Pathology of the stomach-part 1

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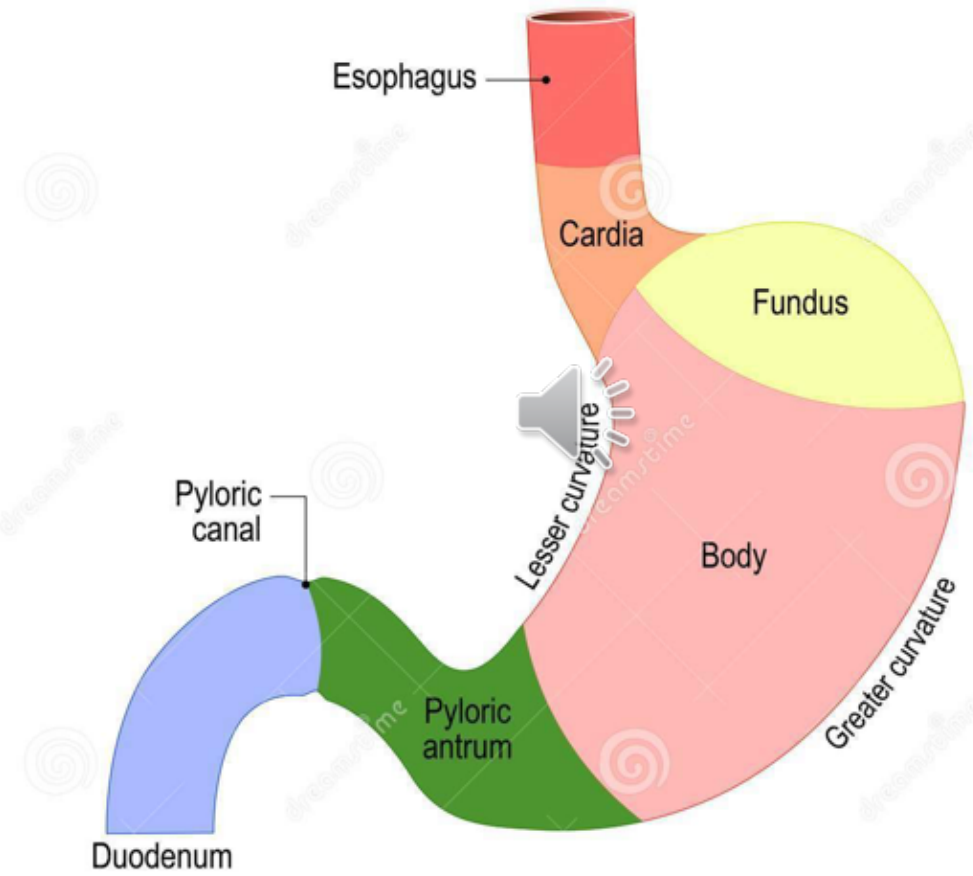
overview

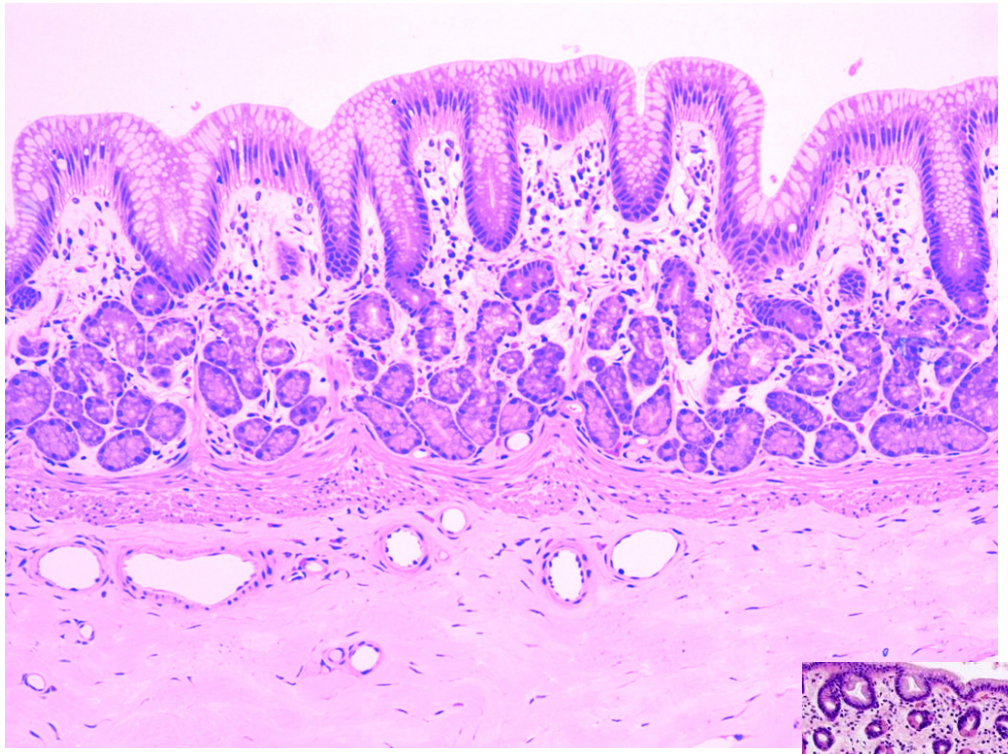
- Gastric diseases:
- 1-Inflammatory.
- 2-Neoplastic.

