Helicobacter pylori and Peptic Ulcer
Objectives

- Definition of peptic ulcer
- Etiology
- Epidemiology
- *Helicobacter pylori*
- Pathogenesis
- Signs and Symptoms
- Diagnosis
- Complications
- Treatment
A peptic ulcer is a sore (ulceration) on the lining of the stomach or duodenum, occurring in areas exposed to acid and pepsin and most often caused by *Helicobacter pylori* infection.

Some ulcers are caused by long-term use of nonsteroidal anti-inflammatory agents (NSAIDs), like aspirin.

In a few cases, cancerous tumors in the stomach or pancreas can cause ulcers.

Peptic ulcers are not caused by stress or eating spicy food, but these can make ulcers worse.
Peptic Ulcers: Gastric & Dudodenal
Other factors

- Steroid therapy
- Smoking
- Excess alcohol intake
- Genetic factors
- Hyperparathyroidism
- Severe physiologic stress (Burns, CNS trauma, Surgery, Severe medical illness)
Epidemiology

*H. pylori* infection occurs worldwide

Prevalence varies greatly among countries and population groups

20 – 50% prevalence in middle age adults in industrialised countries

>80% prevalence in middle age adults in developing countries: may reflect poorer living conditions
**Helicobacter pylori**

- Members of the genus *Helicobacter* are curved or spiral organisms.
- Gram-negative
- They have a rapid, motility resulting from multiple polar flagella.
- Microaerophilic,
- Produces urease.
Helicobacter pylori
Pathogenesis

- The organism survives in the mucus layer that coats the epithelium and causes chronic inflammation of the mucosa.
- Although the organism is noninvasive, it recruits and activates inflammatory cells.
- Urease released by *H. pylori* produces ammonia ions that neutralize stomach acid favoring bacterial multiplication.
- Ammonia may also both cause injury and potentiate the effects of a cytotoxin produced by *H. pylori*.
• Infects mucosa of stomach > inflammatory response > gastritis > increased gastrin secretion > gastric metaplasia > damage to mucosa > ulceration

• Increased risk of developing gastric adenocarcinoma, and gastric B-cell lymphoma (mucosa-associated lymphoid tumors).

• [Note: *H. pylori* infection is found in more than 95 percent of duodenal ulcer patients and in nearly all patients with gastric ulcers who do not use aspirin or other nonsteroidal anti-inflammatory drugs, both risk factors for gastric ulcers.]
Pathogenesis of *H. pylori*

1. *H. pylori* penetrate the mucous layer lining the stomach’s epithelium, attracted to the chemotactic substances hemin and urea.

2. *H. pylori* recruit and activate inflammatory cells. They also release urease that cleaves urea, producing NH₃ that neutralizes stomach acid in its vicinity.

3. *H. pylori* cytotoxin and the ammonia produced by its urease cause destruction of mucous-producing cells, exposing underlying connective tissue to stomach acid.
Signs and Symptoms

- Abdominal discomfort is the most common symptom. This discomfort usually
  - comes and goes for several days or weeks
  - occurs 2 to 3 hours after a meal
  - occurs in the middle of the night when the stomach is empty
- weight loss
- poor appetite
- bloating
- Nausea and vomiting
Diagnosis

• **Invasive diagnosis:**
  involve gastric biopsy specimens obtained by endoscopy. *H. pylori* can be detected in such specimens histologically, by culture, or by a test for urease.

• **Non-invasive diagnostic test:**
  - Urea Breath Test
  - Culture (stool or biopsy)
  - Serology (detecting antigen of bacretia in stool or blood)
  - Occult blood
Urea breath tests

Involve administering radioactively labeled urea by mouth. If *H. pylori* are present in the patient's stomach, the urease produced by the organism will split the urea (1–2 hours) to CO$_2$ (radioactively labeled and exhaled) and NH$_3$.]

**Characteristic of *H. pylori***

*H pylori* is oxidase positive and catalase positive, has a characteristic morphology, is motile, and is a strong producer of urease.
Complications

- Perforation & Penetration—into pancreas, liver and peritoneal space
- Peritonitis
- Bowel obstruction, Gastric outflow obstruction.
- Bleeding--occurs in 25% to 33% of cases and accounts for 25% of ulcer deaths.
- Gastric CA
Treatment

- the recommended primary therapies for H. pylori infection include: clarithromycin, and amoxicillin, or metronidazole (clarithromycin-based triple therapy) for 14 days
- Lansoprazole 30 mg + clarithromycin 500 mg + amoxicillin 1 g × 10 days.
- Omeprazole 20 mg + clarithromycin 500 mg + amoxicillin 1 g × 10 days.
- Esomeprazole 40 mg + clarithromycin 500 mg + amoxicillin 1 g × 10 days.
Thanks for listening