

Pathology of the stomach- 1

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Overview

Gastric diseases:

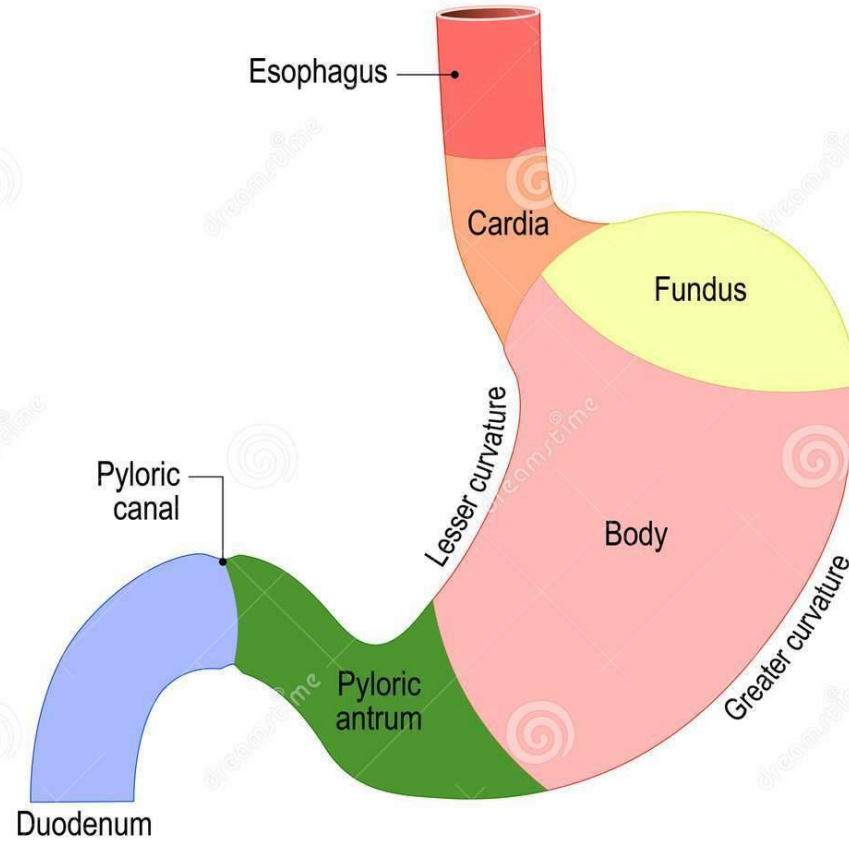
1-Inflammatory.

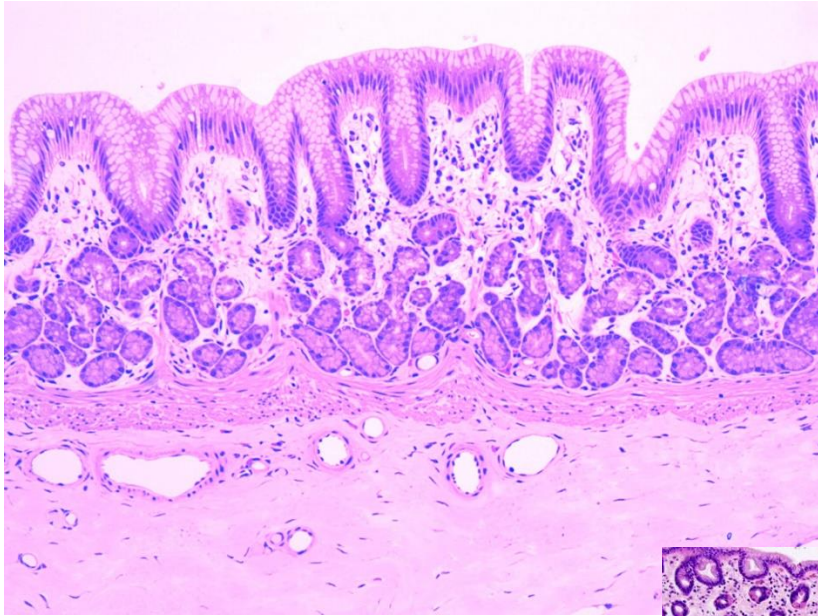
2-Neoplastic.