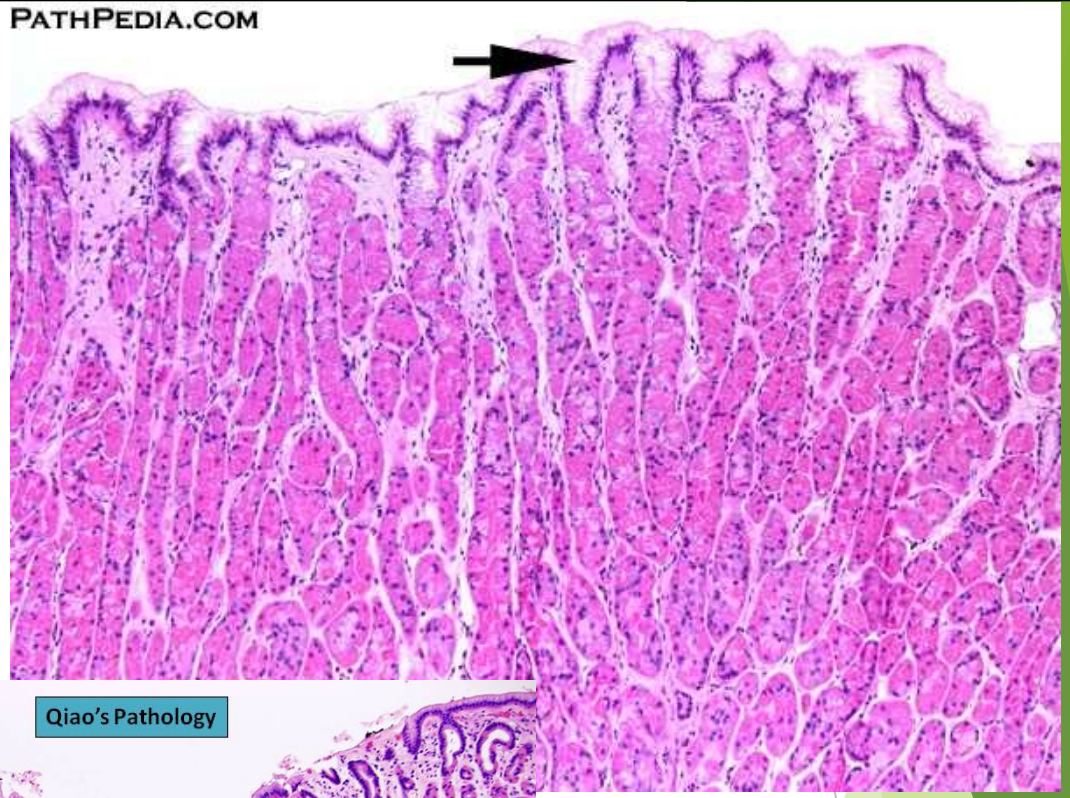
- Stomach parts: cardia, fundus, body, antrum, pylorus.

