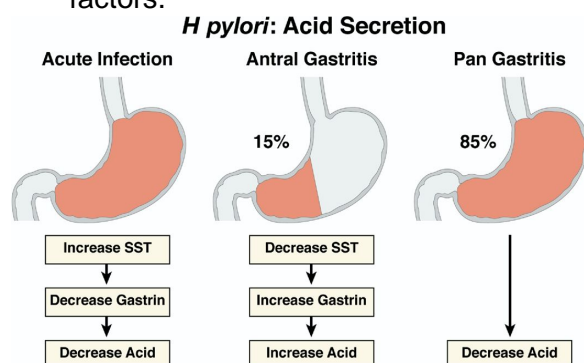


EPIDEMIOLOGY AND PATHOGENESIS

The prevalence of duodenal ulcer disease has a variable geographical distribution and differs amongst population groups. The prevalence in western countries where it was high around the industrialisation period has declined considerably since the latter part of the 20th century. It is still considered to be rare in Africa, especially amongst the black community, but an increase has been noted in those who live in urbanised and industrialised regions.

While several aetiological factors are implicated in peptic ulcer disease the main driving force has now been identified as *Helicobacter Pylori* (HP) and, amongst an increasing elderly population, the common use of non-steroidal anti-inflammatory drugs (NSAIDs) are frequently implicated. Figure 1 shows the current concepts on the effect of HP on the gastric mucosa and acid secretion. It is the antral gastritis group where patients are at risk of developing peptic ulcer disease. Simplistically the pathogenesis of peptic ulcer disease is an imbalance between aggressive (eg. HP, NSAIDs and acid hyper secretion) and protective mechanisms of the integrity of the gut mucosa such as mucus production, mucosal prostaglandins and tissue growth factors.



SST = somatostatin
Figure: Schubert and Peura. Gastroenterology
2008;134: 1842-60

- Upper abdominal pain related to meals, often relieved by food, and coming on again half an hour to 2 hours after meals.
- Nocturnal pain.
- Persistent pain or pain radiating to the back is suggestive but not diagnostic of a penetrating ulcer.
- Associated heartburn is common particularly in the presence of delayed gastric emptying.
- Anorexia, vomiting and weight loss point to delayed gastric emptying.

Untreated, the natural history is characterised by a relapsing and remitting pattern. Patients go into remission for varying periods which can be weeks, months, seasonal or for many years.

SPECIAL INVESTIGATIONS

- **Barium-meal** is now seldom used in the diagnosis of peptic ulcer disease.
- **Endoscopy** confirms the diagnosis and allows for biopsy of the stomach to confirm the presence of H.py/or/ by a RUT test and, to rule out malignancy.
- **Breath and serological tests for HP** in selected cases.
- **Serum gastrin** levels are done in cases with suspected Zollinger Ellison Syndrome. Levels of more than 500 pg/ml in the presence of peptic ulcer disease are highly suspicious of the diagnosis. In the absence of an associated peptic ulcer, atrophic gastritis (eg. Pernicious anaemia) associated with achlorhydria may give similar high levels. PPI therapy may give slightly raised gastrin levels.

Medical therapy

The goals of peptic ulcer management are:

- The relief of ulcer symptoms.
- The healing of the ulcer (and prevention of complications).
- The prevention of recurrence.

Proton Pump Inhibitors (PPIs) combined with eradication of HP when present, is now the standard treatment regime for both duodenal and gastric ulcers. The first line treatment is a PPI for 2 weeks and a one week antibiotic course consisting of amoxicillin (1gram BD) and metronidazole (400mgBD) or clarithromycin (500mg BD). Patients with gastric ulcers and NSAIDs induced ulcers should take PPIs for a month in addition to eradication therapy. More complex regimes are given when first line treatment fails.

Successful HP eradication therapy can now achieve a very high cure rate for peptic ulcer disease. Patients with gastric ulcer should undergo repeat endoscopy to ensure ulcer healing and to rule out malignancy. NSAID associated ulcers represent a special risk group. These patients are often elderly, have co-morbid disease which amplifies the risk of the complications associated with the use of these drugs. When patient are unable to stop NSAIDs, maintenance PPI therapy is recommended.

PEPTIC ULCER SURGERY

With the high ulcer cure rate achieved with HP eradication therapy, definitive operations to reduce acid secretion such as highly selective vagotomy are no longer necessary for uncomplicated ulcer disease. Surgery is therefore reserved in the main for complications such as bleeding, perforation, stenosis and, occasionally, for persistent penetrating ulcers.

COMPLICATIONS

Haemorrhage

The management of patients with bleeding peptic ulcers are covered in detail in chapter on GIT bleeding

Perforation

Incidence: Perforations are seen in 10% - 30% of duodenal ulcers. The peak incidence used to be in the 20-40 year age group with a male predominance but an increasing number of elderly patients are seen with this complication due to NSAIDs use.

