Pathology of the stomach-part 1

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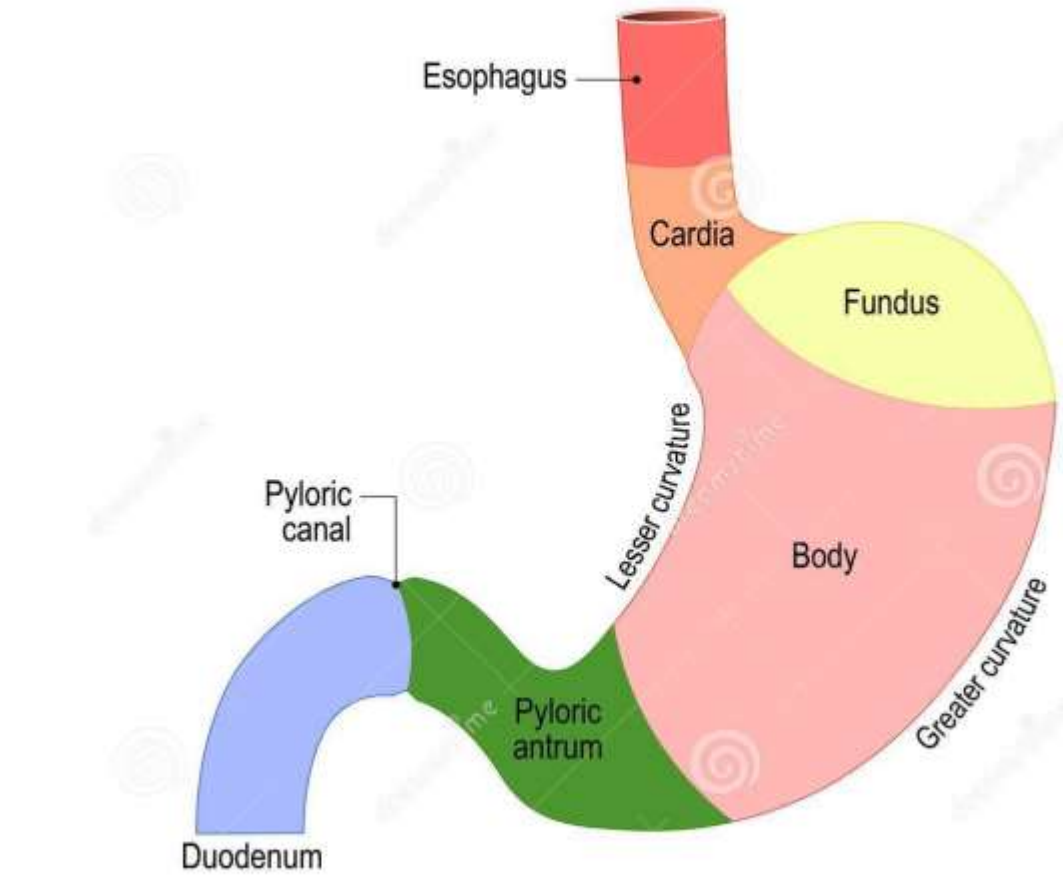
overview

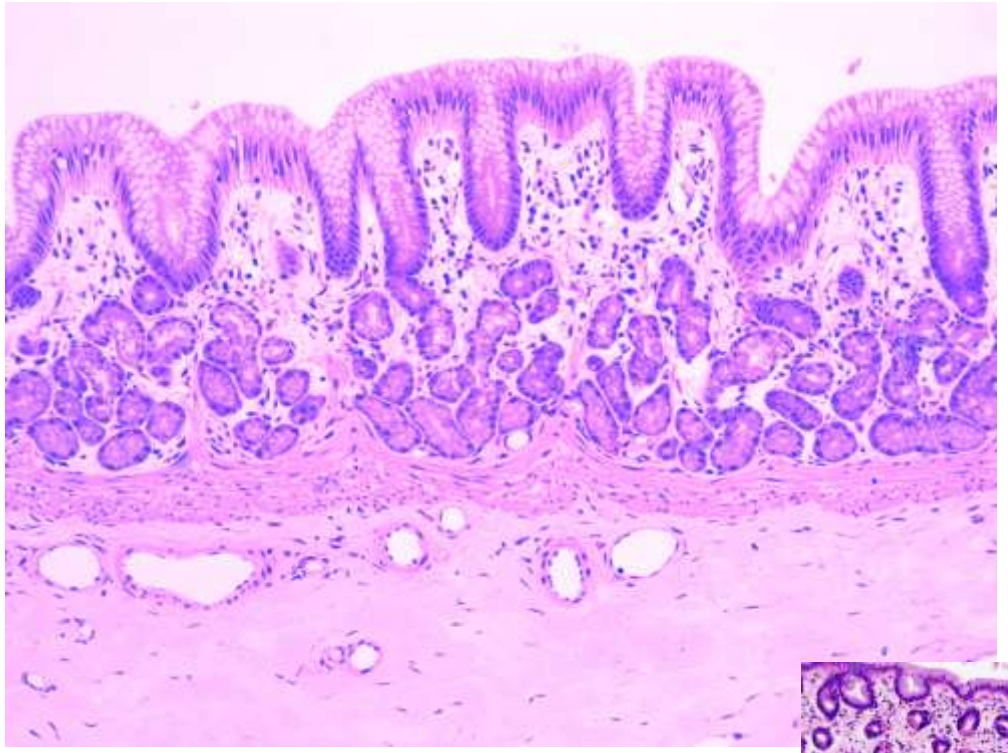
- Gastric diseases:
- 1-Inflammatory.
- 2-Neoplastic.