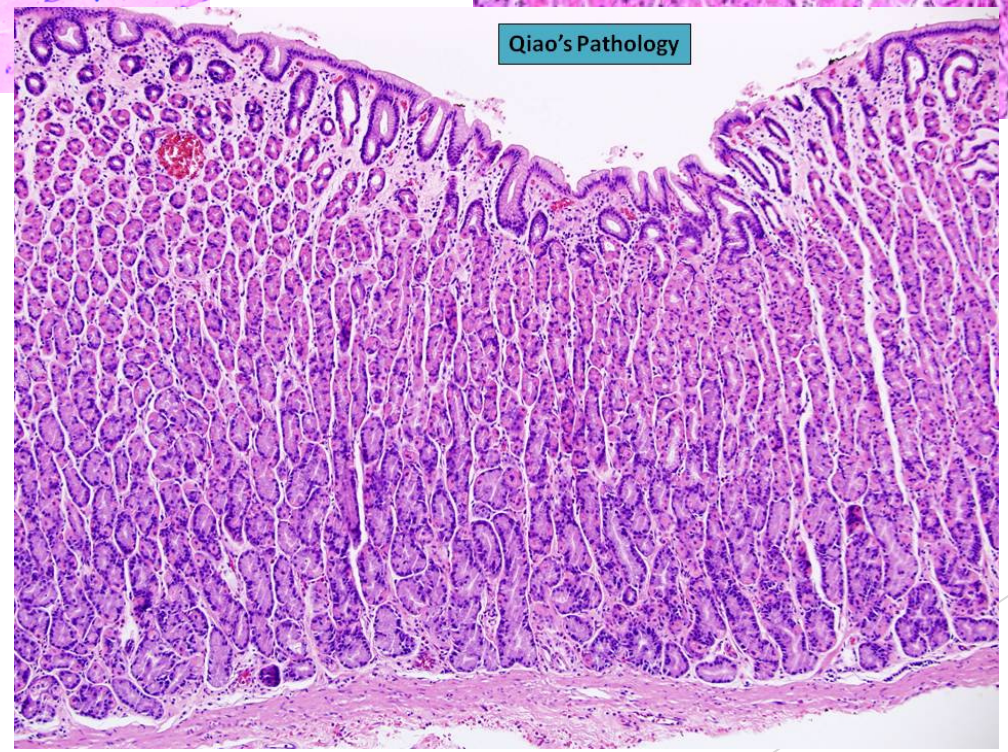
- Cardia: mucin secreting foveolar cells.
- Body and fundus: parietal cells (HCL) and chief cells (pepsin).
- Antrum: neuroendocrine G cells (gastrin)

