Peptic Ulcer Disease

Gastric acid secretion

- Secretion - Parietal cell by the proton pump

- Factors are involved to production of gastric acid

Histamin - acts via the H2-receptor.

Vagus nerve
Gastrin
} Stimulate histamine production

- Secretin - inhibit gastric acid secretion

Gastric mucus and the gastric mucosal barrier

- Essential to the integrity of the gastric mucosa
- Layer of mucopolysaccharides
- Produced - mucus-producing cells of the stomach and pyloric glands
- Physiological barrier to protect the gastric mucosa from mechanical damage and effects of acid and pepsin.

Damaged by

- Bile
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Alcohol
- Trauma
- Shock

Peptic ulcers

- Gastric ulcer
- Duodenal ulcer

- Common sites for peptic ulcers are the first part of the duodenum and the lesser curve of the stomach
- Also occur on the stoma following gastric surgery, the oesophagus and Meckel's diverticulum

- In general, the ulcer occurs at a junction between different types of epithelia

- The ulcer occurring in the epithelium least resistant to acid damage

- **Zollinger-Ellison syndrome (ZES)** - rare disorder, can cause gastric or duodenal ulcers (usually multiple) from excessive acid secretion

Gastric ulcer

- Less common
- Chronic gastric ulcers are much more common on the lesser curve
- Other - junction of the fundus and antrum
- Usually 0.5-2.5 cm in diameter
- Epigastric pain shortly after meal
- Pain is not relived by food or antacids
- Gastric ulcers may become malignant

Duodenal ulcer

- More common
- Both a posterior and an anterior ulcer - ‘kissing ulcers’
- Anteriorly placed ulcers tend to perforate
- Posterior duodenal ulcers tend to bleed
- More than 95% of duodenal ulcers are found in the first part of the duodenum
- Most are less than 1 cm in diameter
- Epigastric pain 2-3 hours after meal
- Food or antacids relieve the pain
- Can develop develop gastric outlet obstruction

Aetiology

- H pylori infection
- Drugs - NSAIDs

Most common

- Lifestyle factors
- Severe physiologic stress
- Hypersecretory states (uncommon)
- Genetic factors

Clinical features

- Epigastric pain
- Gnawing or burning sensation
- Dyspepsia, including belching, bloating, distention, and fatty food intolerance
- Heartburn
- Chest discomfort

- Hematemesis or melena resulting from gastrointestinal bleeding
- Hematochezia(rarely)
- Symptoms consistent with anemia (eg, fatigue, dyspnea)
- Sudden onset of symptoms may indicate perforation.
- NSAID-induced gastritis or ulcers may be silent

Investigations

- If the diagnosis of peptic ulcer disease is suspected,

1. Full blood count
2. Liver function tests (LFTs)
3. Levels of amylase and lipase

- Radiographic and endoscopic confirmation
- Testing for *H pylori* infection is essential in all patients with peptic ulcers

Test for *H. pylori* infection

1. Rapid urease test
2. Histopathology
3. Culture

- Rapid urease tests are considered the endoscopic diagnostic test of choice
- Urea breath tests detect active *H pylori* infection by testing for the enzymatic activity of bacterial urease

Endoscopy

- Diagnostic test in the evaluation of patients with suspected peptic ulcer disease

- Highly sensitive for the diagnosis of gastric and duodenal ulcers
- Allows for,

1. Biopsies and cytologic brushings
2. Antral biopsies for a rapid urease test and/or histopathology

Gastric ulcers

1. Discrete mucosal lesions
2. Punched-out
3. Smooth ulcer base - whitish fibrinoid exudate
4. Solitary
5. Well circumscribed

- Benign ulcers -smooth, regular, rounded edge with a flat smooth base and surrounding mucosa that shows radiating folds
- Malignant ulcers - irregular heaped-up or overhanging margins

Duodenal ulcers

1. Well-demarcated break in the mucosa
2. Extend into the muscularis propria

- Chest X-ray - free abdominal air when perforation is suspected
- Angiography - massive gastrointestinal bleed in whom endoscopy cannot be performed
- Fasting serum gastrin level - screen for Zollinger-Ellison syndrome

Treatment

- Uncomplicated peptic ulcers are treated medically
- Life style modifications (cessation of cigarette smoking)

- H2-receptor antagonists
- Proton pump Inhibitors (PPI)

- Zollinger–Ellison syndrome - long term with proton pump inhibitors

Eradication therapy

- To eradicate H. pylori infection
- Triple therapy (PPI, amoxicillin, and clarithromycin) for 7-14 days

- Quadruple therapy (bismuth, PPI, tetracycline and a nitroimidazole) for10-14 days

Medical Management of NSAID Ulcers

- NSAIDs should be immediately discontinued
- 6-8 weeks of therapy with a PPI - complete healing of a duodenal ulcer
- NSAID and switch to less toxic NSAIDs, such as the newer NSAIDs or COX-2 inhibitors

Surgical treatment

- Aimed

Reducing gastric acid secretion in the case of

Gastric ulceration - removing the diseased mucosa

- **For gastric ulcers-**

Billroth I gastrectomy

For deuodenal ulcers -

- Gastrectomy
- Gastroenterostomy
- Truncal vagotomy and drainage
- Selective vagotomy and drainage
- Highly selective vagotomy
- Truncal vagotomy and antrectomy

Complications

- Perforated peptic ulcer
- Bleeding peptic ulcers
- Obstruction
- Gastrocolic fistula
- Gastric malignancy

prognosis

- When the underlying cause of peptic ulcer disease is addressed, the prognosis is excellent
- NSAID-related ulcers - the incidence of perforation 0.3% per patient year
- Obstruction - 0.1% per patient year
- 1 death per 100,000 cases
- Emergency operations for peptic ulcer perforation carry a mortality risk of 6-30%