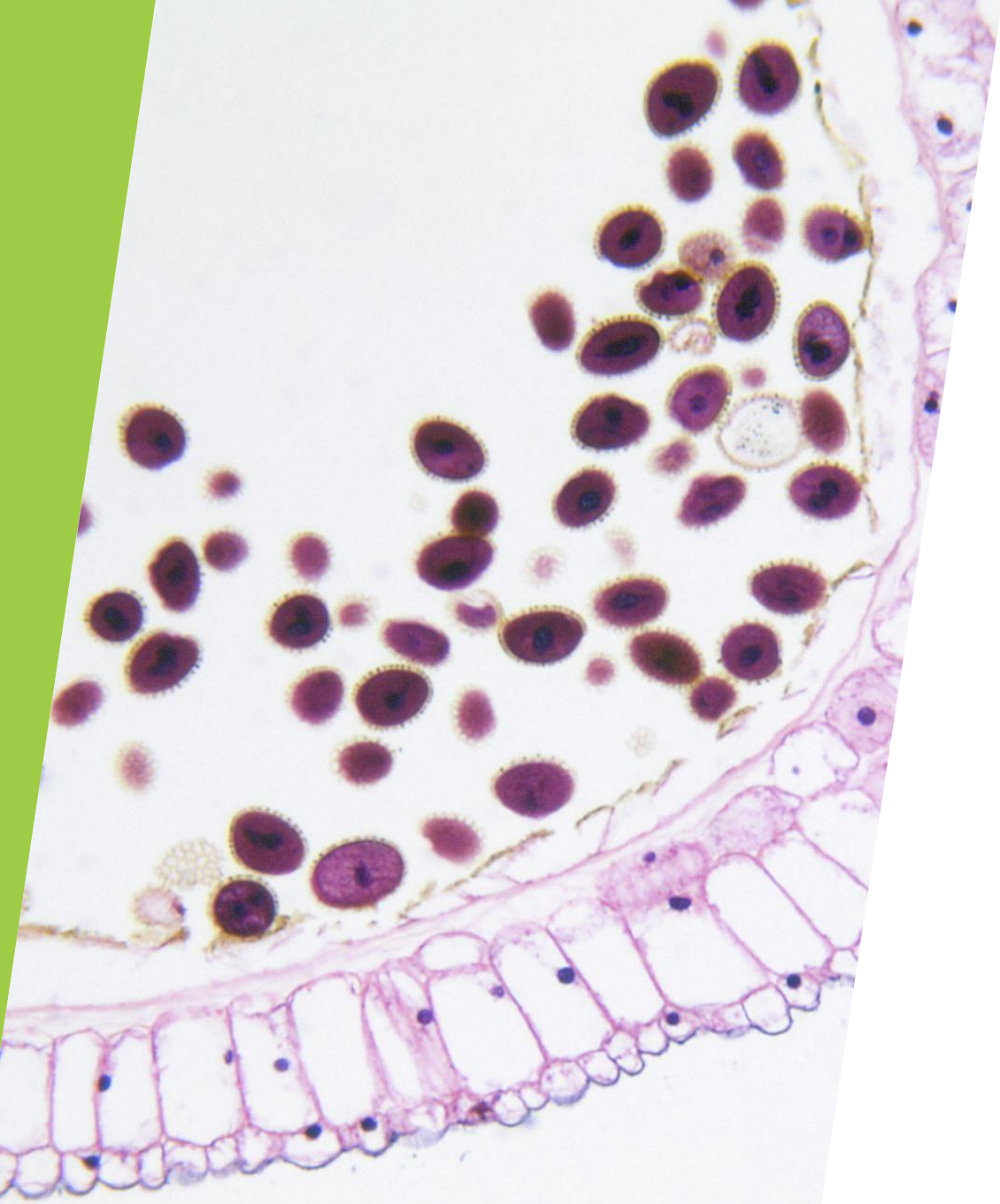


Pathology of the stomach- 1

Manar Hajeer, MD, FRCPath

University of Jordan, School of medicine

Overview

Gastric diseases:

1-Inflammatory.