Sections of human the stomach









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Inflammatory conditions

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

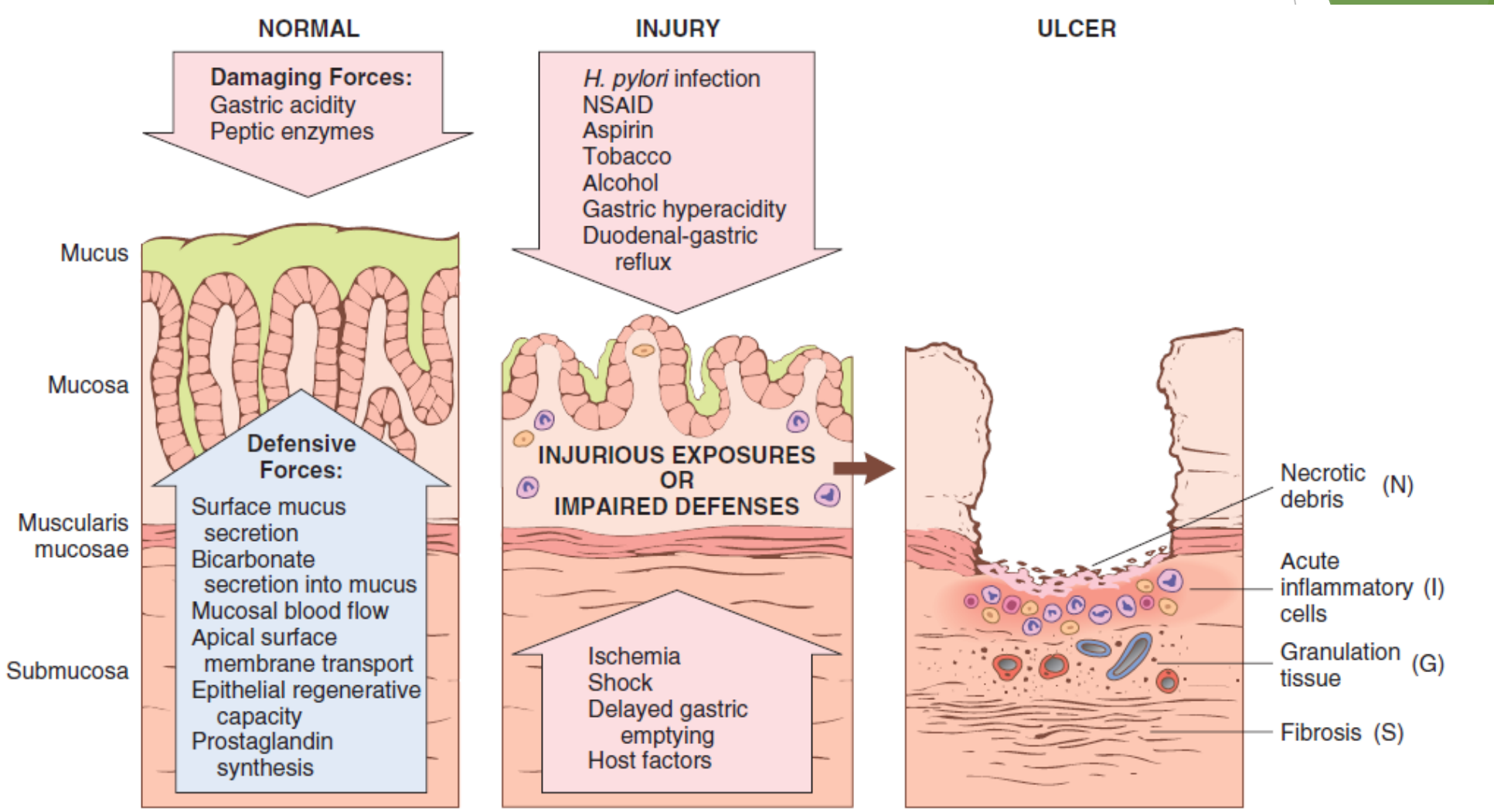
ACUTE GASTRITIS and gastropathy

- **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

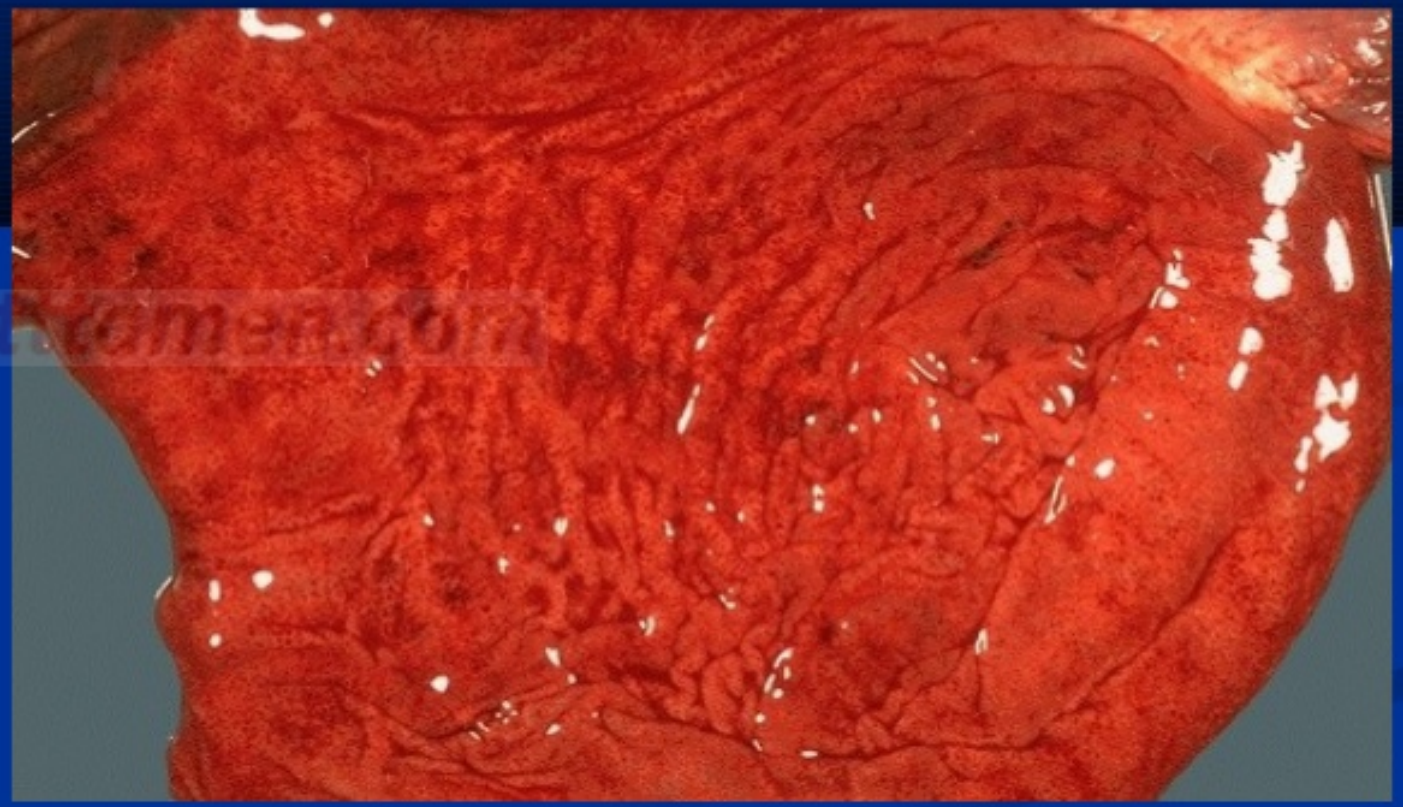
- **Main causes:**
- **NSAIDs**
- **Uremic patients, H pylori** infected patients:
- **Old age**.
- **Hypoxia**
- **Harsh chemicals**, (acids or bases)
- **Alcohol, NSAIDs, radiation therapy:**
- **Chemotherapy**.