Normal anatomy & histology:

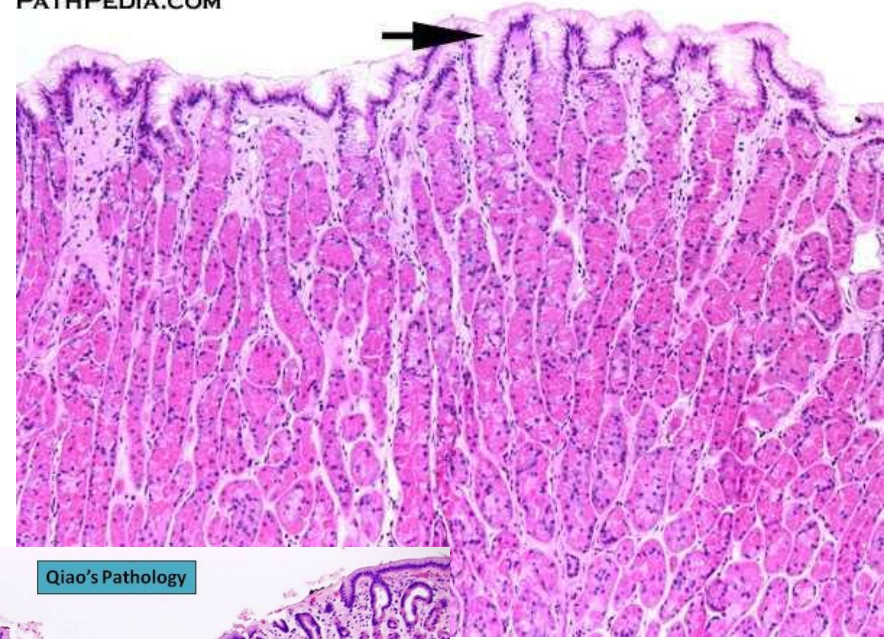
- ▶ 4 main parts: cardia, fundus, body, antrum (pylorus).
- ▶ Cardia: mucin secreting foveolar cells.
- ▶ Body and fundus: parietal cells (HCL) and chief cells (pepsin).
- ▶ Antrum: neuroendocrine G cells (gastrin)