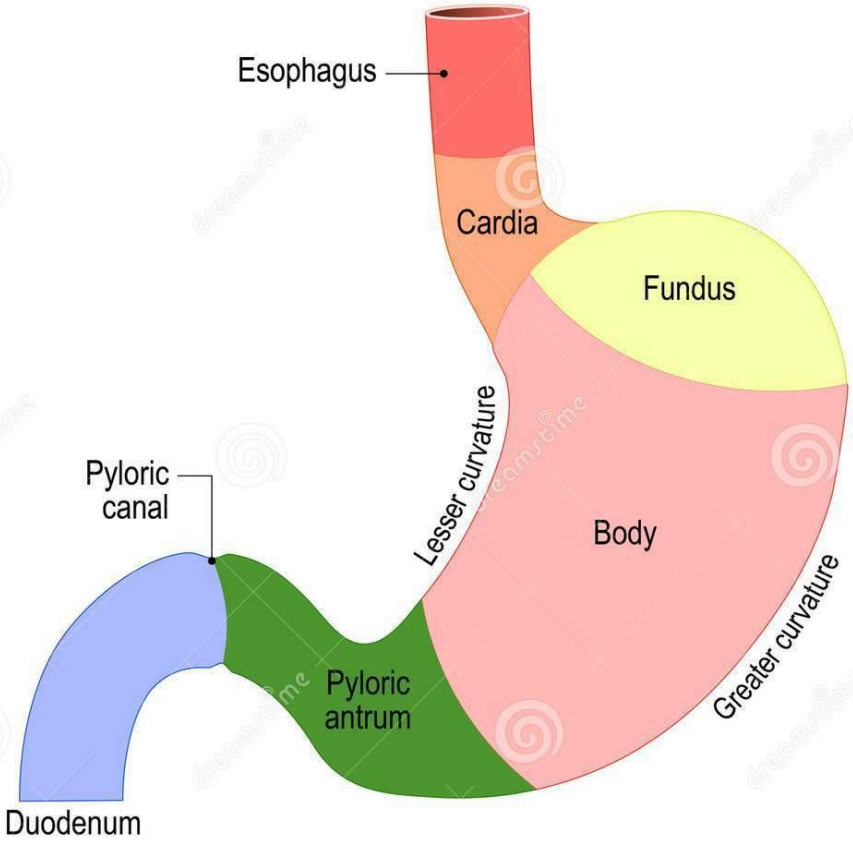
2-Neoplastic.

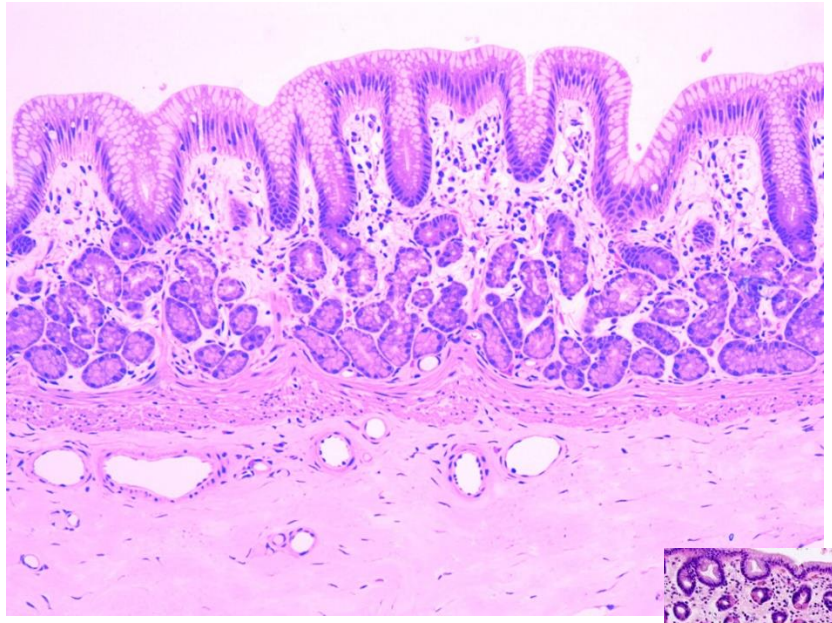
Normal anatomy & histology:

- 4 mains parts: cardia, fundus, body, antrum (pylorus).

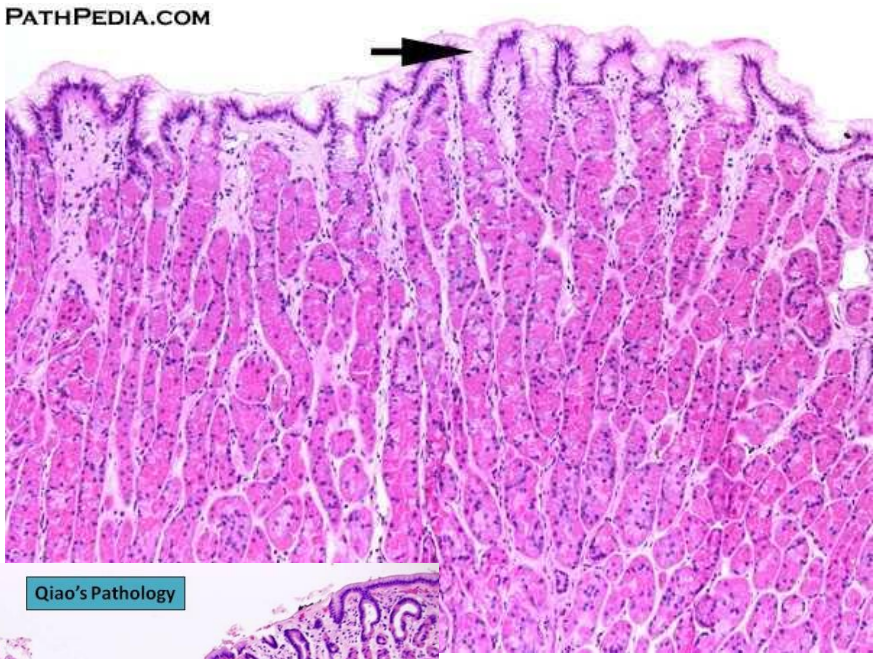
- Cardia: mucin secreting foveolar cells.
- Body and fundus: parietal cells (HCL) and chief cells (pepsin).
- Antrum: neuroendocrine G cells (gastrin)