MORPHOLOGY

- Hyperemia.
- 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



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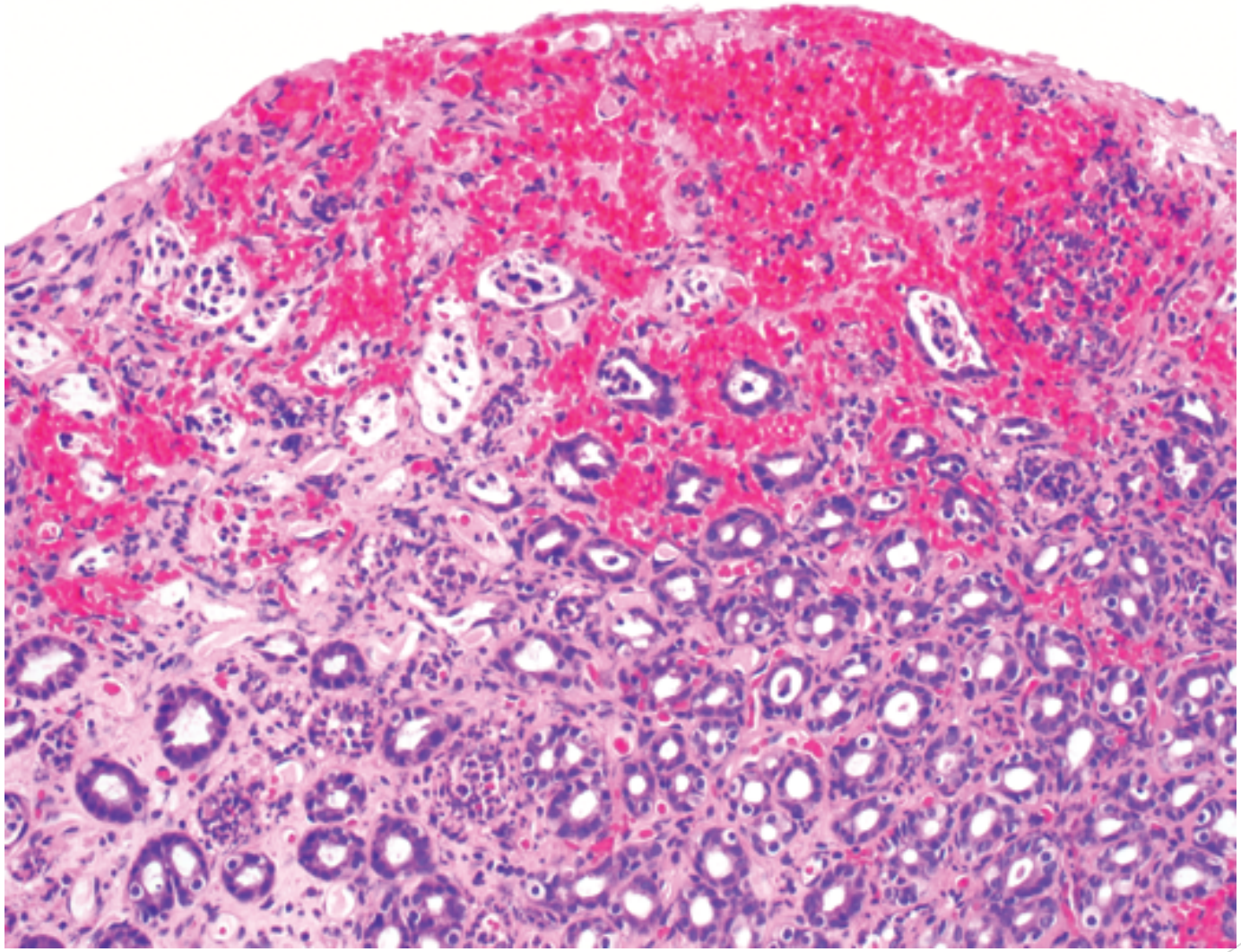

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Stress-Related Mucosal Disease acute gastric ulcers

- 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.