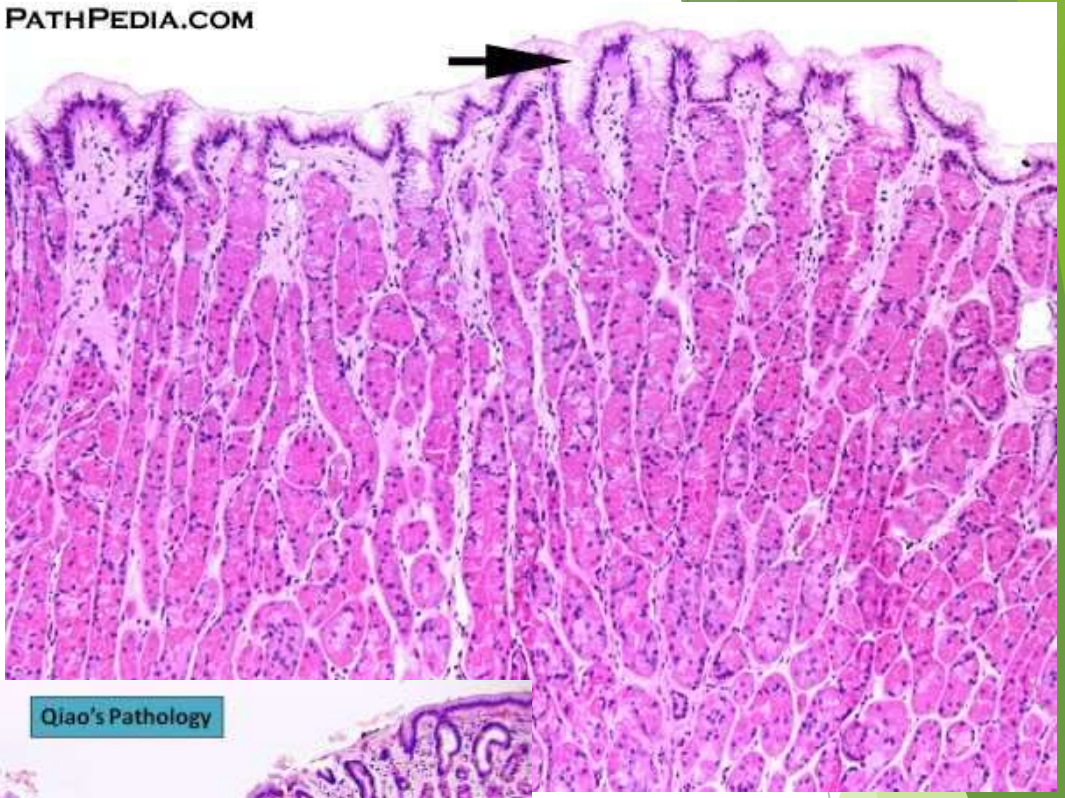
- Stomach parts: cardia, fundus, body, antrum, pylorus.