Sections of human the stomach

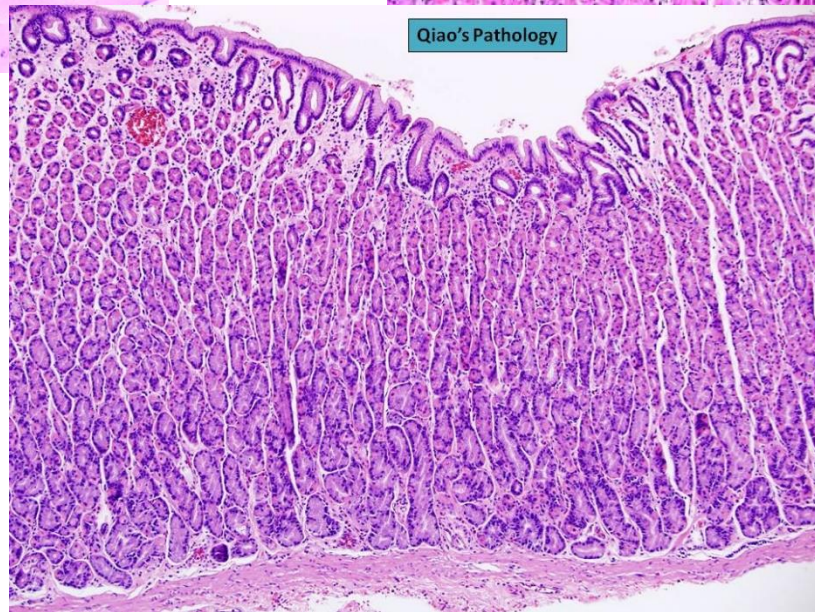




PATHPEDIA.COM



Qiao's Pathology



medcell.med.yale.edu

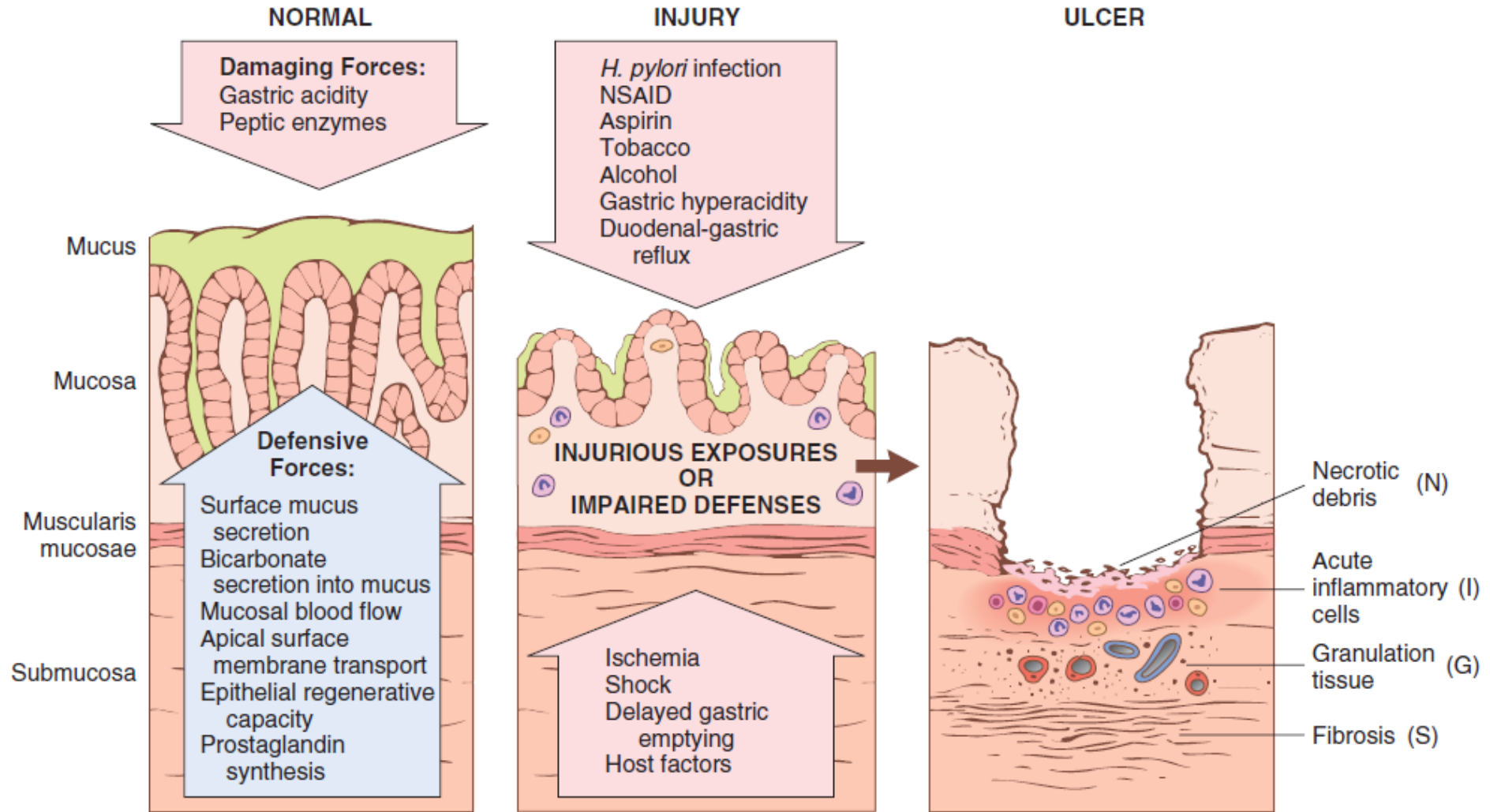
Inflammatory conditions

- ▶ 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