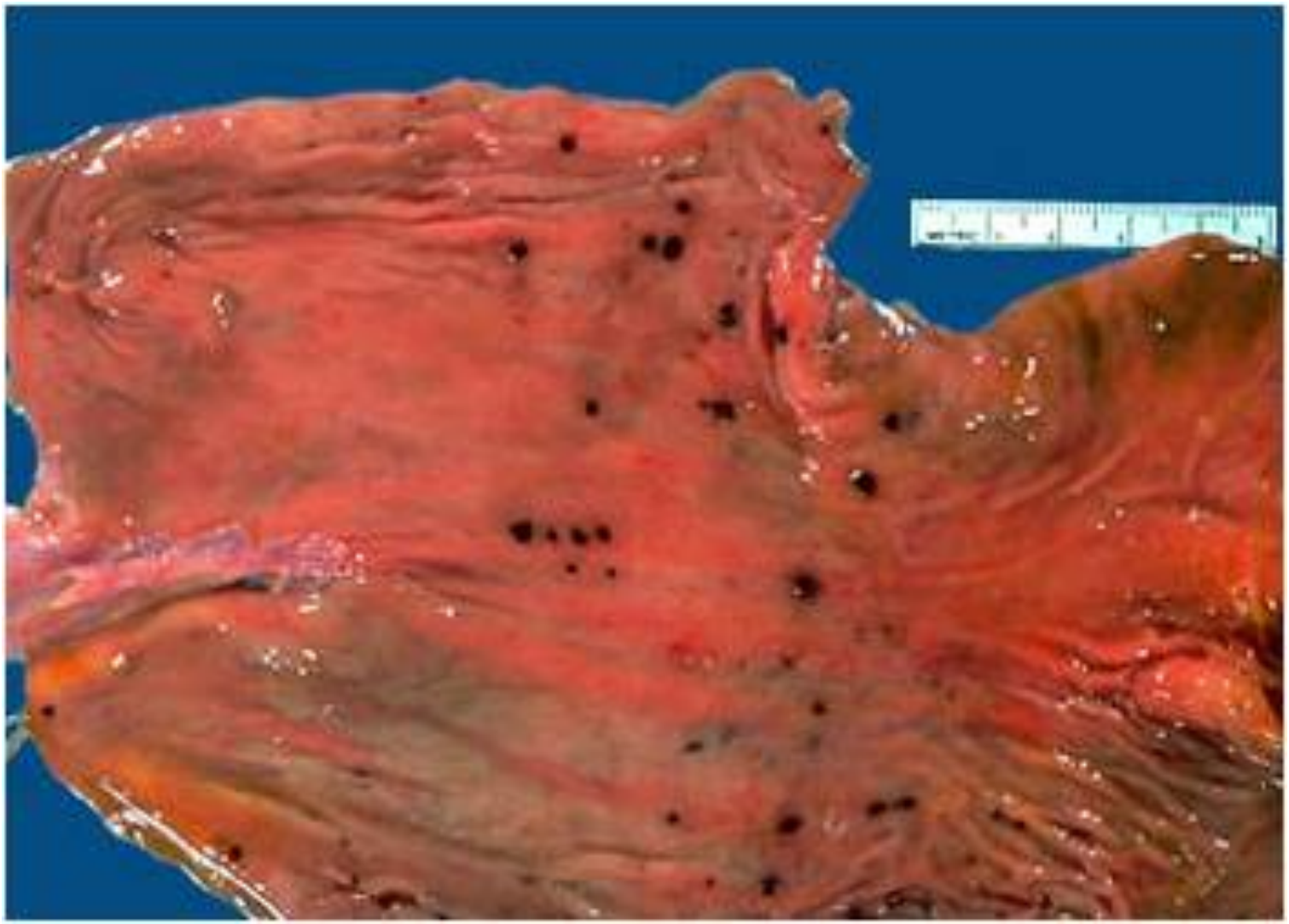
Pathogenesis

- **Stress ulcers:**
- Local ischemia.
- Systemic hypotension.
- Splanchnic vasoconstriction.
- Systemic acidosis (lower 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 reepithelialization 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

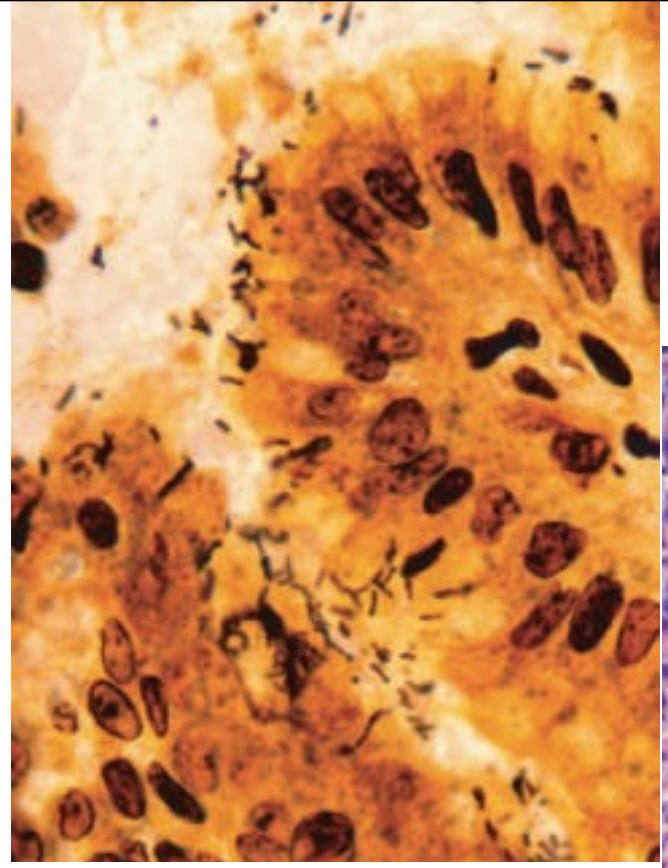
- Discovery of the association of H.pylori *w*ith peptic ulcer disease was a revolution.
- Spiral or curved, G-ve, bacilli.
- Underlying cause for almost all duodenal ulcers.
- Majority of gastric ulcers or chronic gastritis.
- Acute infection is subclinical.
- ***Antral gastritis with increased acid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