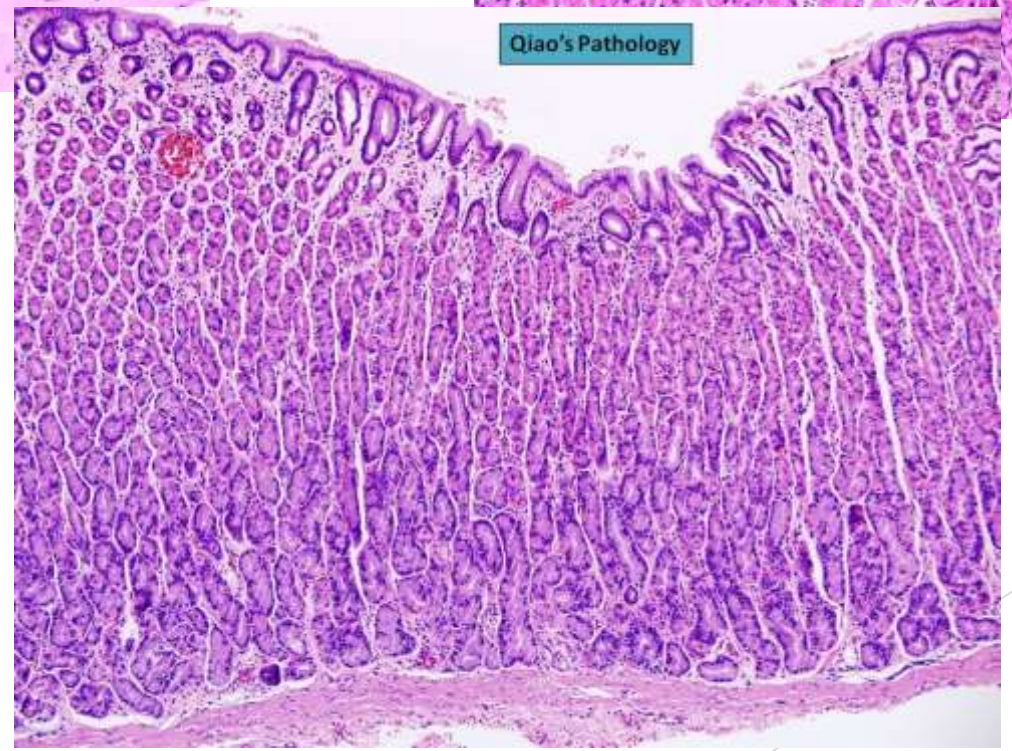
- Cardia: mucin secreting foveolar cells.
- Body and fundus: parietal cells (HCL) and chief cells (pepsin).
- Antrum: neuroendocrine G cells (gastrin)

Sections of human the stomach









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Inflammatory conditions

- Acute gastritis.
- Chronic gastritis.
- Acute gastric ulcer.
- Chronic peptic ulcer.

