

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The classic pain of PUD is described as epigastric and or left hypochondrium in location that occurs 2–5 h after a meal. This pain is related to gastric acid that is secreted into an empty viscus. Furthermore, the pain is pronounced between 11 p.m. and 2 a.m. when circadian stimulation of gastric juices is at its peak.

Given the myriad of symptoms, there is a range of PUD presentations. Bleeding related to PUD may present as haematemesis, haematochezia and hypotension if the volume loss is significant. Furthermore, gastric outlet obstruction may occur with pyloric placed lesions. Fistulisation to adjacent viscus structures such as colon and small bowel may occur.

The signs reported with PUD are limited unless complications arise, e.g. haemodynamic instability or succussion splash with delayed gastric emptying.

11.3 Management

PUD management begins with establishing the aetiology whilst simultaneously resuscitating the patient. All patients diagnosed with PUD should undergo HP infection testing—histopathology or breath-based—depending on the nature of presentation. No assessment is complete without an appreciation of drug, alcohol and smoking history primarily NSAID and steroid use, endoscopy and other evaluations to exclude differentials such as Zollinger-Ellison syndrome, malignancy, pancreatobiliary disease.

Following the confirmation, eradication of HP with commencement of antisecretory therapy and plans to withdraw offending factors should be instituted. Maintenance anti-secretory medications should be restricted to the high-risk population as approximately, 90% of ulcers heal with the abolition of HP infection. It is good practice to perform a surveillance gastroscopy in patients with giant, non-benign appearing ulcers without a clear aetiology. Furthermore, follow-up gastroscopy should be offered to all those who have had inadequate sampling at the index gastroscopy, persisting symptoms despite medical therapy, evidence of dysplasia or malignancy and those with risk factors for gastric cancer.

Similar to surveillance endoscopy, the indications for surgery in PUD is small and finite and include bleeding, perforation, obstruction, recurrent disease recalcitrant to medical treatment and suspected malignancy.

The goals of surgery in PUD is to treat and halt progress to complicated ulcer pathology, gastric acid suppression and in doing so promote tissue healing, and prevent and limit recurrences and post-operative complications, respectively.

When dealing with bleeding duodenal ulcers, therapeutic gastroscopy is the mainstay following fluid resuscitation and transfusion. However, with refractory bleeding, the approach is to inspect for the bleeding vessel and control directly by suture ligation or under running the gastroduodenal artery at the superior and inferior aspect of the ulcer via a longitudinal duodenotomy (distal to the pylorus in some instances—pyloroduodenotomy) with a large non-absorbable suture placed in a ‘U’ configuration. This will also aim to ligate the transverse pancreatic branch in its course posterior to the ulcer. Closure of the enterotomy is with the

Heineke-Mikulicz technique, this way not only is mucosal apposition ensured but there is less risk of stenosis. During this time, performing a highly selective vagotomy can be considered but needs to be undertaken with a pyloroduodenotomy. Be cautious in performing a vagotomy in a hemodynamically unstable patient.

With giant duodenal ulcers, partial gastrectomy or antrectomy with Roux-en-Y reconstruction (or gastrojejunostomy) can be considered. In the event of difficulty closing the enterotomy, the duodenal stump can be closed over a T-tube using the Nissen technique or draining it into the second part of the duodenum via a Foley catheter.

When dealing with gastric ulcers, options of management include either a partial gastrectomy with Billroth reconstruction or excisional biopsy of the ulcer with pyloroplasty combined with truncal vagotomy. It should be appreciated that to remove all offending exocrine cells, the gastrin producing cells extend to within 2 cm of the gastroesophageal junction along the lesser curve and at least 35% of the stomach distally needs to be resected. It is important to consult speciality surgical/medical services.

In those with a perforated duodenal ulcer, a modified Graham patch is undertaken following a generous peritoneal lavage. Close the defect primarily where able with absorbable sutures in an orientation that is parallel to the defect as much as possible. This is followed with the application of an omental patch that is secured with non-strangulating absorbable sutures. This is particularly important in the duodenum to prevent unwanted narrowing and possible stricturing. When repairing the duodenum, the risk of leak is perceived to be high and though primary repair may be performed, one should consider the option for gastrectomy and pyloric exclusion (pyloric closure and formation of a gastrojejunal bypass) with gastric drainage for duodenal diversion.

In gastric ulcer perforations, choice of approach extends from patch closure to partial gastrectomy. The fundamental difference between gastric and duodenal ulcers is the risk of malignancy in gastric ulcers. Thus, principles of surgery with gastric ulcers are such that the ulcer should be generously biopsied if not excised.

11.4 Summary

Management of PUD in low resourced or developing countries encompasses medical, endoscopic and surgical arms. Eradication therapy is highly recommended and significantly reduces the burden of PUD.