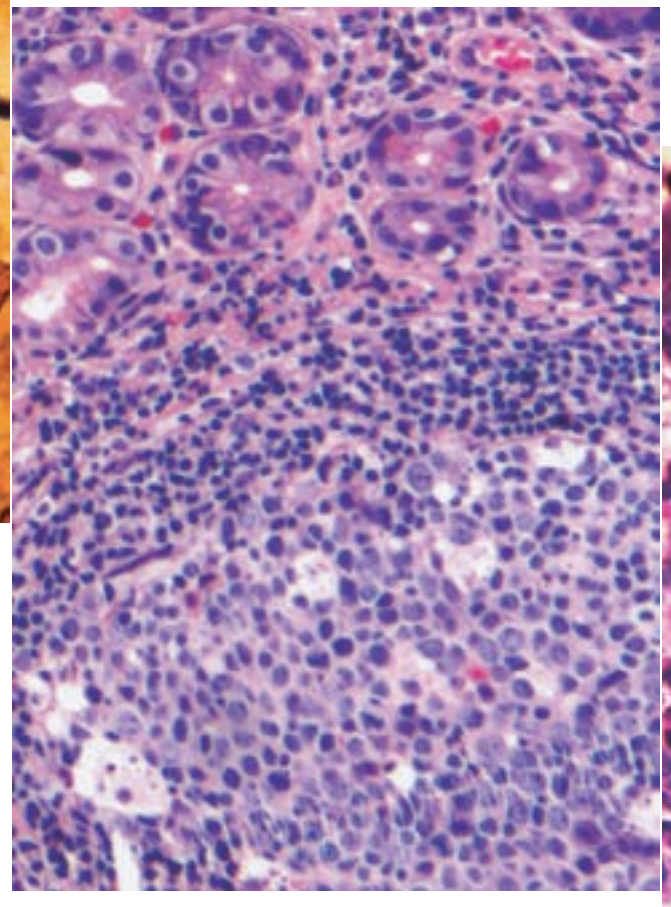
- Poverty, household crowding, limited education, poor sanitation
- Infection is typically acquired in childhood, persists to adult-life.

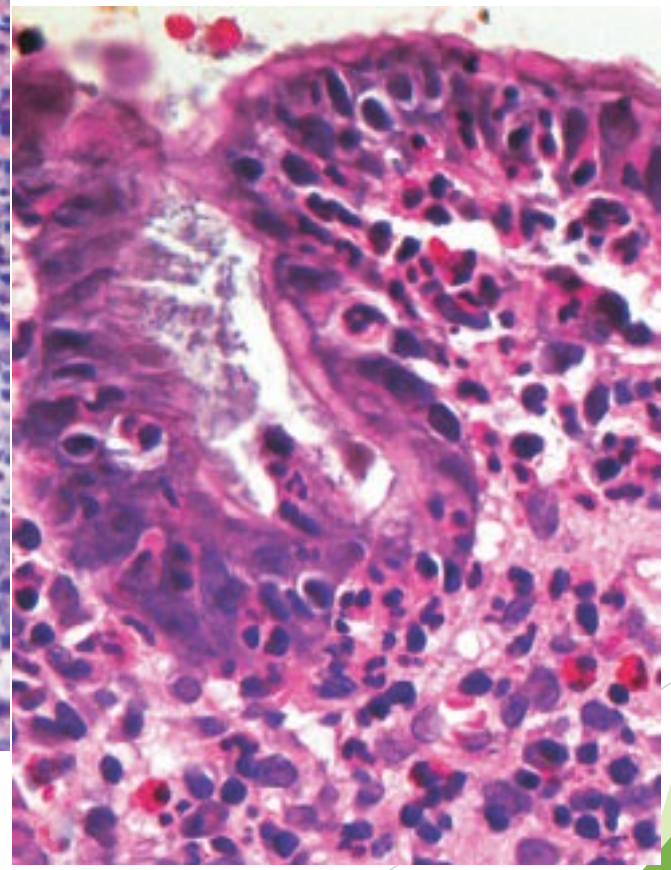
- **Pathogenesis:**
- H.pylori adapted to live in the mucus layer, non-invasive, by
- **Flagella**: allow motility.
- ***Urease****: split urea to ammonia,* protect bacteria from acidic pH.
- ***Adhesins****:* bacterial adherence to foveolar cells
- ***Toxins****: CagA,* for ulcer or cancer development