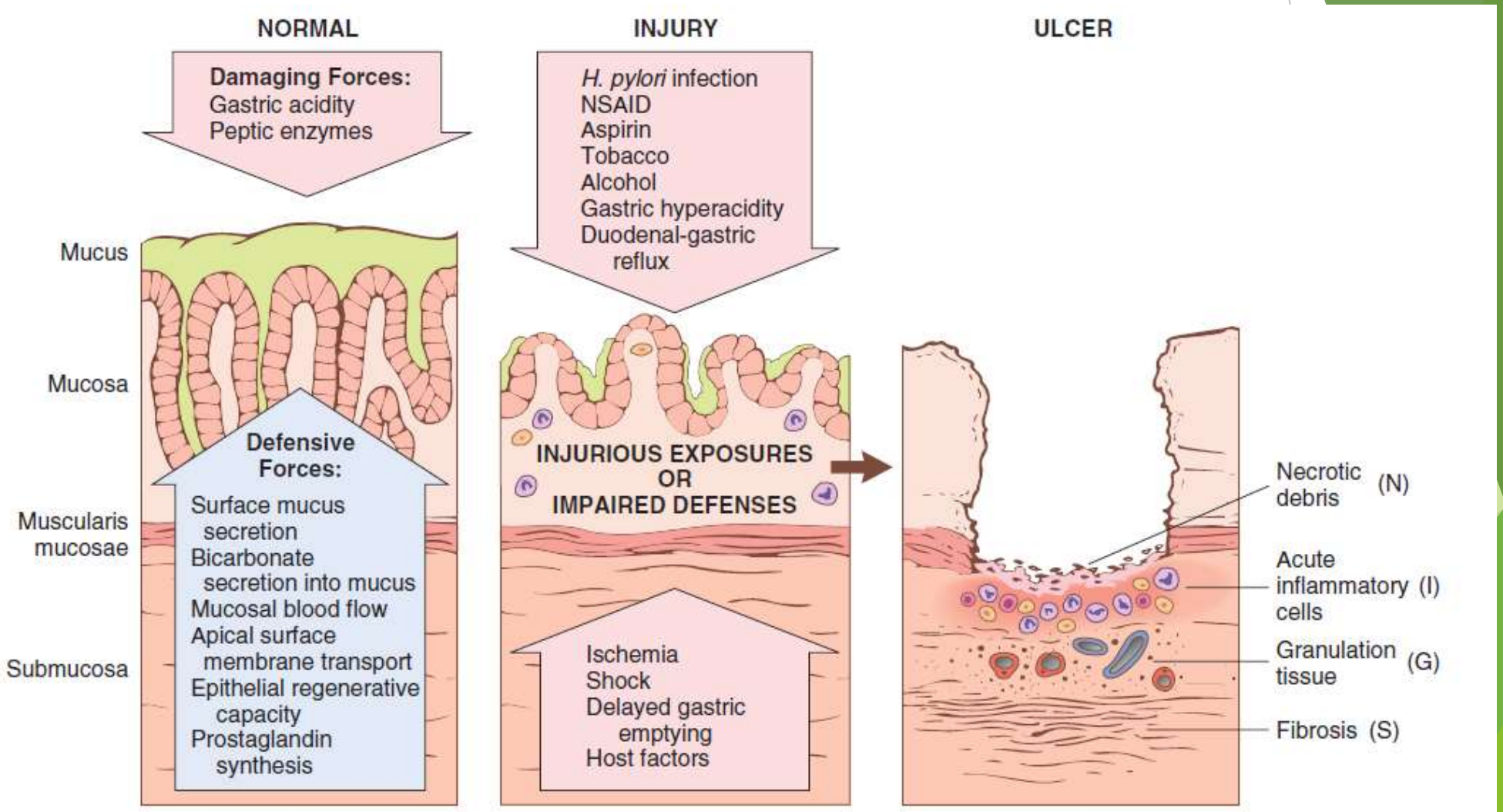
ACUTE GASTRITIS and gastropathy

- **Acute gastritis**: Mucosal injury, neutrophils present.
- **Gastropathy**: regenerative, no inflammation.

- **Causes:**
- NSAIDs, alcohol, bile, and stress-induced

- **Clinical features:**
- Asymptomatic, epigastric pain, nausea, vomiting.

Pathogenesis



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Pathogenesis

- Imbalance between protective and damaging forces

- **Main causes:**
- **NSAIDs (COX1 and COX2 inhibitors)**
- **Uremic patients (ammonia inhibit bicarbonate transport)**
- **H pylori (urease produces ammonia)**
- **Old age (reduced mucin and bicarbonate secretion)**
- **Hypoxia**
- **Harsh chemicals**, (acids or bases) (direct epithelial injury)
- **Alcohol, NSAIDs, radiation therapy (direct mucosal damage)**
- **Chemotherapy (inhibit DNA synthesis and cellular renewal)**

prostaglandins E2 and I2:

- Stimulate nearly all of the defense mechanisms including
- Mucus and bicarbonate secretion,
- mucosal blood flow
- Epithelial restitution.
- Risk for development of NSAID- induced gastric injury is greatest with nonselective inhibitors, but selective COX2 inhibition, can also result in gastropathy or gastritis.