Pathology: The site of perforation is usually the anterior wall of the first part of the duodenum. General chemical peritonitis (bile, acid and pancreatic juice) follows in the majority of cases. After ± 6 hours, contamination occurs from organisms in the oesophagus and stomach resulting in bacterial peritonitis. The perforation may seal off spontaneously by omentum or the under surface of the liver.

Clinical presentation

- In 30-40% of cases the history of dyspepsia is absent or of less than 3 months duration.
- The onset is acute in the majority of cases. In some this may be preceded by epigastric pain with increasing severity over a 24-48 hour period. The patient can usually recall the exact time and place when it occurred. The pain is severe and generalised, but when there is early sealing of the perforation symptoms and signs may be confined to the epigastrium, or when it leaks along the right paracolic gutter - settles in the right iliac fossa mimicking acute appendicitis. The patient is acutely distressed with initial neurogenic shock. Hypovolaemia and severe metabolic derangement is seen in delayed cases.
- Abdominal examination: board-like rigidity with absent bowel sounds. Gross distension occurs later on as a result of accumulation of gas and fluid in the peritoneal cavity and/or

the development of ileus.

Special investigations

- Chest (erect) and abdomen X-ray will show air under the diaphragm in 80% of cases (Figure 2)
- Serum amylase may be raised.
- Gastrografin swallow is indicated only in doubtful cases or when conservative therapy is considered.

NB: Endoscopy is contra-indicated.



Figure 2 Chest X- ray showing bilateral air under the diaphragm.

Treatment

- Resuscitation.
- Analgesics for neurogenic shock.
- Nasogastric tube.
- Hypovolaemia resuscitation.
- Broad spectrum antibiotics.

Surgery

Primary closure with an omental patch is the operation of choice followed by HP eradication if tested positively (Figure 3).

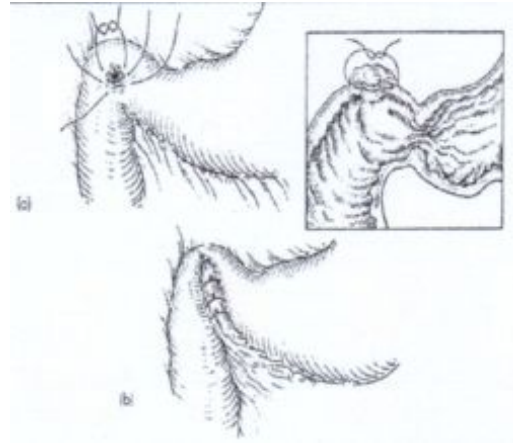


Figure 3

Conservative therapy, with nasogastric suction, intravenous fluids and antibiotics, is only indicated in patients with localized / sealed perforation and those too ill for surgical intervention.

The mortality remains high if there is a delay in the diagnosis and management, particularly in elderly patients with co-morbid diseases.

DUODENAL STENOSIS

Incidence - 10%

Pathology

1. Large penetrating ulcers with associated inflammation and oedema.
2. Healed ulcer with fibrosis.

With the slow onset of benign stenosis, compensatory muscular hypertrophy occurs.

Ultimately decompensation sets in with gastric dilatation and stasis.

Metabolic effects

Gastric fluid contains an average of 100 meq/l of Cl, 45 meq/l of Na⁺ and 10 meq/l of potassium. Initially excessive loss of chloride occurs with a fall in plasma chloride and a rise in plasma bicarbonate. This alkalotic tendency is compensated for by renal excretion of sodium bicarbonate. A large sodium deficit develops due to

losses in the vomitus and from excessive excretion in the urine. In advanced cases the urine becomes acidic as a result of compensatory excretion of H⁺ and K⁺ to conserve sodium losses.

End result

- Severe dehydration.
- Raised urea and haematocrit
- Low serum chloride, sodium and potassium.
- Alkalosis (serum) and intra-cellular acidosis in advanced cases.
- Decrease in serum ionised calcium which can present with tetany.
- Urine: alkalotic and in advanced cases acidotic.

Clinical Features

- Long-standing history of dyspepsia and loss of weight.
- Anorexia, nausea and vomiting of undigested food - usually non bile stained.
- Metabolic and nutritional derangements.

On examination:

- Dehydration.
- Upper abdominal distension.
- Visible peristalsis (left to right).
- Succussion splash

Special Investigations:

- X-ray abdomen - dilated stomach with food.
- Ba meal - hold-up at the duodenum with a dilated stomach (Figure 4)
- Endoscopy to exclude carcinoma.