MORPHOLOGY

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







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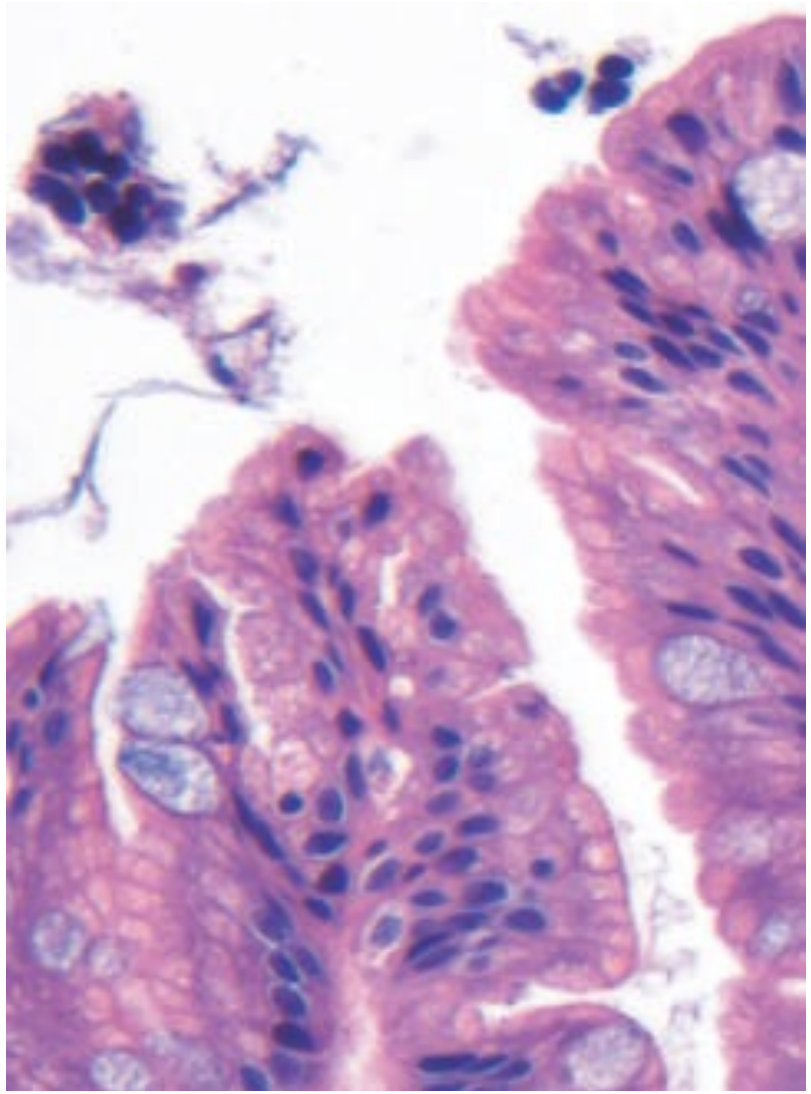
Intestinal metaplasia



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Diagnosis and treatment

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

- Gastric biopsy
- Bacterial culture.
- PCR test for bacterial DNA.

- Treatment: combinations of antibiotics and PPI.

^{13}C-Urea Breath Test –How to collect breath

for UBIT Tablet

Simplified test procedure

No gargling necessary

Breathe into the first sample bag (before taking the UBIT tablet).

Immediately (within 5 sec) swallow one UBIT tablet on an empty stomach with 100 ml of water. Do not chew, crush or dissolve the tablet.

After taking the UBIT tablet, Lie down on your left side for 5 minutes.

Remain seated for a further 15 minutes.

Twenty minutes after taking the UBIT tablet, collect breath again using the second sample bag. The two sample bags containing breath before and after administration will be collected for analysis

※Things to keep in mind when collecting breath

1)Hold the sample bag against the mouth, breathe in through the nose and hold the breath for 5-10 seconds.

2)Breathe slowly into the bag.

3)If you have difficulty holding your breath, make two or three short breaths into the bag instead.

4)When blowing into the bag, make sure to breathe out from the lungs.

www.nagase.com.

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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