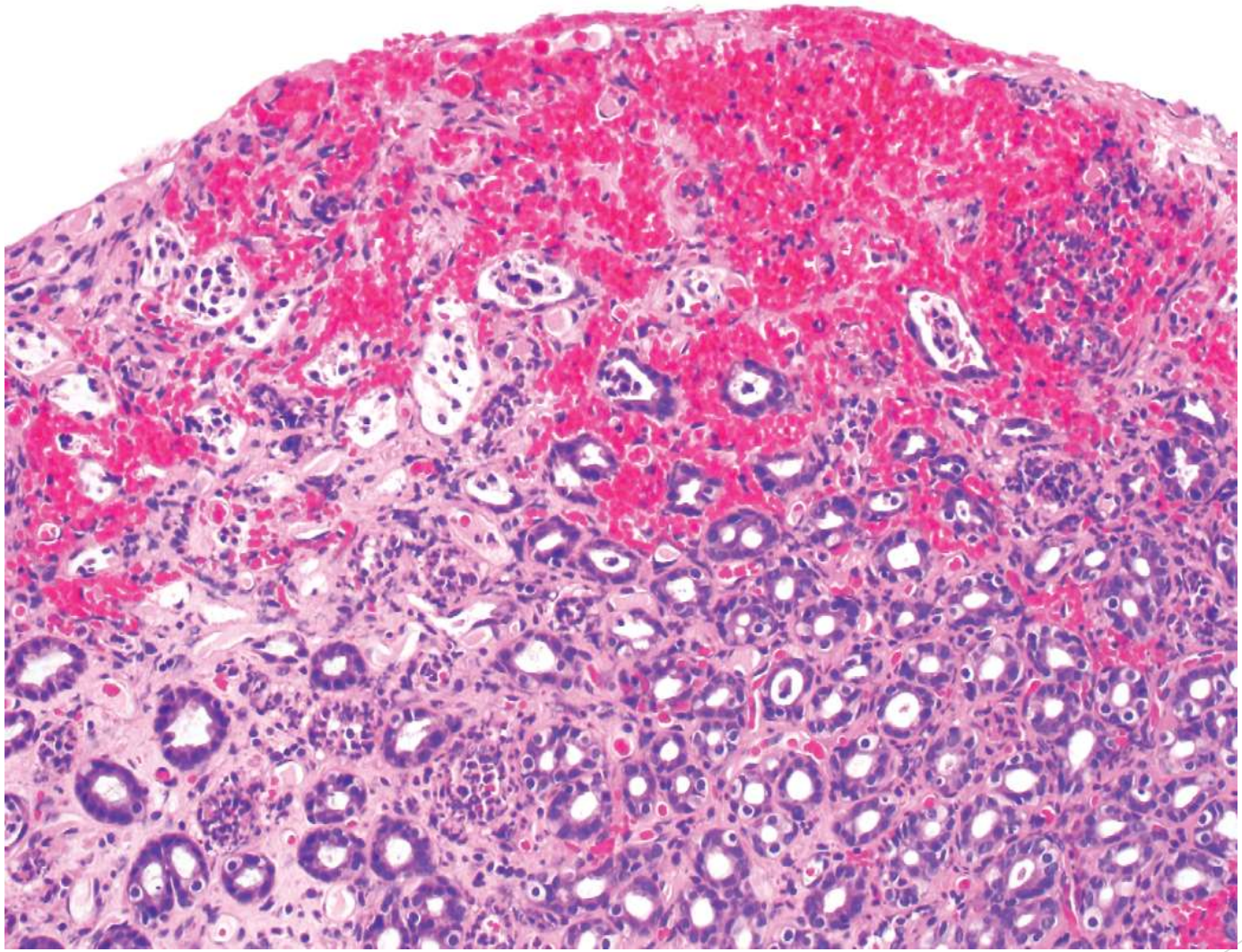
MORPHOLOGY

- Hyperemia (redness).
- Edema and slight vascular congestion
- Neutrophils, lymphocytes, and plasma cells are not prominent.
- Intact surface epithelium.
- Advanced: Erosions and hemorrhage, acute erosive hemorrhagic gastritis.

- Active inflammation (neutrophils) is not necessary.

ACUTE GASTRITIS

B



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Stress-Related Mucosal Disease acute gastric ulcers

- Severe physiologic stress:

- Trauma
- Extensive burns
- Intracranial disease
- Major surgery
- Serious medical disease
- Critically ill patients

Acute gastric ulcers:

- ***Stress ulcers****:* critically ill patients with shock, sepsis, or severe trauma.
- ***Curling ulcers****:* proximal duodenum , severe burns or trauma.
- ***Cushing ulcers****:* stomach, duodenum, or esophagus, intracranial disease, high risk of perforation.

