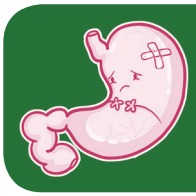

NOTES
GASTRIC DISEASE

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Diseases affecting gastric mucosa, gastric outlet, etc.
- Inflammation due to infection; ulceration

SIGNS & SYMPTOMS

- May be asymptomatic
- Epigastric pain, nausea, vomiting
- Anemia; fecal, urinary incontinence; ulcers; bleeding

DIAGNOSIS

DIAGNOSTIC IMAGING

- Endoscopy

LAB RESULTS

- Biopsy

TREATMENT

MEDICATIONS

- Proton pump inhibitor (PPI)
- Correct fluid, electrolyte deficits
- Discontinue nonsteroidal anti-inflammatory drugs (NSAIDs)

SURGERY

- Endoscopic ligation/coagulation
- Surgical repair

OTHER INTERVENTIONS

- Dietary modification; exercise
- Avoid smoking

CYCLIC VOMITING SYNDROME

osms.it/cyclic-vomiting

PATHOLOGY & CAUSES

- An uncommon disorder characterized by recurrent episodes of vomiting separated by asymptomatic periods
- *Median onset age:* 5–6 years old

CAUSES

- Cause unknown; triggers may include psychological stress (e.g. interpersonal conflict, holidays) or physical stress (e.g. infections, exhaustion), certain foods (e.g. cow's milk, chocolate, cheese, monosodium glutamate) menses

RISK FACTORS

- Children > adults
  - *In children:* mitochondrial DNA deletions and polymorphisms
- Females > males
- Family history of migraines
- Autonomic abnormalities (elevated sympathetic tone)
- Hypothalamic-pituitary-adrenal activation (Sato variant)
- Chronic cannabis use

COMPLICATIONS

- Erosive esophagitis
- Mallory-Weiss tear
- Dehydration
- Electrolyte imbalance
- Unintended weight loss

SIGNS & SYMPTOMS

- Symptoms tend to develop at night, in the early morning hours, or upon awakening
- Prodromal period is common
- *Gastrointestinal:* vomiting which may include bile or blood; retching, abdominal pain, diarrhea
- *Autonomic:* lethargy, pallor, excessive salivation, low grade fever
- *Neurologic:* headache, photophobia, phonophobia, vertigo
- Social withdrawal

DIAGNOSIS

OTHER DIAGNOSTICS

- History and physical examination
  - No identifiable organic cause
- Diagnostic criteria (Rome IV criteria)
  - ≥ three recurrent, discrete episodes of vomiting in the prior year, with two episodes in the past six months occurring at least one week apart
  - Variable intervals between vomiting episodes and asymptomatic baseline
  - Stereotypical characteristics regarding timing of onset, symptoms, and duration

TREATMENT

OTHER INTERVENTIONS

- During cyclic vomiting episodes
  - IV fluids, antiemetics, sedatives; comfort care in dark, quiet room

Prevention

- Prophylactic therapy
  - H1-antagonists (e.g. cyproheptadine) for children ≤ five years old
  - Tricyclic antidepressants (e.g. amitriptyline) > years of age
- Abortive therapy
  - Triptans; neurokinin-1 receptor antagonists
- Avoidance of triggers

GASTRIC DUMPING SYNDROME

osms.it/gastric-dumping

PATHOLOGY & CAUSES

- Iatrogenic post-gastric surgery syndrome; impaired gastric motility → rapid stomach emptying
- Surgical intervention → disruption in gastric anatomy, mucosal function, fundus tone, antropyloric regulatory mechanisms, duodenal feedback on motility → rapid emptying of stomach contents into duodenum
- 50% of individuals undergoing gastric surgical procedures
- More common in individuals who are biologically female

SIGNS & SYMPTOMS

- *GI:* early satiety; abdominal colic; nausea, vomiting; explosive diarrhea; bloating; malabsorption
- *Vasomotor:* diaphoresis; palpitations; vertigo
- Early dumping syndrome
  - 30–60 minutes post-meal
  - Accelerated stomach emptying → hyperosmolar contents poured into small bowel → osmotic activity → bowel distention, motility stimulated → GI symptoms
- Late dumping syndrome
  - 60–180 minutes post-meal
  - Accelerated stomach emptying → ↑ carbohydrate concentration in proximal intestine → rapid glucose absorption → rapid, sustained insulin response → hypoglycemia → vasomotor symptoms