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)**

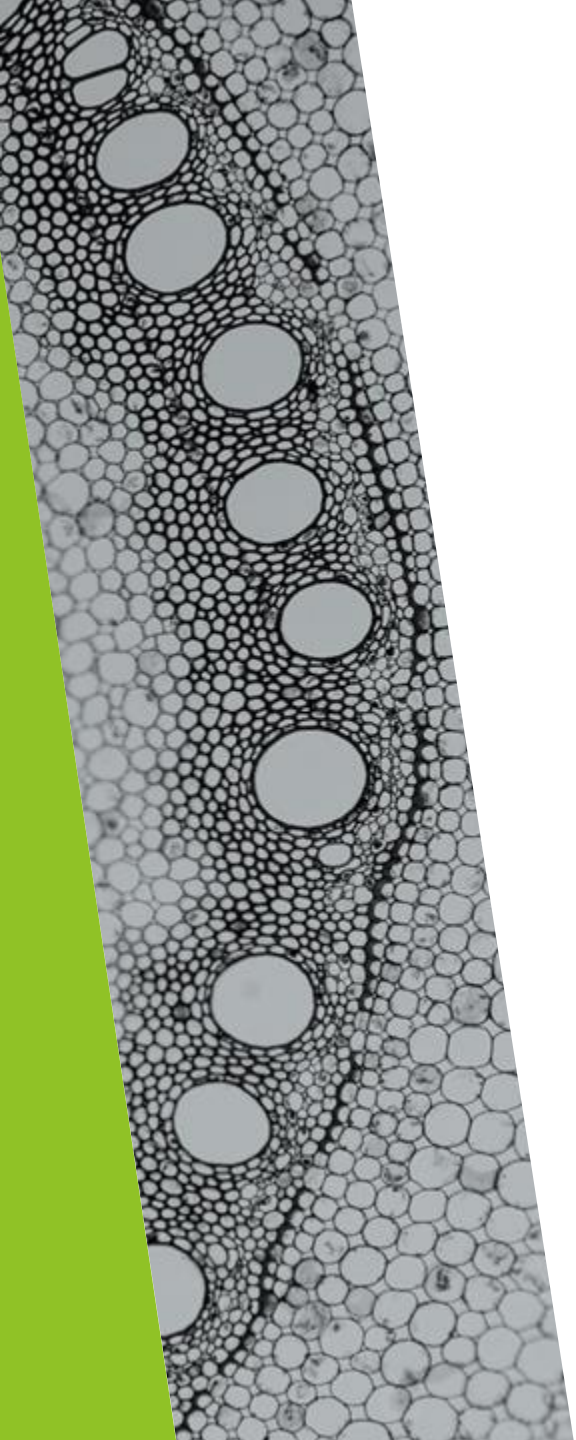


prostaglandins E2 and I2:

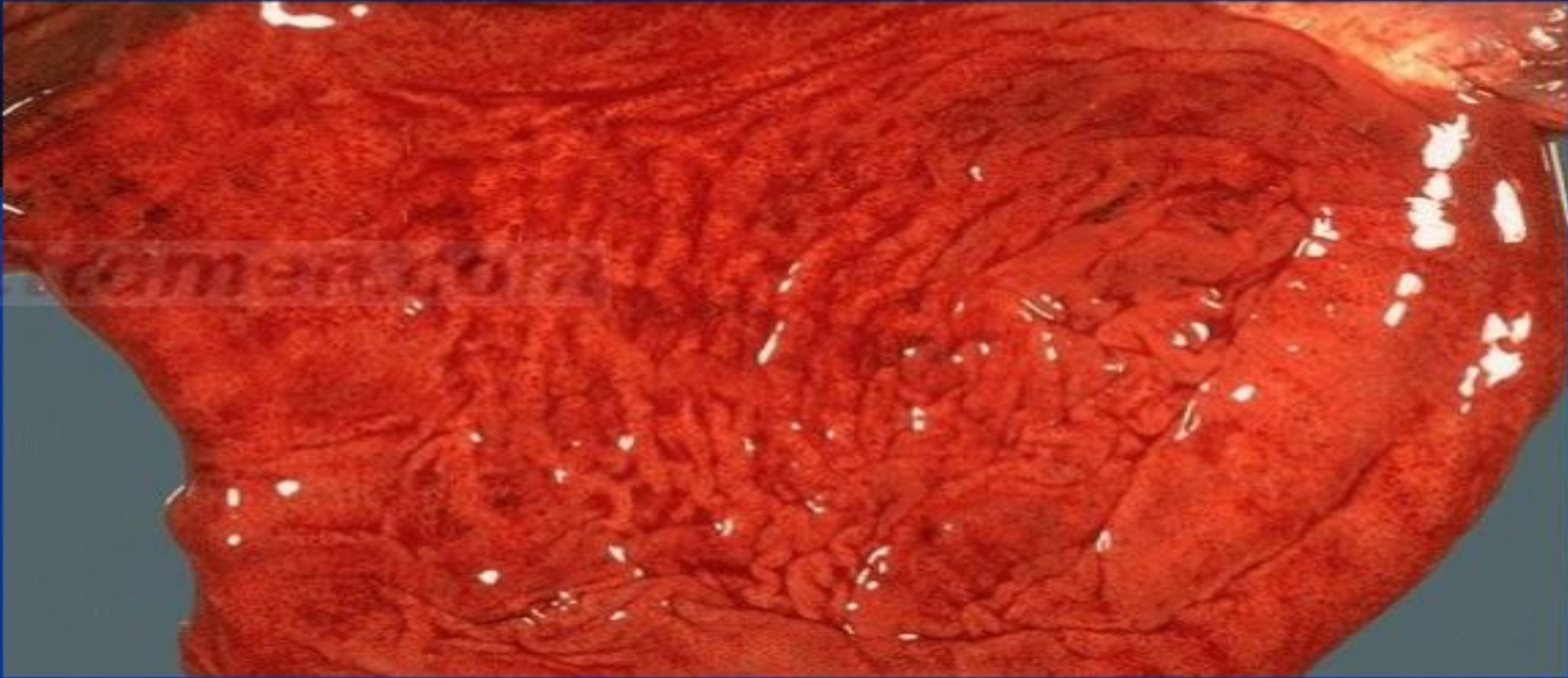
- ▶ Stimulate nearly all the defense mechanisms including
 1. Mucus and bicarbonate secretion,
 2. mucosal blood flow
 3. Epithelial restitution.