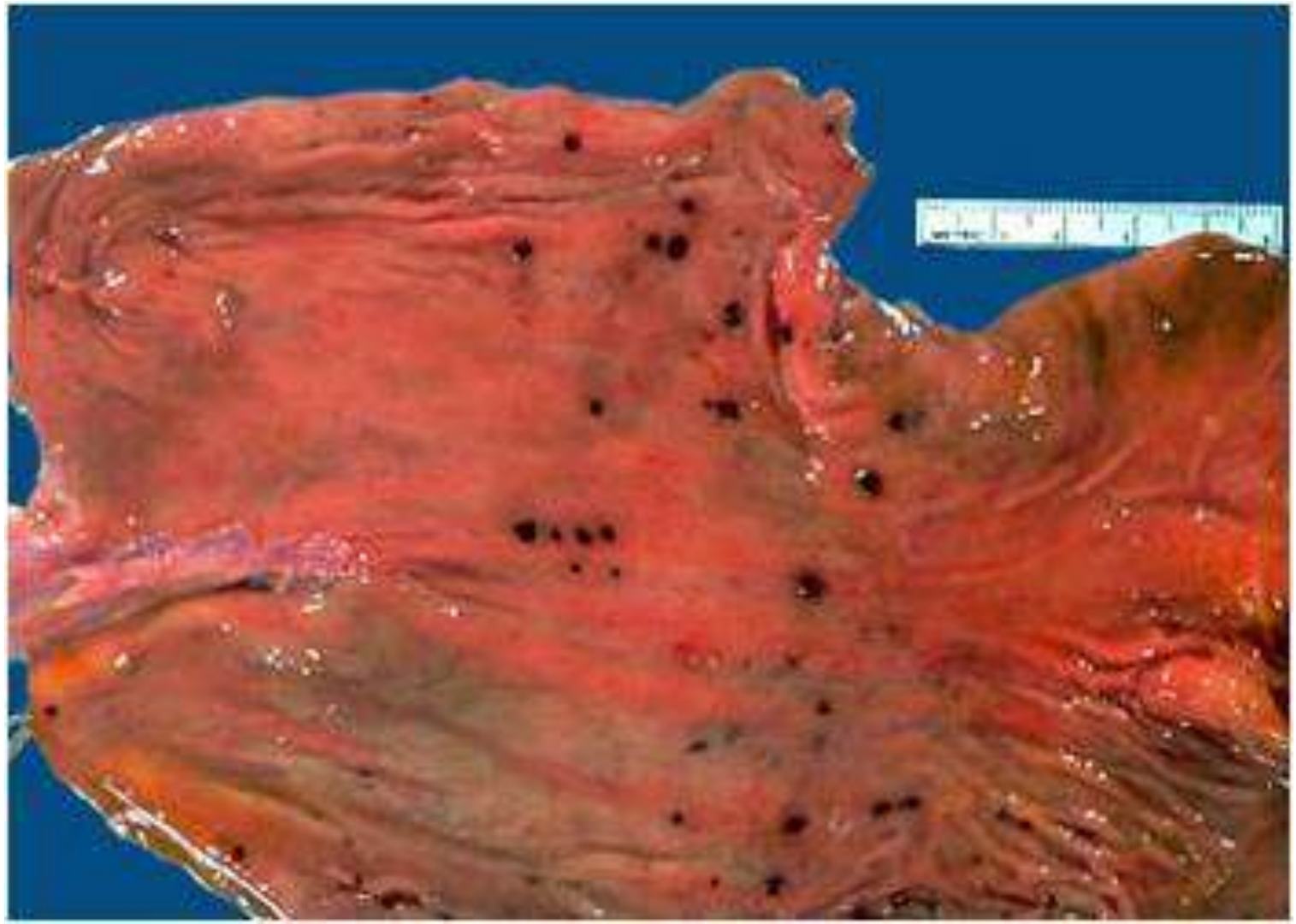
Pathogenesis

- **Stress ulcers:**
- Mostly due to Local ischemia caused by.
- Systemic hypotension.
- Splanchnic vasoconstriction (stress induced).
- Systemic acidosis (lower intracellular PH).
- COX2 expression is protective.

- **Cushing ulcers:**
- Direct vagal stimulation, acid hypersecretion.

MORPHOLOGY

- Acute ulcers are rounded and typically less than 1 cm in diameter
- Shallow to deep.
- Ulcer base brown to black
- Anywhere in stomach
- Usually, multiple.
- Normal adjacent mucosa
- No scarring
- Healing with complete epithelialization occurs days or weeks after removal of injurious factors



Clinical features

- Nausea, vomiting,
- Melena
- Coffee -ground hematemesis
- Perforation complication.

- Prophylaxis with proton pump inhibitors
- Outcome depends on severity of underlying cause.

CHRONIC GASTRITIS

- *Causes:*

- ***Helicobacter pylori associated gastritis: most common.***
- ***Autoimmune atrophic gastritis: less than 10% of cases.***

- **Less common**
- Chronic NSAID
- Radiation injury
- Chronic bile reflux.

Clinical features

- Nausea and upper-abdominal discomfort
- Vomiting
- Hematemesis uncommon.

- Less severe but more prolonged symptoms.

Helicobacter pylori Gastritis

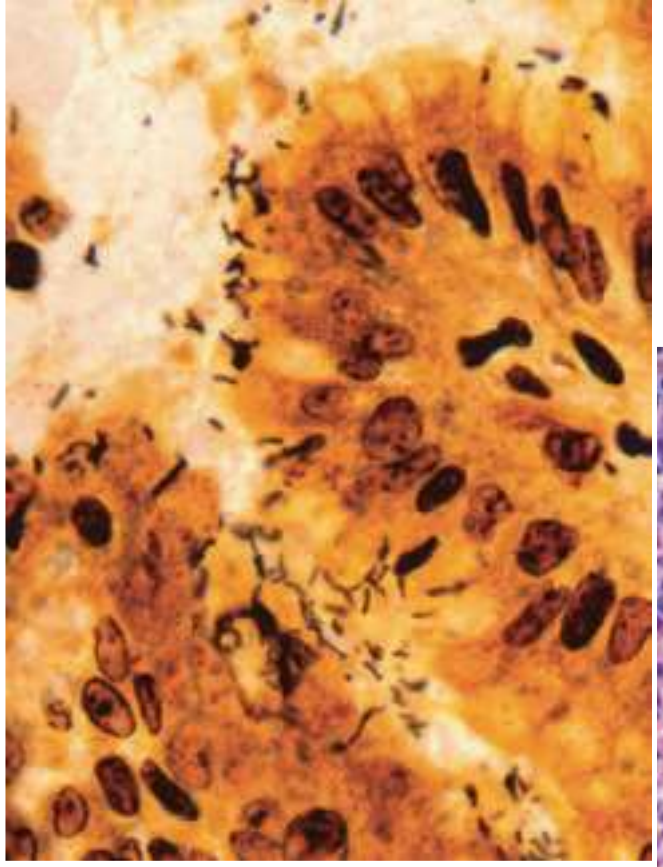
- Discovery of the association of H.pylori *w*ith peptic ulcer disease was a revolution.
- Spiral or curved, G-ve, bacilli.
- Underlying cause for almost all duodenal ulcers.
- Majority of gastric ulcers or chronic gastritis.
- Acute infection is subclinical.
- ***Antral gastritis with increased acid production >> peptic ulcer***
- **Pangastritis if severe with hypochlorhydria.**
- **Intestinal metaplasia and increased risk of gastric cancer.**