DIAGNOSIS

DIAGNOSTIC IMAGING

- Endoscopy

LAB RESULTS

- Oral glucose challenge test elicits symptoms
- Hydrogen breath test after glucose ingestion

OTHER DIAGNOSTICS

- Gastric emptying study
- Clinical indices
  - *Sigstad's diagnostic index:* > 7
  - *Visick classification:* heart rate variations after oral glucose challenge

TREATMENT

MEDICATIONS

Acarbose

- Interferes with carbohydrate reabsorption

Octreotide

- Inhibits insulin release

OTHER INTERVENTIONS

Dietary modification

- Avoid simple sugars, fluid intake during meals; low carbohydrate, high protein diet

GASTRITIS

osms.it/gastritis

PATHOLOGY & CAUSES

- Inflammation of the lining of the stomach
- May occur as a short episode or may be of a long duration

TYPES

Acute gastritis

- Inflammation of gastric mucosa; compare to gastropathy (without active inflammation)
- Gastritis, gastropathy
  - Clinically identical, histologically distinct

Atrophic gastritis

- AKA chronic gastritis, metaplastic gastritis, gastric atrophy
- Chronic inflammation of gastric mucosa → epithelial metaplasia, mucosal atrophy, gland loss
  - *Metaplasia:* reversible change of one epithelium into another, response to stress
  - *Intestinal metaplasia*: goblet cells

CAUSES

Acute gastritis

- Certain medications, alcohol, corticosteroids, uremia
- NSAIDs block cyclooxygenase → ↓ prostaglandin E2, I2 production → ↓ gastric defense mechanisms (mucus, HCO_3 secretion) → mucosal injury
- *H. pylori* infection → gastric mucosa infiltrates antrum, corpus → inflammation involving neutrophil, mononuclear cells
- Alcohol, cigarette smoke, caffeine → irritates, erodes stomach mucosa lining
- Extreme physiological stress (e.g. shock, sepsis, burns)

Atrophic gastritis

- *Two main causes:* infectious and autoimmune
  - Infectious
    - Most common cause (80%)
    - *H. pylori* → chronic gastritis → gastric atrophy → metaplasia → dysplasia → cancer (associated with intestinal-type gastric carcinoma)
    - Cytotoxin-associated gene A (CagA); carcinogenic virulence factor of *H. pylori*
    - Normal gastrin levels, no hypochloridia, no anti-parietal cell/anti-intrinsic factor antibodies (compare to autoimmune atrophic gastritis; hypochloridia, anti-parietal/anti- intrinsic factor antibodies)
    - Gastric ulcers

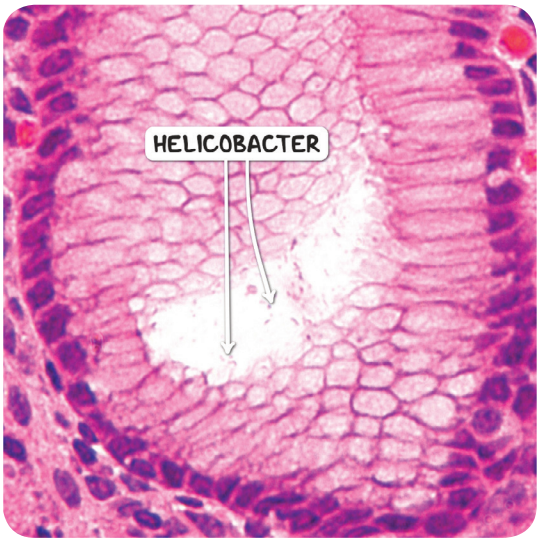

Figure 31.1 A high magnification image of Helicobacter organisms within a gastric crypt. Helicobacter are a common cause of gastritis.

