

# Peptic Ulcer Disease



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# 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 relieved 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
  - Lifestyle factors
  - Severe physiologic stress
  - Hypersecretory states (uncommon)
  - Genetic factors
- Most common



# 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



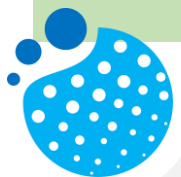
# Endoscopy

- 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)
- H<sub>2</sub>-receptor antagonists
- Proton pump Inhibitors (PPI)
- Zollinger–Ellison syndrome - long term with proton pump inhibitors

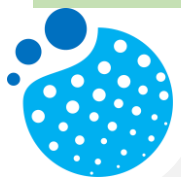


# Eradication therapy

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



- Quadruple therapy (bismuth, PPI, tetracycline and a nitroimidazole) for 10-14 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 duodenal 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%

