



CHRONIC GASTRITIS

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GROUP-6,SEM-5.**

What is chronic gastritis?

- Chronic gastritis is a condition in which the stomach lining is damaged long-term, often due to infection by *H. pylori*. Chronic gastritis does not usually cause indigestion or pain, but severe damage may result in anemia due to vitamin B12 deficiency.

ETIOLOGY

- Chronic gastritis has a number of possible causes, some of which overlap with the possible causes of acute gastritis.
- bacterial infection, most commonly with *Helicobacter pylori* bacteria
- excessive alcohol consumption
- bile reflux
- drug use (certain recreational and over the counter drugs can irritate the stomach linings if used frequently)
- stress
- radiation
- certain illnesses, such as [diabetes](#) or [kidney failure](#)
- a weakened immune system



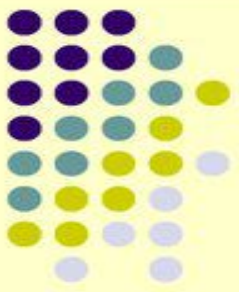
Types of chronic gastritis

Type A Gastritis (Autoimmune gastritis).

- antibodies against parietal cells and intrinsic factor.
- other autoimmune diseases .
- gastric atrophy
- hypo- or achlorhydria.

Type B Gastritis (Helicobacter pylori-related).

- excessive secretion of acid (hypersecretory gastritis)
- associated peptic ulcer



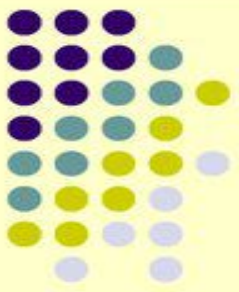
Types of chronic gastritis

Type AB Gastritis (environmental)

- gastric atrophy
- caused by environmental factors.

Type C Gastritis (Chemical)

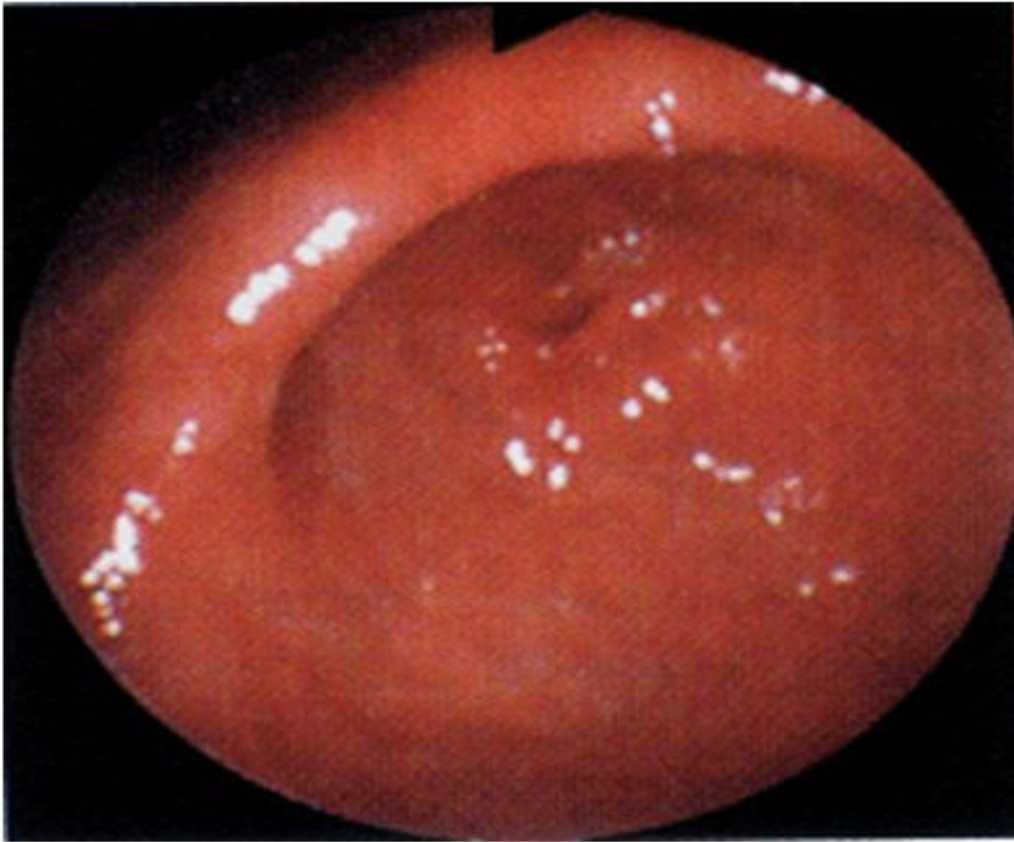
- due to reflux of alkaline duodenal contents, pancreatic secretions, and bile into the stomach.
- in persons after GIT surgery, with gastric ulcer, gallbladder diseases.