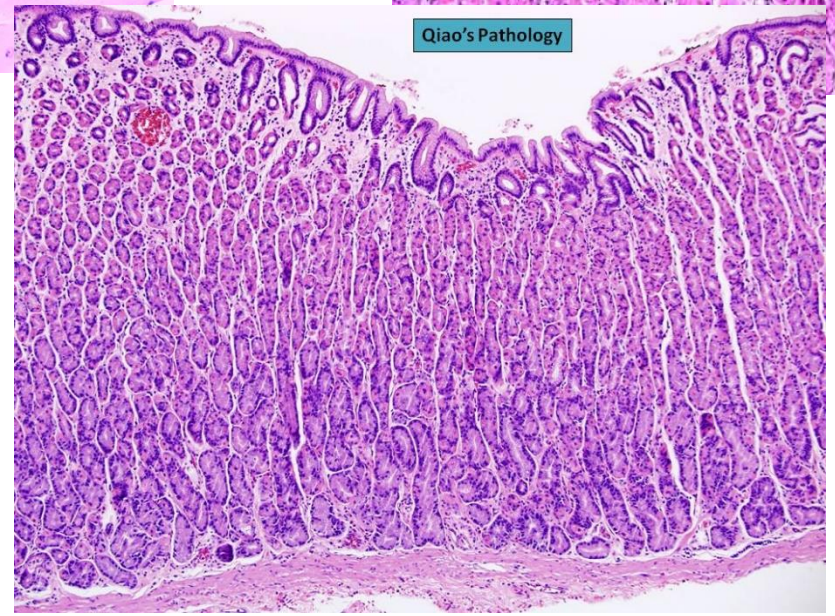
Sections of human the stomach





medcell.med.yale.edu





Inflammatory conditions

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

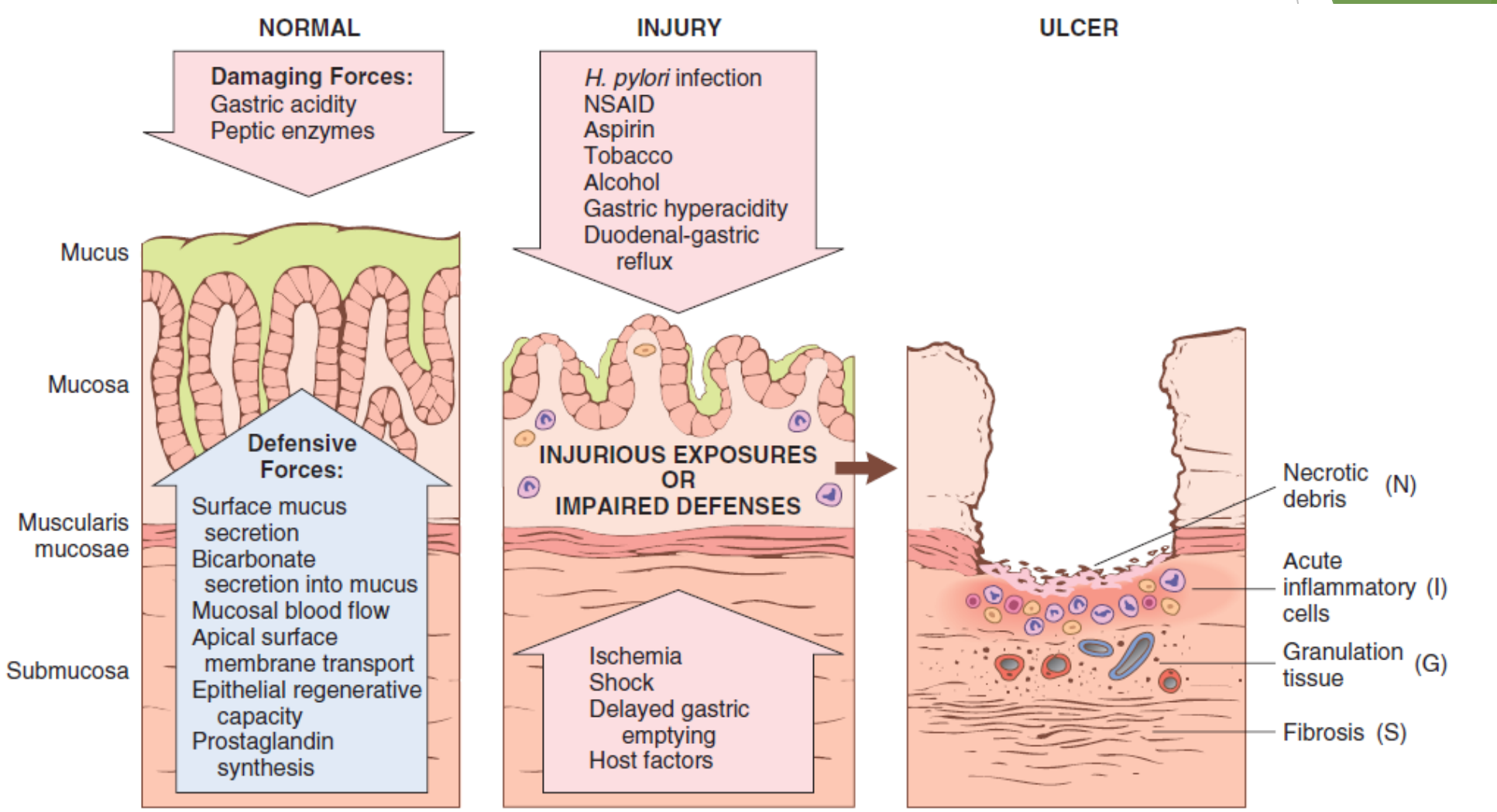
ACUTE GASTRITIS and gastropathy

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

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

- **Clinical features:**
- Asymptomatic.
- Epigastric pain, nausea, vomiting.
- Severe: erosions, ulcers, hematemesis, melena.

Pathogenesis



Robbins Basic Pathology 10th edition