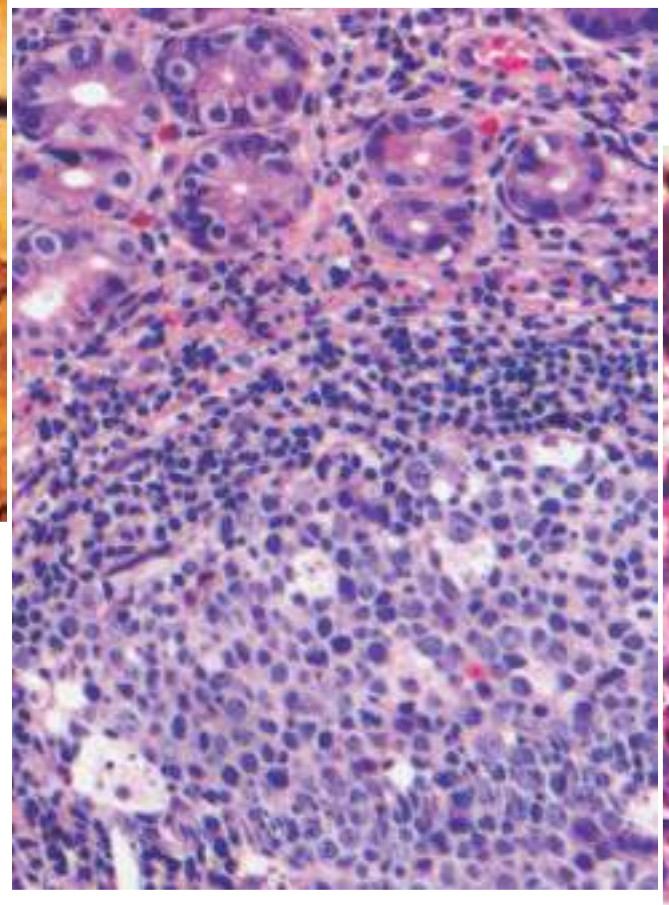
- Poverty, household crowding, limited education, poor sanitation
- Infection is typically acquired in childhood, persists to adult-life.

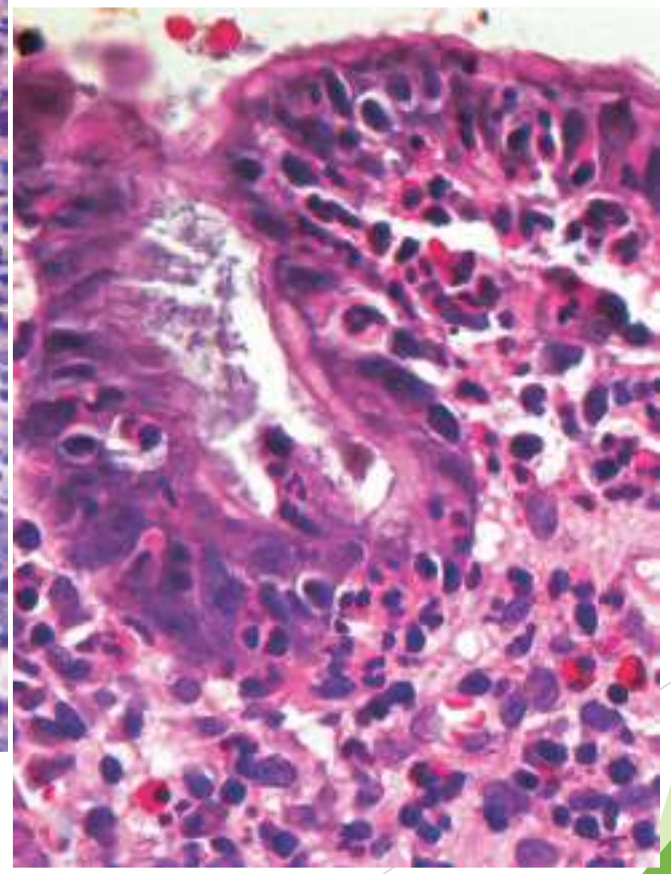
- **Pathogenesis:**
- H.pylori adapted to live in the mucus layer, non-invasive, by
- **Flagella**: allow motility.
- ***Urease****: split urea to ammonia*, protect bacteria from acidic pH.
- ***Adhesins****:* bacterial adherence to foveolar cells
- ***Toxins****: CagA*, for ulcer or cancer development

MORPHOLOGY

- Gastric biopsy: H. pylori in mucus layer, antrum.
- Neutrophils, Plasma cells, lymphocytes & macrophages.
- **Lymphoid aggregates>>> increased risk of MALT lymphoma.**
- **Intestinal metaplasia (goblet cells)>>> dysplasia >> increased risk of adenocarcinoma**