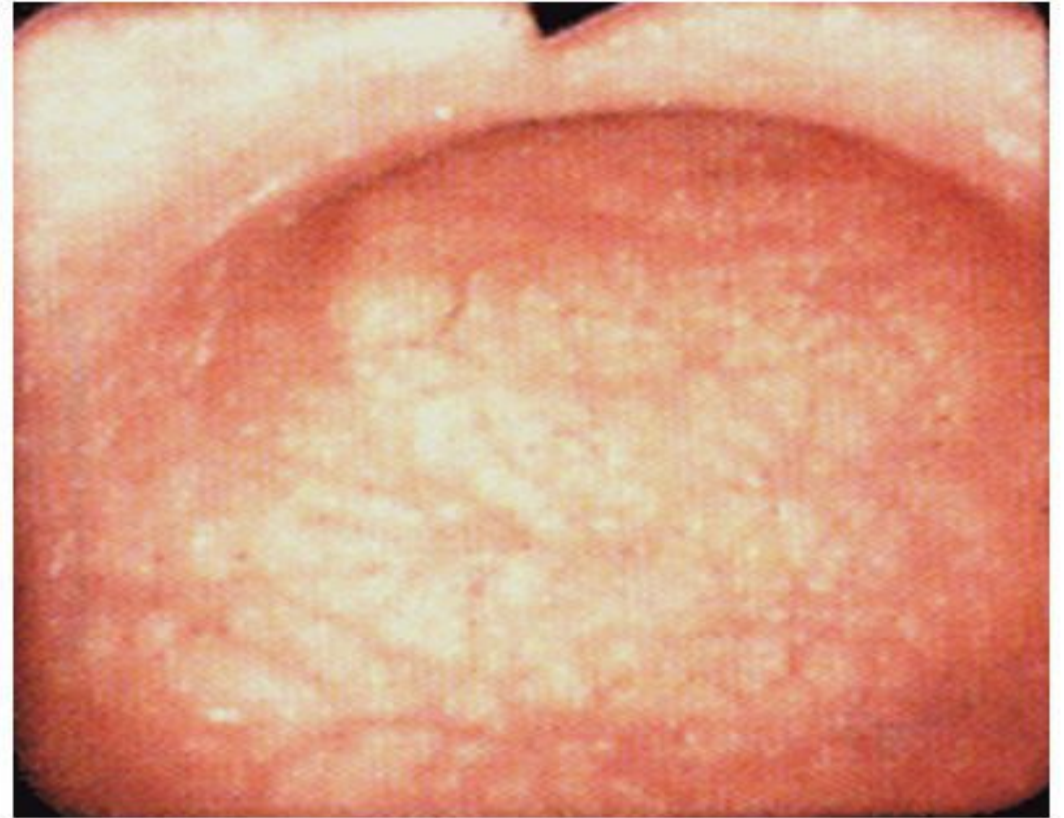
Clinical manifestations

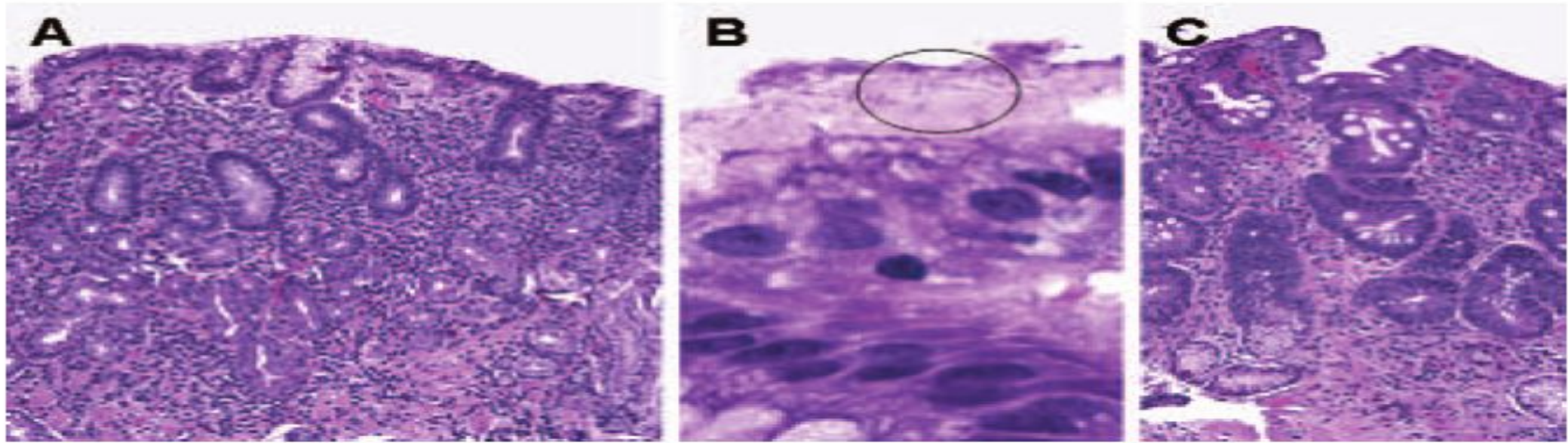
- affection of one or all layers of stomach
- remissions and exacerbation
- healing with scar formation
- stomach discomfort and pain.
 - periodicity of pain (on empty stomach).
 - recurrence of pain.
 - pain is relieved by food or antacids.

Type B (Chronic antral gastritis)



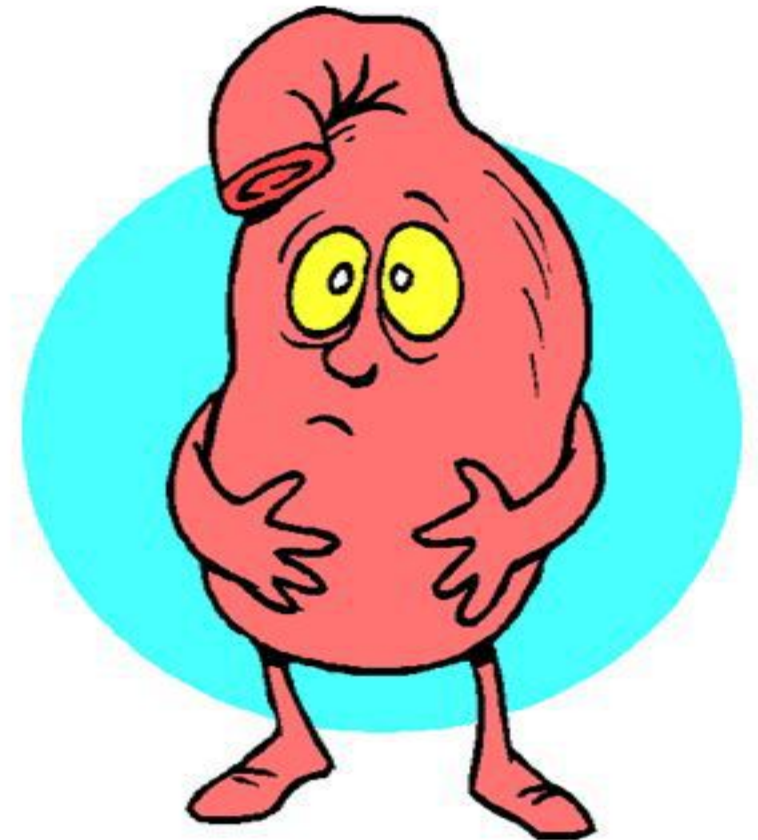
Type A (autoimmune gastritis)





Helicobacter pylori–associated chronic active gastritis. A, Chronic inflammation oriented toward the surface of the mucosa. Neutrophils cannot be seen at this magnification (original magnification 10) but were identified with high power (hematoxylin-eosin stain). B, Circled area shows H pylori organisms within the mucus layer close to the surface of gastric epithelial cells (hematoxylin-eosin, original magnification 40). C, Gastric mucosa with intestinal metaplasia (hematoxylin-eosin , original magnification 20).