Pathogenesis of gastropathy, acute and chronic gastritis:

- Imbalance between protective and damaging forces
- **Main causes:**
- **NSAIDs (COX1 and COX2 inhibitors)**
- **Uremic patients (ammonia inhibit bicarbonate transport)**
- **H pylori (urease produces ammonia)**
- **Aging (reduced mucin and bicarbonate secretion)**
- **Hypoxia (high altitudes)**
- **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:

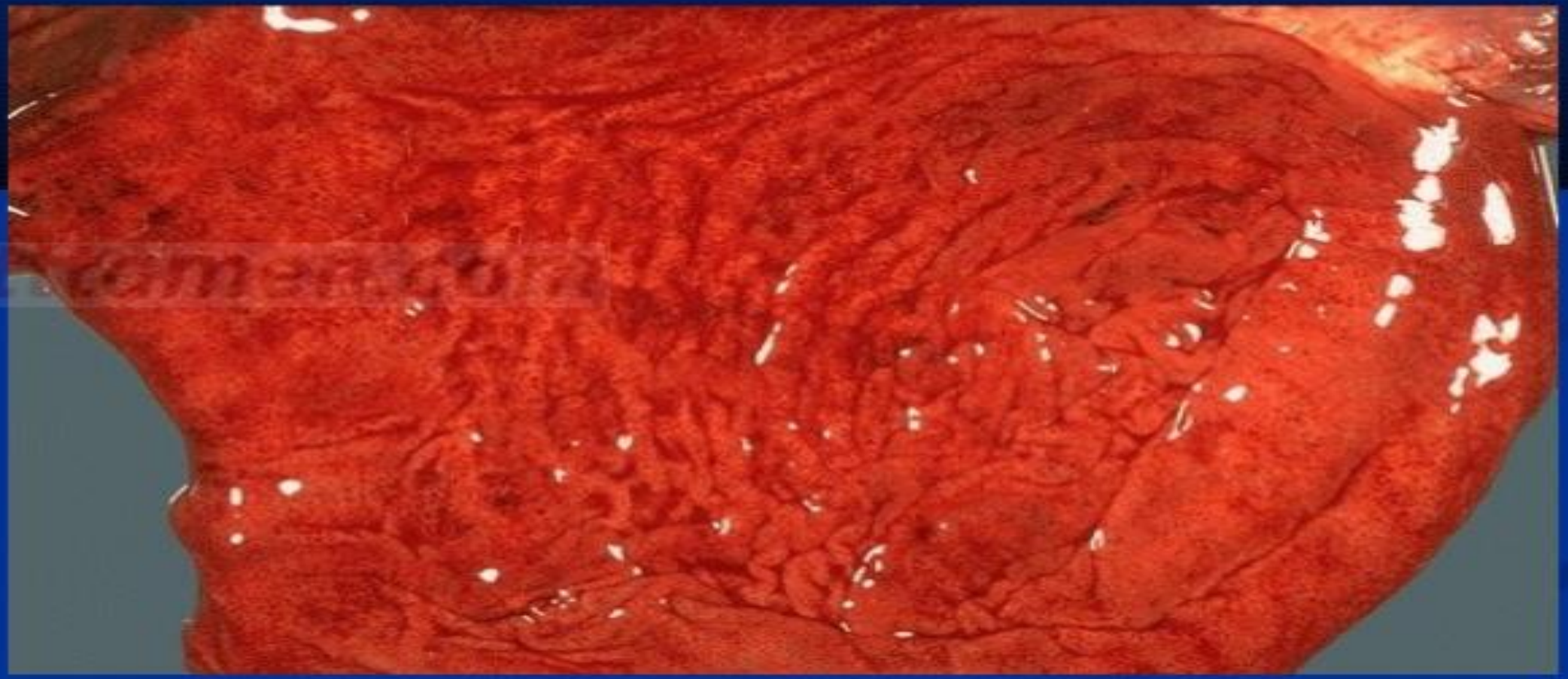
- Stimulate nearly all the defense mechanisms including