MORPHOLOGY

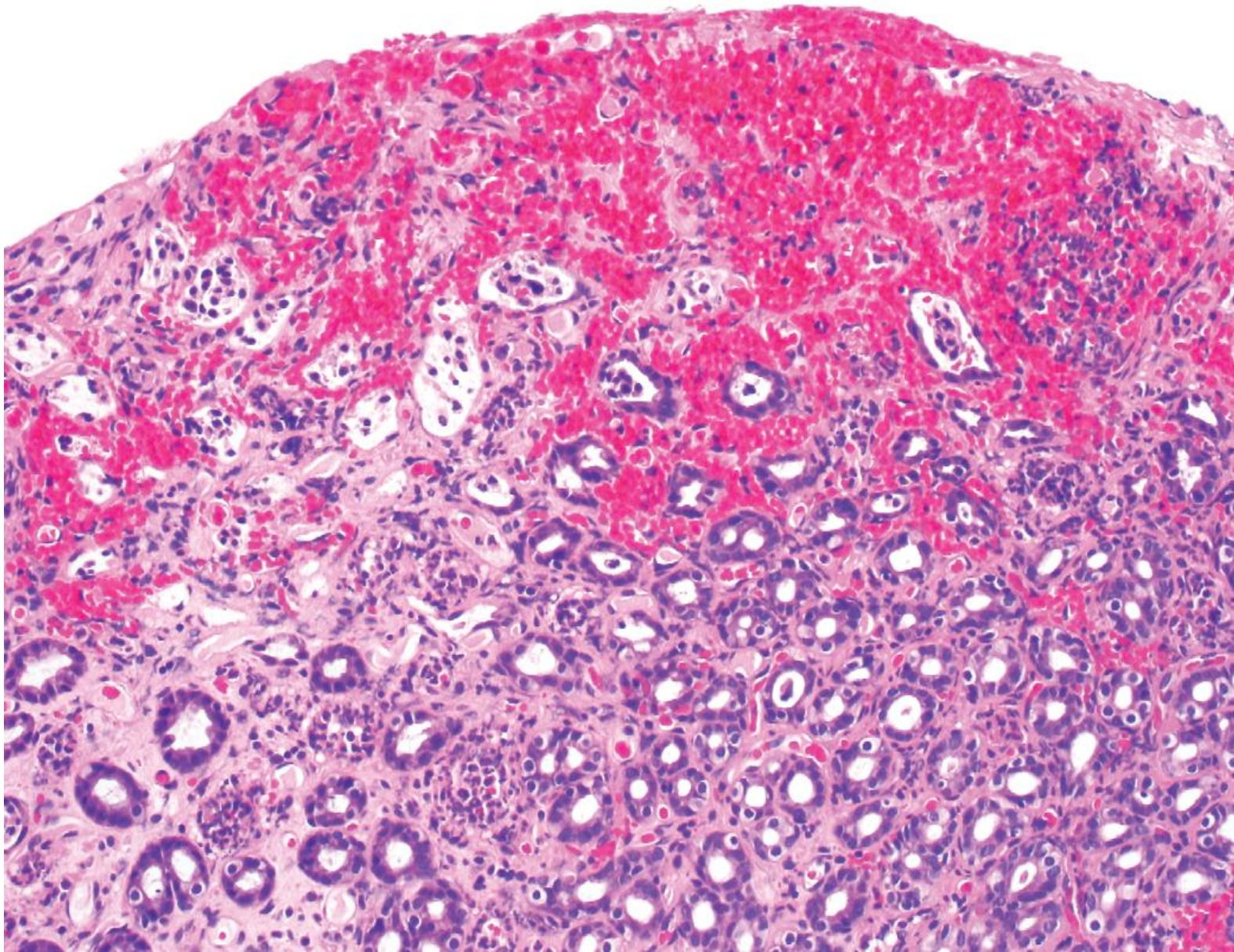
- ▶ Hyperemia (redness).
- ▶ Edema and slight vascular congestion
- ▶ Neutrophils, lymphocytes, and plasma cells are not prominent.
- ▶ Neutrophils : Active inflammation (gastritis) .
- ▶ Intact surface epithelium if mild.
- ▶ **Acute erosive hemorrhagic gastritis (Advanced)**