- Signs and Symptoms
- Abdominal pain with a burning or gnawing sensation
- Heartburn
- Indigestion (dyspepsia)
- Belching
- Nausea
- Vomiting
- Poor appetite
- Weight loss



What are the risk factors for chronic gastritis?

- Your risk for chronic gastritis increases if your lifestyle and dietary habits activate changes in the stomach lining.
- It may be useful to avoid:
 - high-fat diets
 - high-salt diets
 - [smoking](#)
- Long-term consumption of alcohol can also lead to chronic gastritis.



Complications

Hemorrhage

- bleeding from granulation tissue
- erosion of an ulcer into an artery or vein

Hematemesis or melena.

Acute hemorrhage – signs of circulatory shock depending on the amount of blood loss.



Complications

Obstruction

- edema, spasm or contraction of scar.
- epigastric fullness, vomiting of undigested food.

Perforation

- GI contents enter the peritoneum (peritonitis),
- ulcer penetrate adjacent structures (pancreas),
- severe pain radiating into the back.

Helicobacter pylori
Infection:

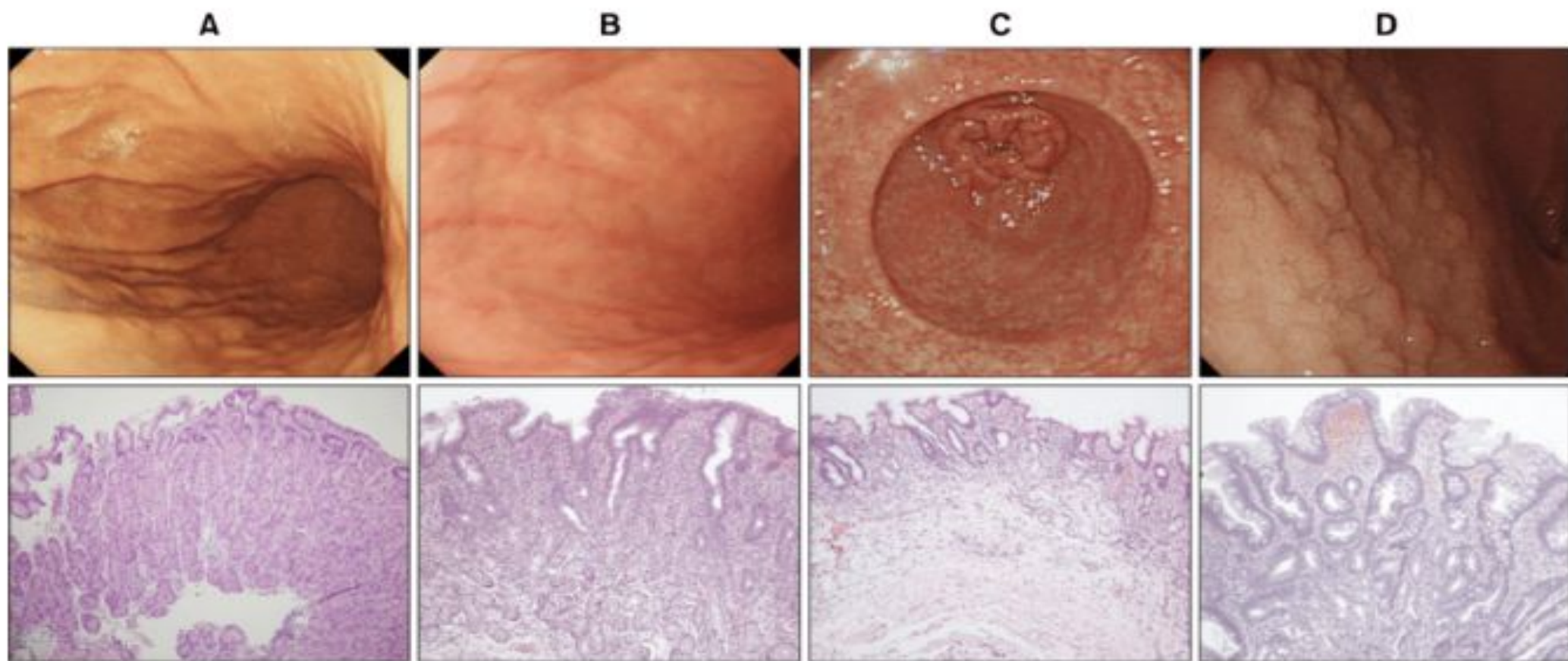
Multifocal
atrophic gastritis



Automimmune
gastritis:

Corpus-fundus
restricted atrophic
gastritis





Classification of gastritis (H&E, $\times 400$). (A) Normal, (B) superficial gastritis, (C) atrophic gastritis, (D) intestinal metaplasia. Adapted from Schindler. 61

How is chronic gastritis diagnosed?

- a test for the bacteria that cause stomach ulcers
- a stool test to look for stomach bleeding
- a blood count and an **anemia** test
- an **endoscopy**, in which a camera attached to a long tube is inserted into your mouth and down into your digestive tract

TREATMENT

- **Combinations of antibiotics and proton pump inhibitors.**
- **Individuals with *H. pylori* gastritis usually improve after treatment, although relapses can occur after incomplete eradication or re-infection.**
- **Prophylactic and therapeutic vaccine development is still at an early stage of development.**

PEPTIC ULCER

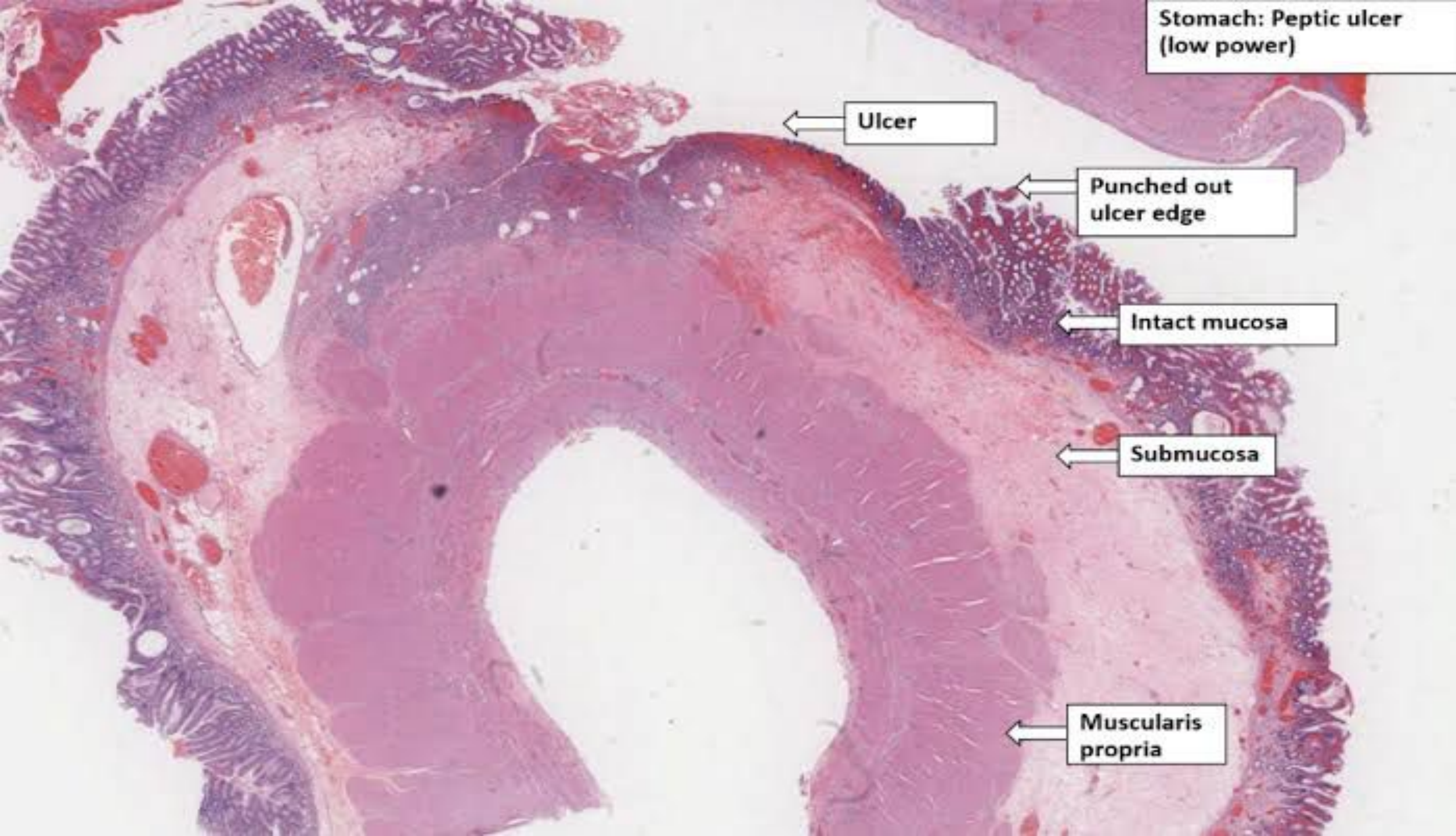
What are peptic ulcers?