1. Mucus and bicarbonate secretion,
2. mucosal blood flow
3. Epithelial restitution.

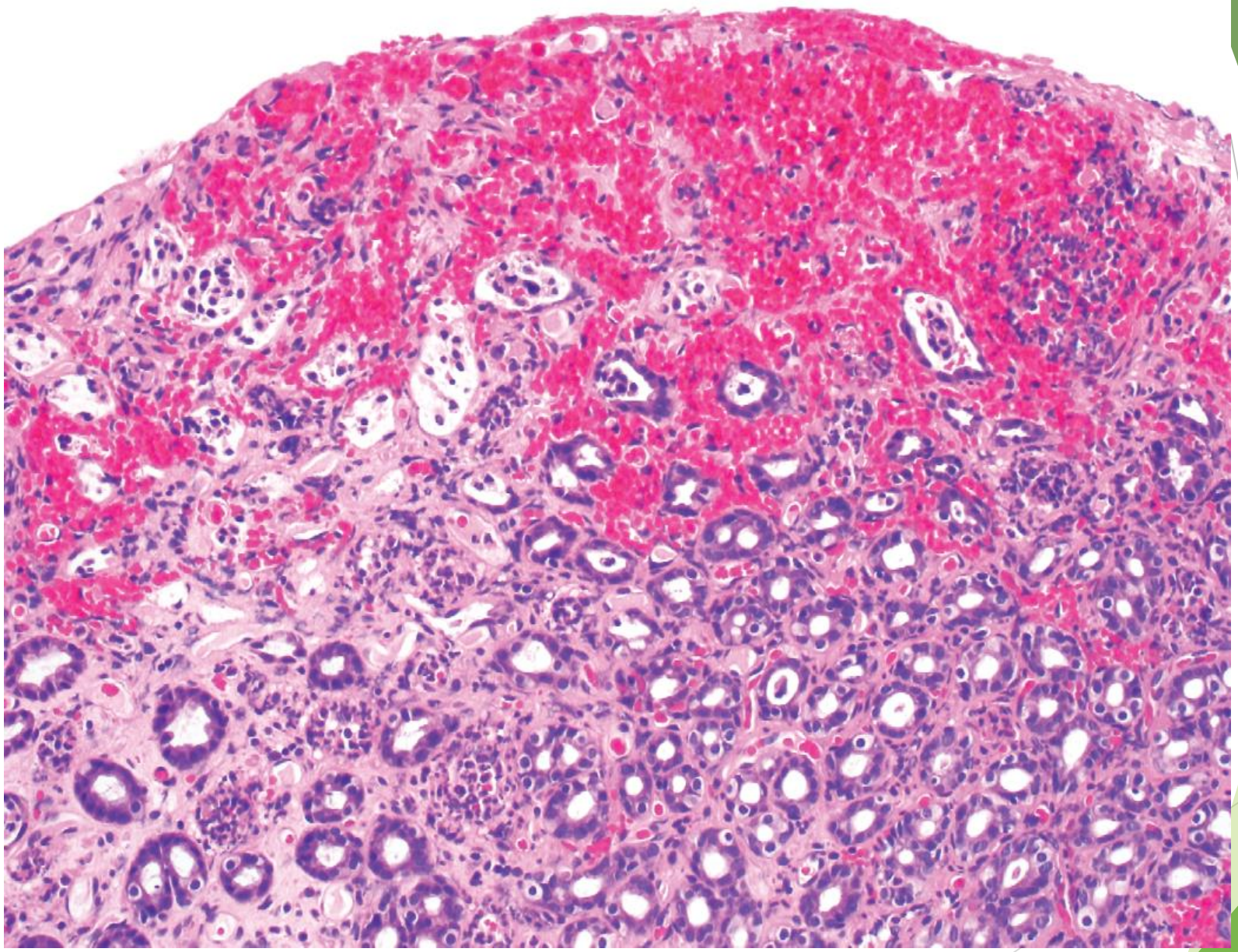
MORPHOLOGY

- Hyperemia (redness).
- Edema and slight vascular congestion
- Neutrophils, lymphocytes, and plasma cells are not prominent.
- Neutrophils : Active inflammation (gastritis) .
- Intact surface epithelium if mild.
- **Acute erosive hemorrhagic gastritis (Advanced)**

ACUTE GASTRITIS



B



librepathology.org

Stress-Related Mucosal Disease

- Severe physiologic stress:

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

Stress-Related Mucosal Disease

- **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, CNS injury as stroke, high risk of perforation.