ACUTE GASTRITIS



B



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.

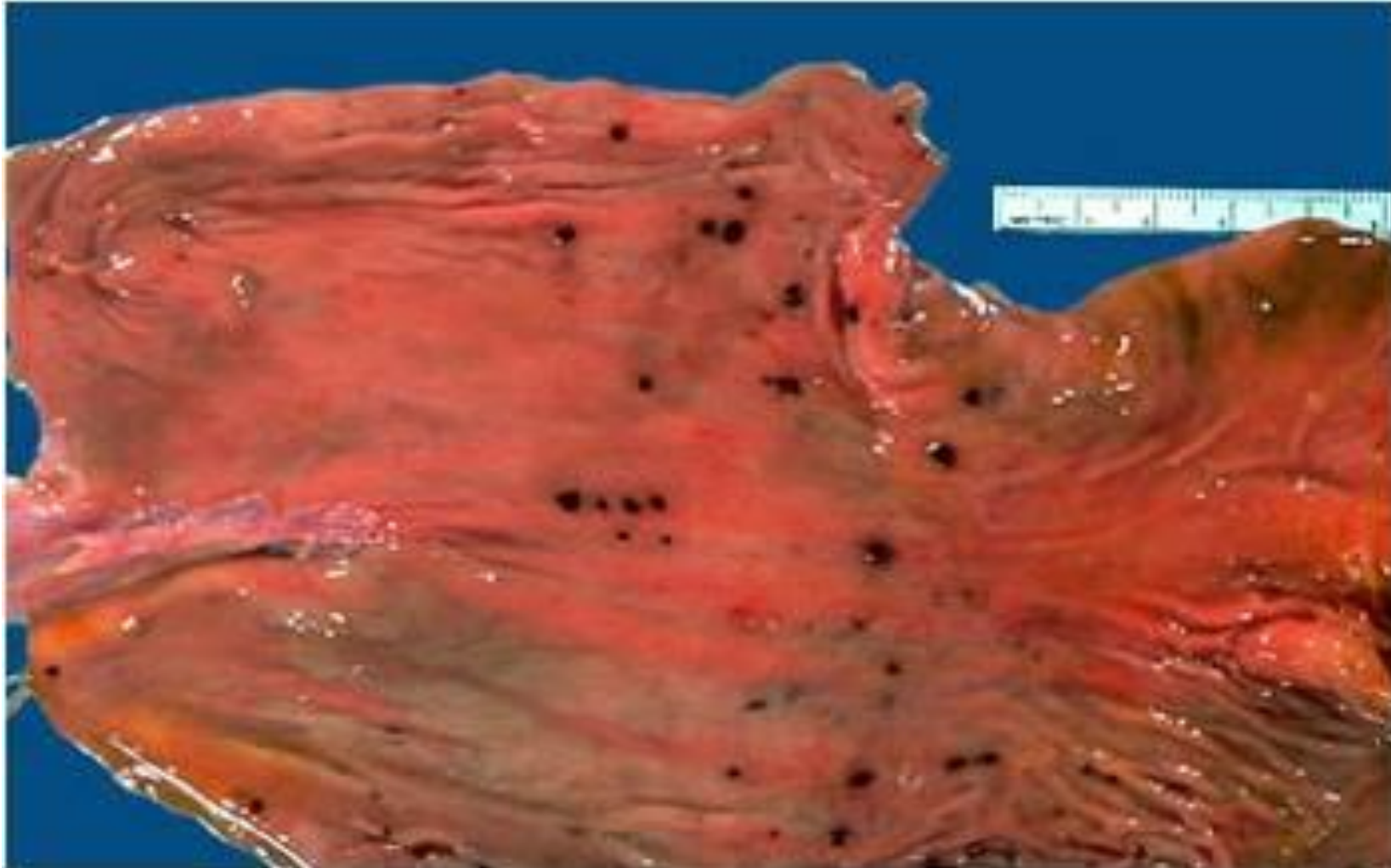
- ▶ **CNS injury and Cushing ulcers:**
- ▶ Direct vagal stimulation, acid hypersecretion.