Peptic ulcers are sores that develop in the lining of the stomach, lower esophagus, or small intestine. They're usually formed as a result of inflammation caused by the bacteria [H. pylori](#), as well as from erosion from stomach acids.

ETIOLOGY

- ***Helicobacter pylori* (*H. pylori*)**, a type of bacteria that can cause a stomach infection and inflammation
- frequent use of aspirin (Bayer), ibuprofen (Advil), and other anti-inflammatory drugs (risk associated with this behavior increases in women and people over the age of 60)
- [smoking](#)
- drinking too much [alcohol](#)
- [radiation therapy](#)
- [stomach cancer](#)
-

Stomach: Peptic ulcer (low power)



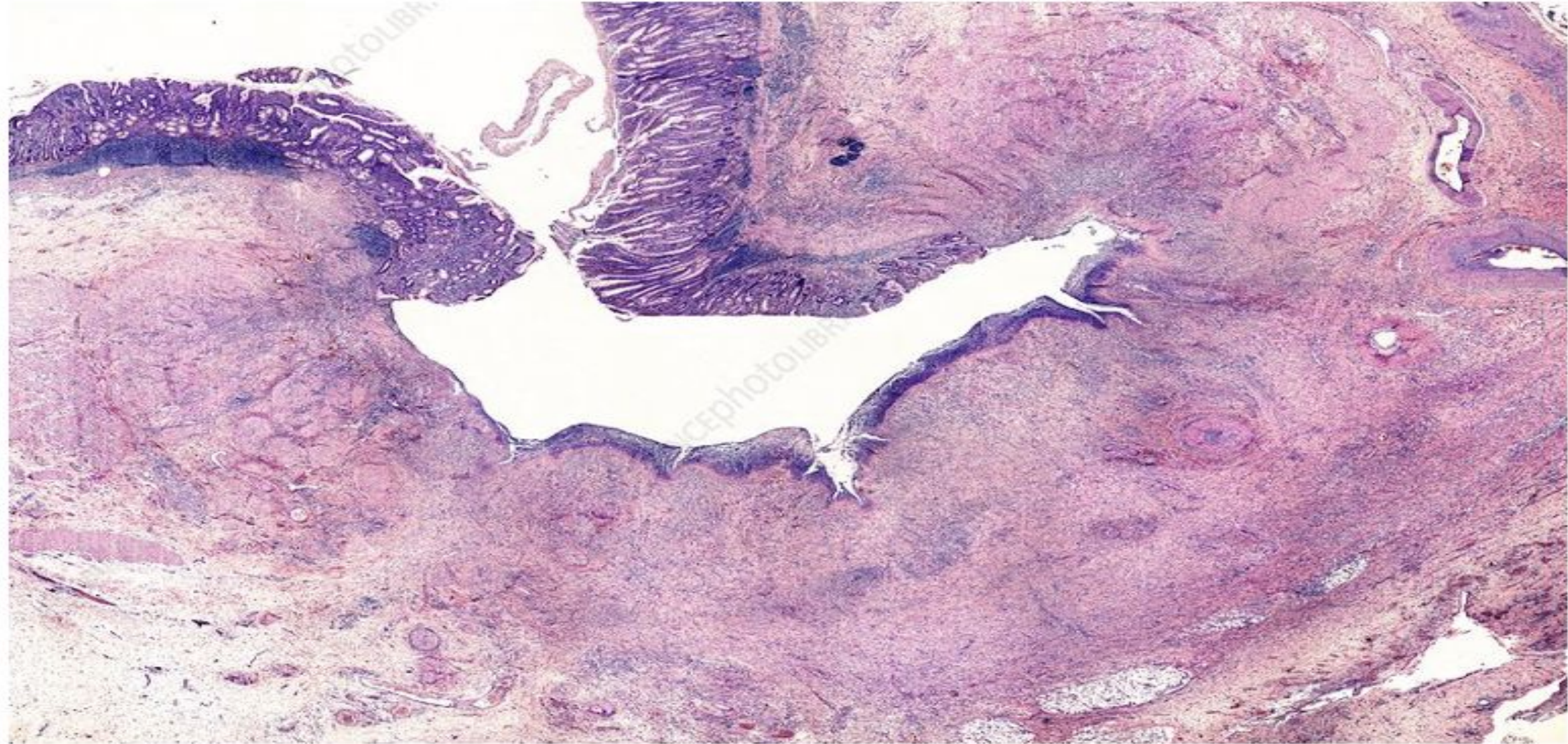
← Ulcer

← Punched out ulcer edge

← Intact mucosa

← Submucosa

← Muscularis propria

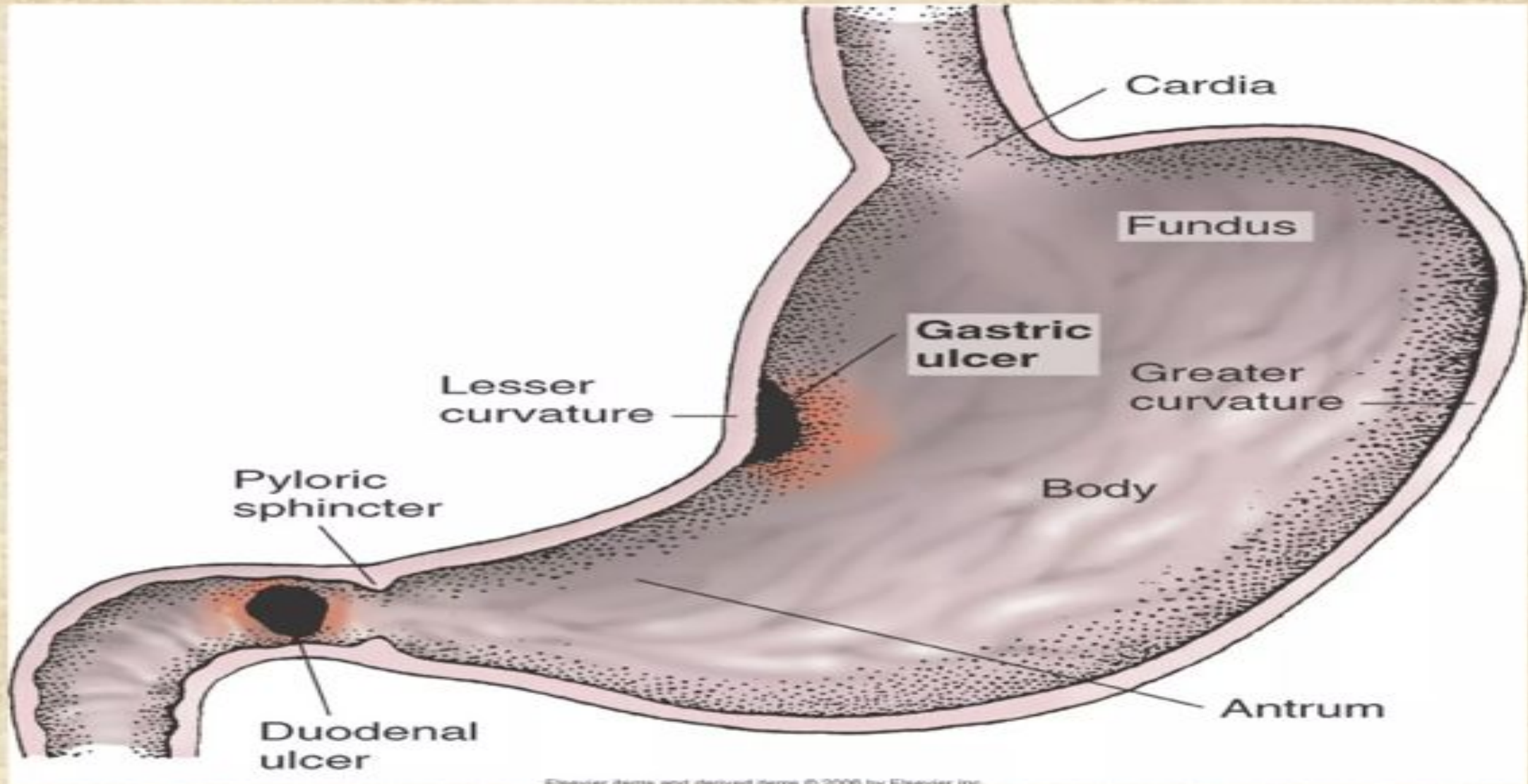


Peptic ulcer, light micrograph

CLINICAL MANIFESTATIONS

- The most common symptom of a peptic ulcer is burning abdominal pain that extends from the navel to the chest, which can range from mild to severe. In some cases, the pain may wake you up at night. Small peptic ulcers may not produce any symptoms in the early phases.