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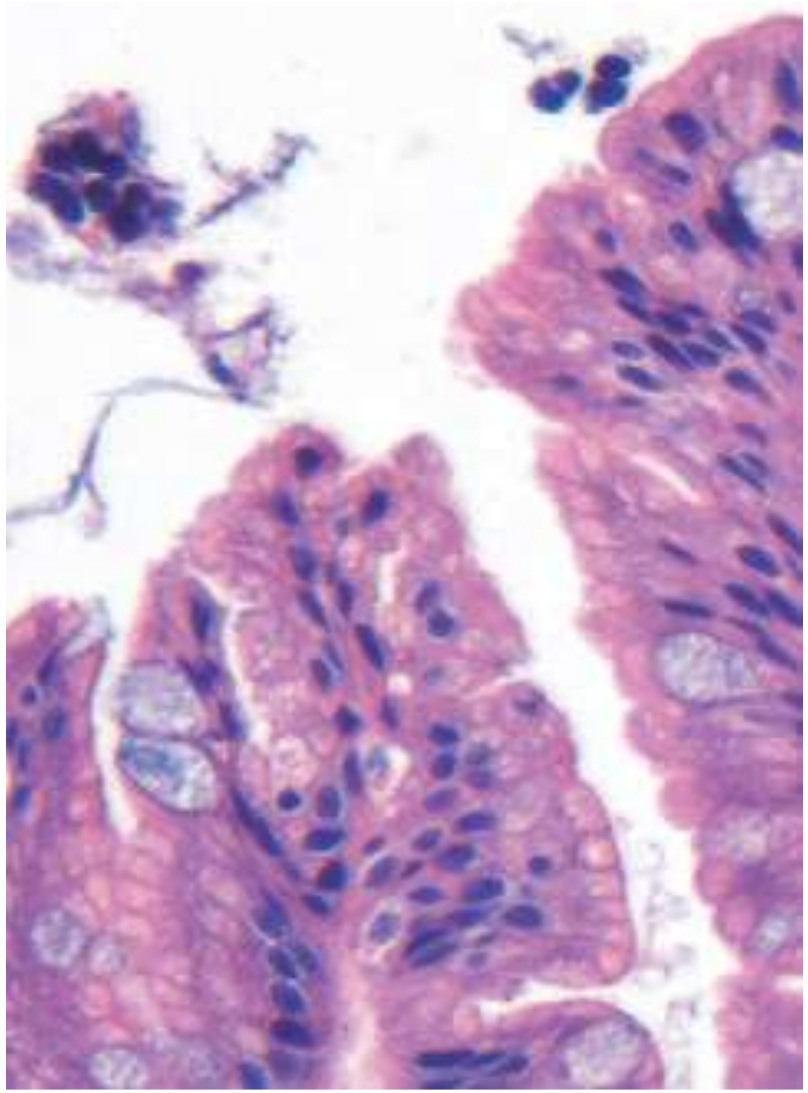
Intestinal metaplasia



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Diagnosis and treatment

- Serologic test: anti-H .pylori antibodies.
- Stool test for H.pylori.
- Urea breath test.

- Gastric antral biopsy (rapid urease test during endoscopy)
- Bacterial culture.
- PCR test for bacterial DNA.

- Treatment: combinations of antibiotics and PPI (triple therapy).

Autoimmune Gastritis

- Antibodies to parietal cells and intrinsic factor in serum.
- Reduced serum pepsinogen I levels
- Antral endocrine cell hyperplasia
- Vitamin B12 deficiency >>> pernicious anemia and neurologic changes
- Impaired gastric acid secretion (*achlorhydria)*
- Spares the antrum.
- Marked *hypergastrinemia*

Pathogenesis

- Immune-mediated loss of parietal cells >>> reductions in acid and intrinsic factor secretion.
- Acid reduction leads to hypergastrinemia
- Hyperplasia of antral G cells
- Deficient intrinsic factor >> deficient ileal VB12 absorption >> megaloblastic anemia.
- Some chief cell damage >> reduced pepsinogen

MORPHOLOGY

- Damage of the oxyntic (acid-producing) mucosa.
- Diffuse atrophy, thinning of wall, loss of rugal folds
- Lymphocytes, plasma cells, macrophages, less likely neutrophils.
- Intestinal metaplasiac >>> dysplasia >> carcinoma.
- Neuroendocrine cell hyperplasia >>> tumors.

Clinical features

- 60 years, slight female predominance.
- Often associated with other autoimmune diseases

Table 15.2 Characteristics of *Helicobacter pylori*–Associated and Autoimmune Gastritis

Feature	*H. pylori*–Associated	Autoimmune
Location	Antrum	Body
Inflammatory infiltrate	Neutrophils, subepithelial plasma cells	Lymphocytes, macrophages
Acid production	Increased to slightly decreased	Decreased
Gastrin	Normal to markedly increased	Markedly increased
Other lesions	Hyperplastic/inflammatory polyps	Neuroendocrine hyperplasia
Serology	Antibodies to *H. pylori*	Antibodies to parietal cells (H^+,K^+-ATPase, intrinsic factor)
Sequelae	Peptic ulcer, adenocarcinoma, lymphoma	Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor
Associations	Low socioeconomic status, poverty, residence in rural areas	Autoimmune disease; thyroiditis, diabetes mellitus, Graves disease

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