Pathogenesis

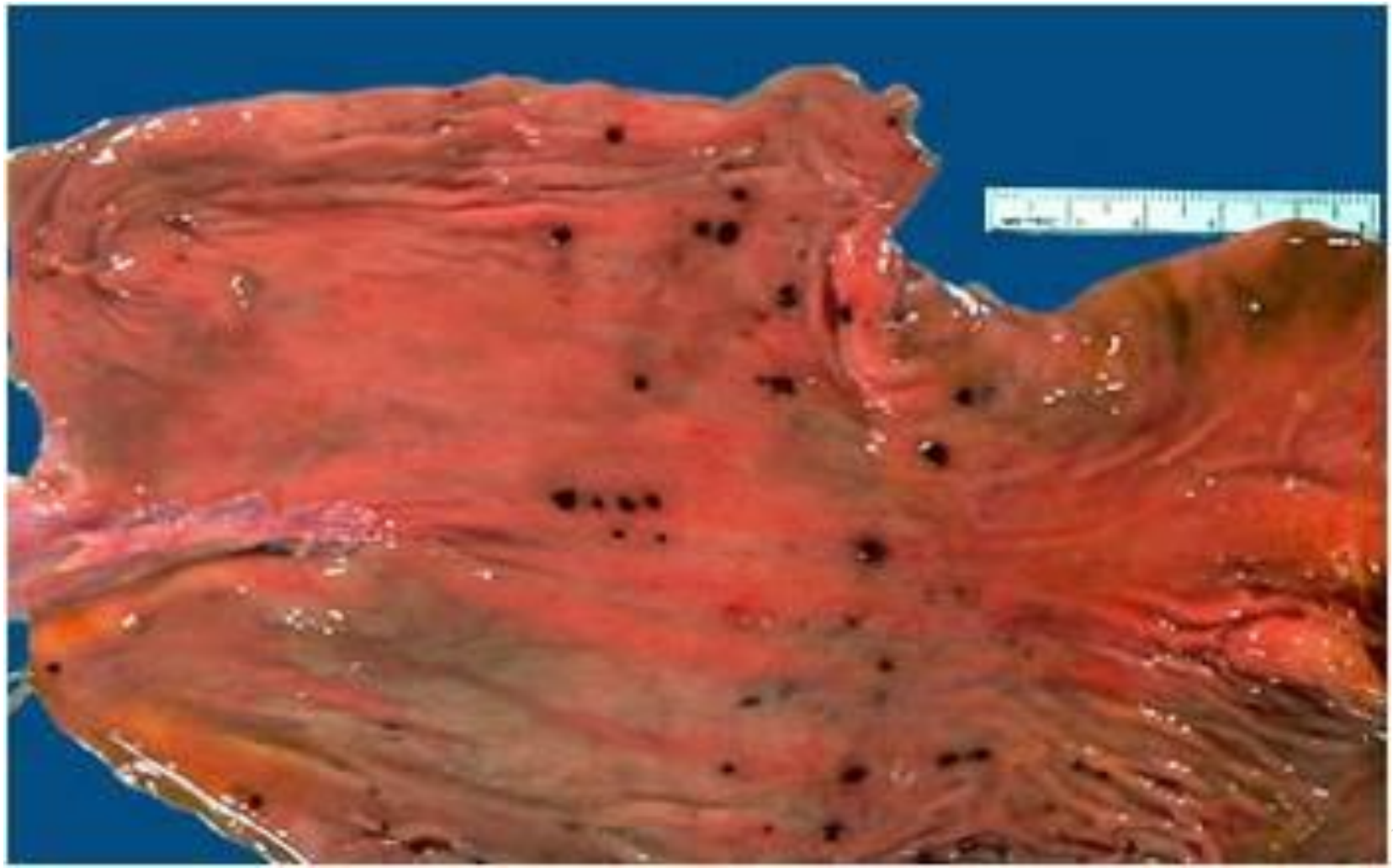
- **Stress related injury:**
- Mostly due to Local ischemia caused by.
- Systemic hypotension.
- Decreased blood flow (Splanchnic vasoconstriction)
- Systemic acidosis (lower intracellular PH).
- COX2 expression is protective.

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

MORPHOLOGY

- Spectrum (Shallow to deep).
- Acute ulcers are rounded and typically < 1 cm.
- Ulcer base brown to black.
- Multiple, anywhere in stomach
- Normal adjacent mucosa
- No scarring
- Healing with complete epithelialization occurs days or weeks after removal of injurious factors

Stress ulcers



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 with peptic ulcer disease was a revolution.
- Spiral or curved, G-ve, bacilli.
- In almost all duodenal ulcers and majority of gastric ulcers or chronic gastritis.