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:

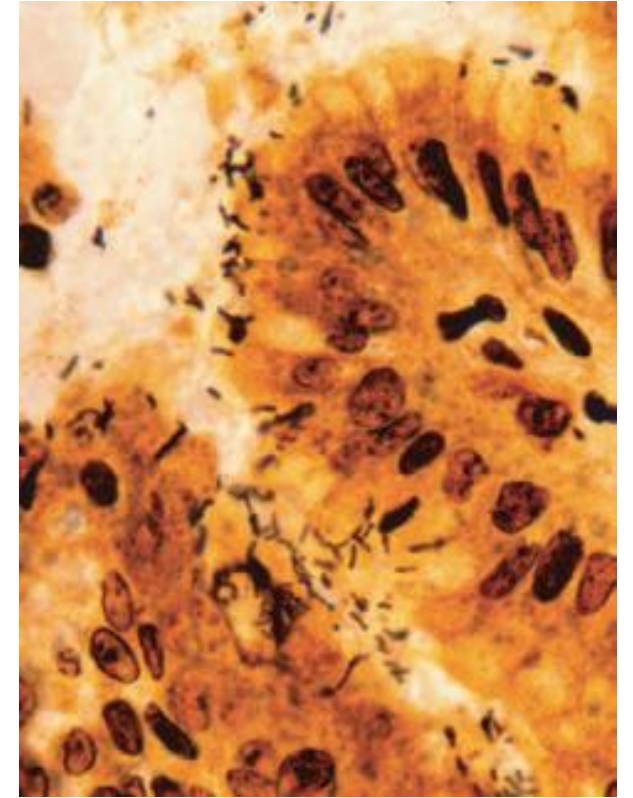
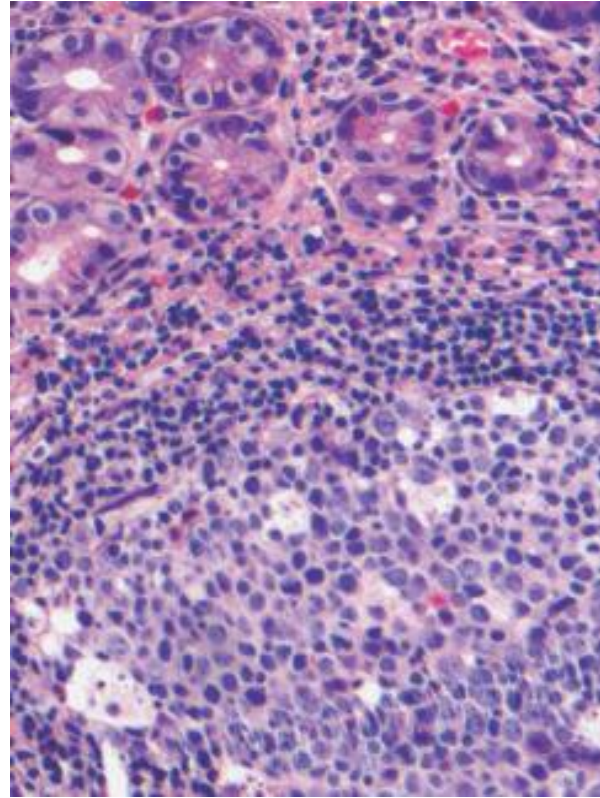
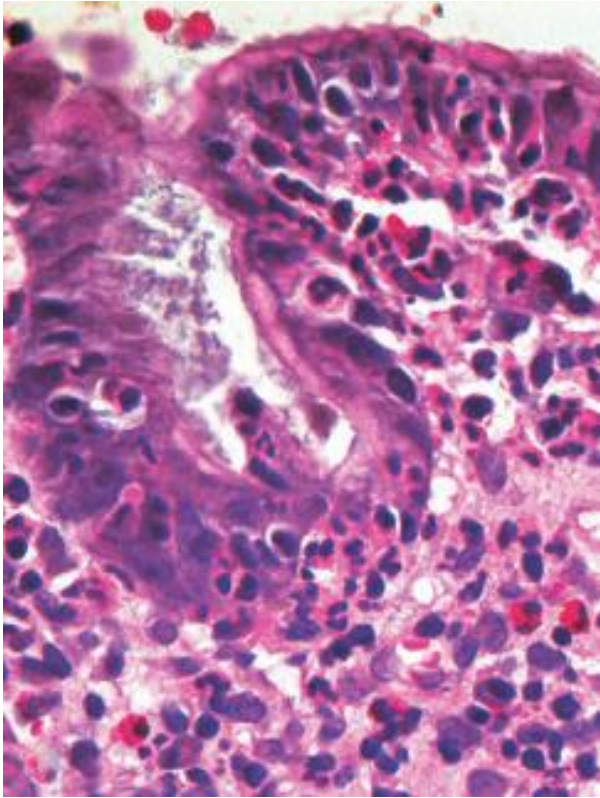
Starts as Antral gastritis >>stimulate G cells >> increased acid production >> peptic ulcer