- Other common signs of a peptic ulcer include:
 - changes in appetite
 - [nausea](#)
 - [bloody or dark stools](#)
 - unexplained weight loss
 - [indigestion](#)
 - [vomiting](#)
 - [chest pain](#)

Gastric and Duodenal Ulcers



Difference between Clinical Features of Gastric Ulcer and Duodenal Ulcer

Gastric Ulcer

- Pain after food intake.
- Periodicity less common.
- Hematemesis more common.
- Weight loss.
- Equal in both sexes.

Duodenal Ulcer

- Pain before food intake.
- Periodicity more common.
- Melena more common.
- Weight gain occurs.
- Common in males.

Complications of Peptic Ulcers

- Hemorrhage
 - Blood vessels damaged as ulcer erodes into the muscles of stomach or duodenal wall
 - Coffee ground vomitus or occult blood in tarry stools
- Perforation
 - An ulcer can erode through the entire wall
 - Bacteria and partially digested food spill into peritoneum=peritonitis
- Narrowing and obstruction (pyloric)
 - Swelling and scarring can cause obstruction of food leaving stomach=repeated vomiting

DIAGNOSTIC TEST

- Esophagogastrodeuodenoscopy (EGD)
 - Endoscopic procedure
 - Visualizes ulcer crater
 - Ability to take tissue biopsy to R/O cancer and diagnose H. pylori
 - Upper gastrointestinal series (UGI)
 - Barium swallow
 - X-ray that visualizes structures of the upper GI tract
 - Urea Breath Testing
 - Used to detect H.pylori
 - Client drinks a carbon-enriched urea solution
 - Exhaled carbon dioxide is then measured

INVESTIGATION

- Stool examination for fecal occult blood.
- Complete blood count (CBC) for decrease in blood cells.

MANAGEMENT

- LIFE STYLE MODIFICATION
- HYPOSECRETORY DRUG THERAPY
- *H. pylori* ERADICATION THERAPY
- SURGERY

LIFE STYLE MODIFICATION



Discontinue NSAIDs



Smoking cessation.



Alcohol cessation.



Stress reduction.

Hyposecretory Drugs

- Proton Pump Inhibitors
 - Suppress acid production
 - Prilosec, Prevacid
- H₂-Receptor Antagonists
 - Block histamine-stimulated gastric secretions
 - Zantac, Pepcid, Axid
- Antacids
 - Neutralizes acid and prevents formation of pepsin (Maalox, Mylanta)
 - Give 2 hours after meals and at bedtime
- Prostaglandin Analogs
 - Reduce gastric acid and enhances mucosal resistance to injury
 - Cytotec
- Mucosal barrier fortifiers
 - Forms a protective coat
 - Carafate/Sucralfate
 - cytoprotective

THANK YOU