- **Epidemiology:**
- Poverty, poor sanitation. Acquired in childhood, persists to adult-life.
- Acute infection is subclinical.

- **Pathogenesis:**
- Non-invasive, adapted to live in the mucus layer:
- **Flagella**: allow motility.
- **Urease**: split urea to ammonia, protect bacteria from acidic pH.
- **Adhesins**: bacterial adherence to foveolar cells
- **Toxins**: (CagA) mucosal damage.

Pathogenesis:

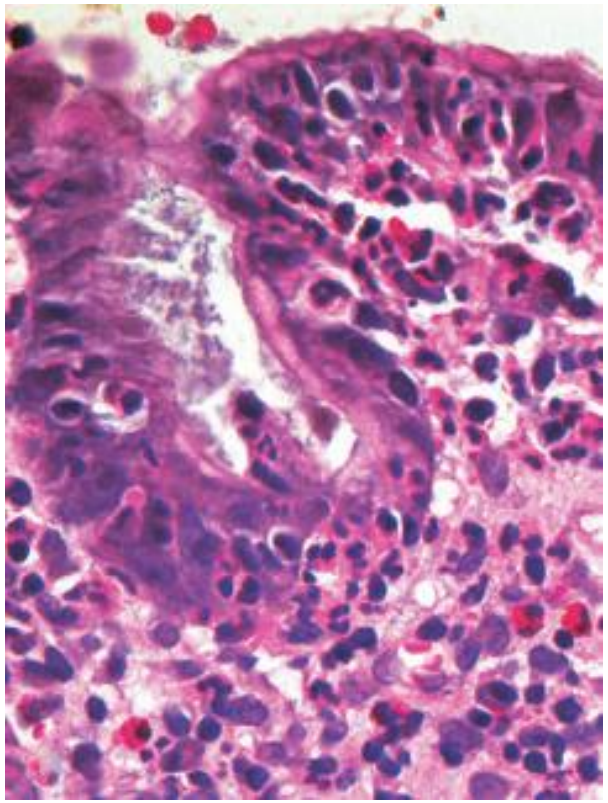
Starts as Antral gastritis >>stimulate G cells >> increased acid production >> peptic ulcer

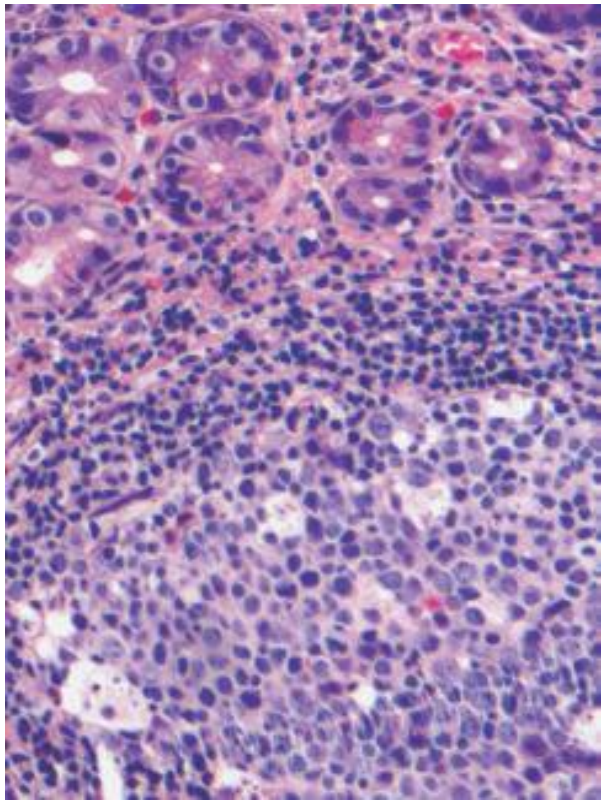
If severe: spread to body with atrophy (damage Parietal cells).

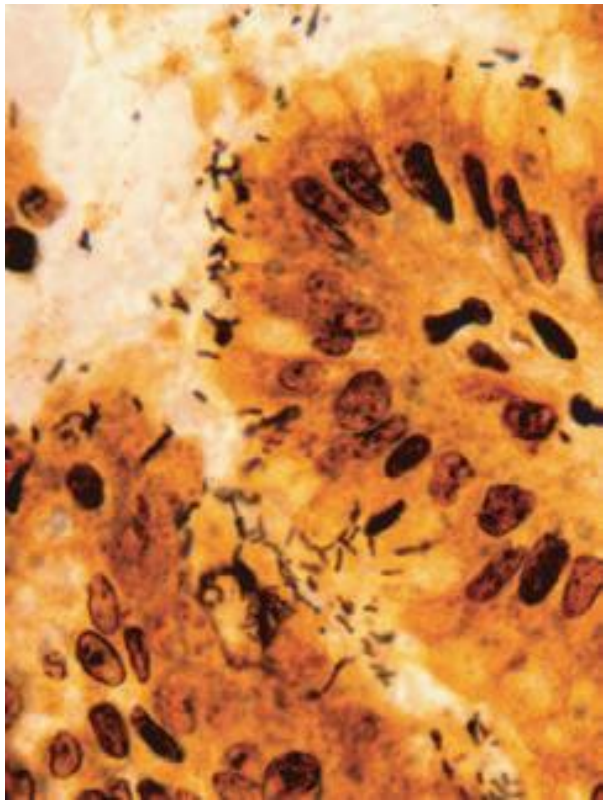
Intestinal metaplasia and increased risk of gastric cancer.