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

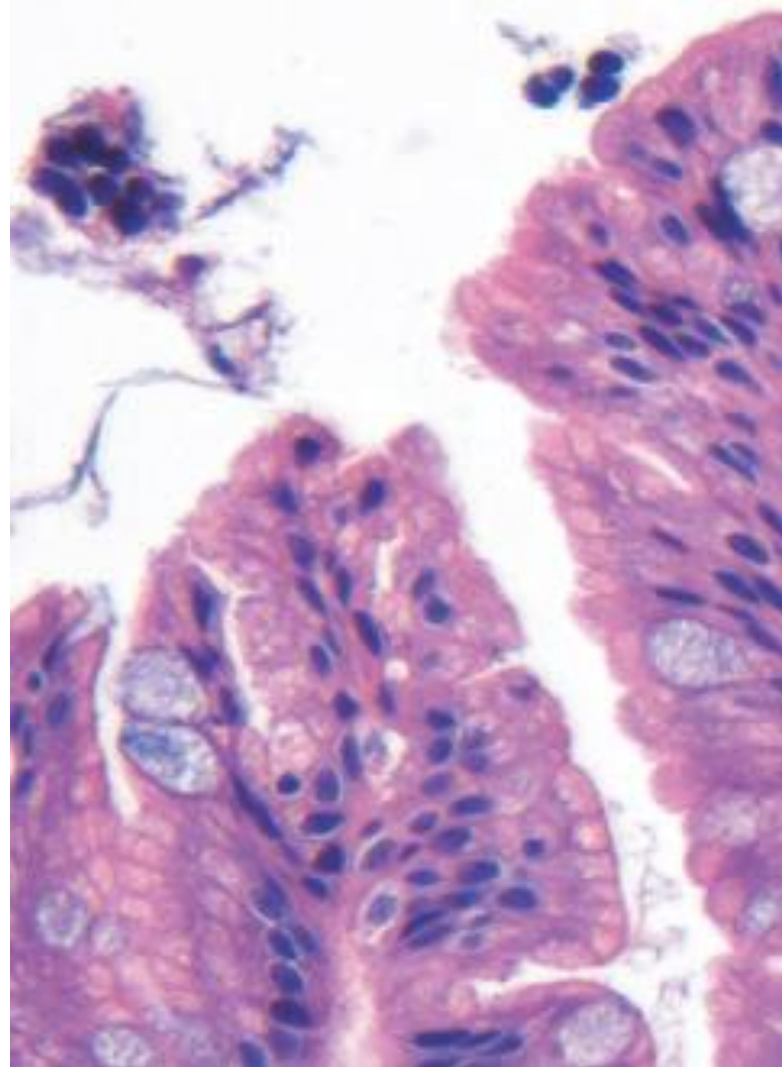
Intestinal metaplasia and increased risk of gastric cancer.

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	<i>H. pylori</i> -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 <i>H. pylori</i>	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

Complication of chronic gastritis

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