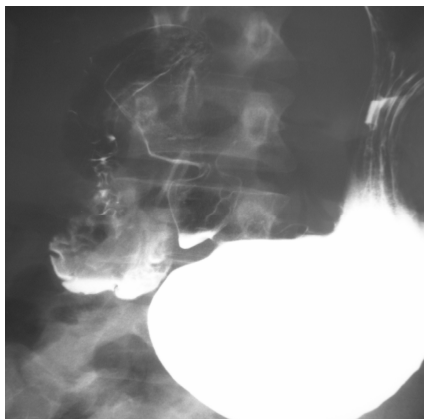


Figure 4

Treatment

- Rehydration with normal saline and correction of potassium deficit. Patients can be severely dehydrated and may require up to 10 litres of fluid to achieve adequate rehydration.
- Hyperalimentation or enteral feeding via a feeding tube placed endoscopically beyond the stenosis may be required.
- Stomach washouts with a wide bore tube.
- Peptic ulcer therapy.
- Endoscopic dilatation may avoid or defer surgery in selected high risk patients.
- Surgery is required in the majority of patients. There is no urgency and adequate decompression of the stomach is important to prevent post operative stasis.

The objective of surgery is to overcome the obstruction which usually entails some form of pyloroplasty (Figure 5).

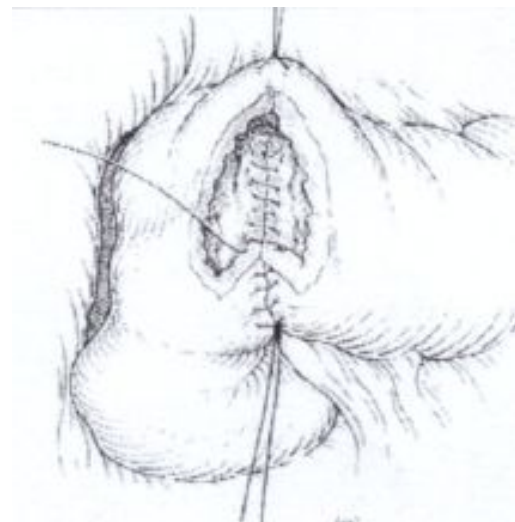


Figure 5

GASTRIC ULCER

Epidemiology

Gastric ulcers occur more commonly in lower socio-economic population groups.

Like duodenal ulcer disease, a decline in the incidence has been noted in most western countries. Gastric ulcers are now more commonly seen in elderly patients and are often related to analgesic abuse.

Types of gastric ulcers

Traditionally there are three types of gastric ulcers, namely; prepyloric (<2 cm from the pylorus), combination of a duodenal and gastric ulcer (the former is usually inactive when patients present with a gastric ulcer), and ulcers >2 cm from the pylorus on the lesser curve, usually above the angulus. The latter ulcers usually occur at the junction zone of the antrum and parietal cell mass. Therefore, the higher the ulcer on the lesser curve the more extensive the chronic gastritis, and then smaller the parietal cell mass, hence the low acid output associated with these ulcers. Ulcers occurring elsewhere in the stomach should raise the suspicion of analgesic abuse or gastric carcinoma.

Clinical Presentation

Dyspepsia

Periodicity, as in duodenal ulcers, is also a feature of gastric ulcers. Pain precipitated by meals occurs more commonly than with duodenal ulcers. Patients are therefore often afraid to eat. Weight loss is not uncommon, particularly when associated with anorexia, nausea and vomiting. Epigastric fullness, mild cramps and belching may also form part of the symptom complex. However, the history is often unhelpful to distinguish this from a duodenal ulcer.

Complications

- Bleeding.
- Perforation
 - into the peritoneal cavity.
 - into the lesser sac.
 - sealed off by the liver.
- Penetration into the pancreas.
- Gastric outlet obstruction is seen more commonly with prepyloric ulcers
- Malignant transformation is questionable.

Special Investigations

Barium meal - typically an ulcer crater on the lesser curve of the stomach or an ulcer crater without irregular raised margins and thickened folds. Malignancy can, however, not be excluded. Endoscopy with 4-quadrant biopsies should be routinely performed to exclude a carcinoma and, should be repeated to confirm healing. Tests for HP should also be done.

Treatment

- See medical management under duodenal ulcer disease.
- As with duodenal ulcer disease definitive surgery (eg. gastrectomy) for uncomplicated disease is seldom required today with HP eradication therapy.
- Bleeding ulcers: See bleeding ulcer section
- Perforations: Primary closure and biopsy is preferred when (a) the perforation is small, or (b) in the presence of a long-standing perforation with excessive contamination. For large ulcers and those where malignancy is suspected, a standard Billroth I gastrectomy is recommended (Figure 6).

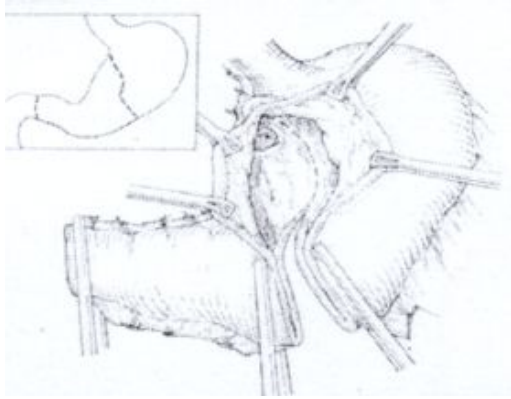


Figure 6



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