MORPHOLOGY

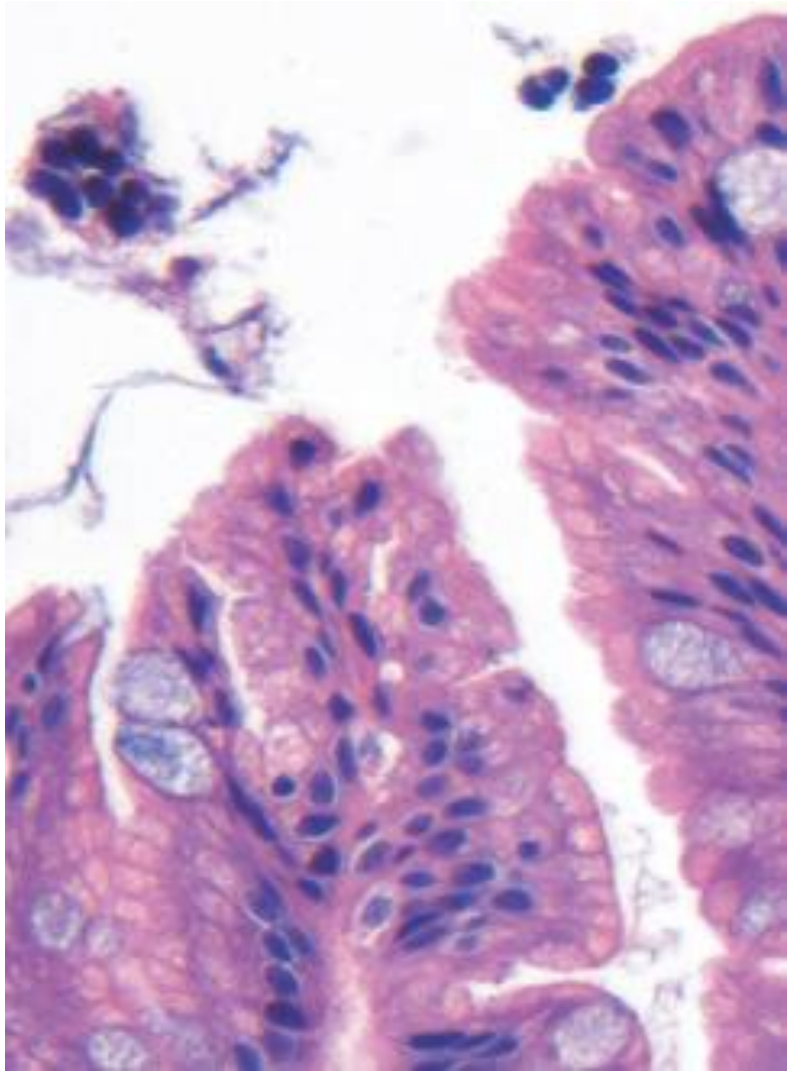
- **Gastric antral biopsy**: H. pylori in mucus layer.
- Regenerative changes (hyperplastic polyps)
- Neutrophils, Plasma cells, lymphocytes & macrophages.
- **Lymphoid aggregates>>> increased risk of MALT lymphoma.**
- **Intestinal metaplasia (goblet cells)>>> dysplasia >> increased risk of adenocarcinoma**







Intestinal metaplasia



- Robbins Basic Pathology 11th edition

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)*
- Marked *hypergastrinemia*
- Spares the antrum.

Pathogenesis

Immune-mediated loss of parietal cells >>> reductions in acid and intrinsic factor secretion.

Acid reduction >>> Hyperplasia of antral G cells >>> hypergastrinemia

Deficient intrinsic factor >> deficient ileal VB12 absorption >> pernicious anemia.

MORPHOLOGY

- Damage of the oxyntic (acid-producing) mucosa.
- Diffuse atrophy, thinning of wall, loss of gastric folds
- Lymphocytes, plasma cells, macrophages, less likely neutrophils.
- **Intestinal metaplasia >>> dysplasia >> carcinoma.**
- **G- cell hyperplasia >>> carcinoids.**

Clinical features

- 60 years, slight female predominance.
- Often associated with other autoimmune diseases
- Dyspepsia.
- Anemia (VB12 or iron)

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

Robbins Basic Pathology 10th edition

Complication of chronic gastritis

- Peptic ulcer.
- Mucosal atrophy.
- Intestinal Metaplasia
- Dysplasia.