  - Autoimmune
    - Most common cause in individuals without *H. pylori*
    - Inherited autoimmunity against intrinsic factor, H^+/K^+ ATPase in parietal cells → inhibition of gastric acid secretion (hypochloridia). ↓ intrinsic factor →

cobalamin (B_{12}) malabsorption → pernicious anemia
    - Hypochloridia (impaired iron absorption /G-cell hyperplasia, hypergastrinemia → ↑ neuroendocrine tumor formation)
    - ↑ gastric adenocarcinoma, neuroendocrine tumors
    - Damage limited to gastric fundus, body

RISK FACTORS

Atrophic gastritis

- Infectious
    - Household crowding; rural areas; poor sanitation
- Autoimmune
    - Associated with HLA-DR3, B8, other autoimmune diseases; more common in biologically-female individuals

SIGNS & SYMPTOMS

- May be asymptomatic
- Epigastric pain, nausea, vomiting
- Mucosal ulcers
- Hemorrhage, hematemesis, melena

Autoimmune atrophic gastritis

- Iron deficiency anemia
    - Hypochlorhydria → dietary iron in ferric form → ↓ iron absorption → iron deficiency
- Pernicious anemia (symmetrical neuropathy predominantly affecting lower limbs)
    - Anti-intrinsic factor (IF) antibodies, ↓ cobalamin (B_{12}) absorption → depletion of 5-methyl-tetrahydrofolate → homocysteine cannot convert into methionine → impaired myelin regeneration → subacute combined degeneration of spinal cord posterior columns
    - Weakness, paraplegia, paresthesias, ataxia, loss of position/vibration sense
    - Spasticity, clonus; atrophic glossitis; fecal/urinary incontinence; diarrhea; dementia

DIAGNOSIS

LAB RESULTS

Endoscopic biopsy

- Distinguish gastropathy from gastritis, nonspecific; mucosal erosions, erythema, absence of rugae
- Infectious atrophic gastritis
    - Multifocal atrophy; gastric/duodenal ulcers; erythematous, nodular mucosa; thickened rugal folds in early disease, loss of rugal folds in late disease; damage limited to gastric antrum
- Autoimmune atrophic gastritis
    - Diffuse atrophy, absent rugae, mucosal thinning, visible submucosal blood vessels

H. pylori detection

- Serology, stool antigen test, urease breath test, biopsy
- Atrophic gastritis
    - *H. pylori* curved bacilli (hematoxylin, eosin; Giemsa; Warthin-Starry stain); intraepithelial neutrophil, plasma cell invasion

Other lab results

- Autoimmune atrophic gastritis
    - Anti-IF antibodies, anti-parietal cell antibodies
    - ↑ *serum gastrin*: parietal cell loss → achlorhydria → unrestricted gastrin secretion
    - ↓ *serum pepsinogen:* gastric oxyntic mucosa damaged → ↓ chief cells → ↓ serum pepsinogen
    - Lymphocytosis, eosinophilia, plasma cell invasion; oxyntic gland destruction; metaplasia (intestinal, pyloric, pancreatic)

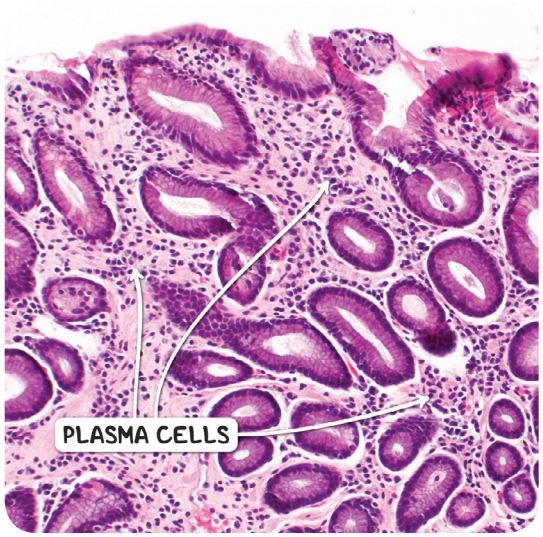

Figure 31.2 Histological appearance of chronic gastritis. The lamina propria contains numerous plasma cells.

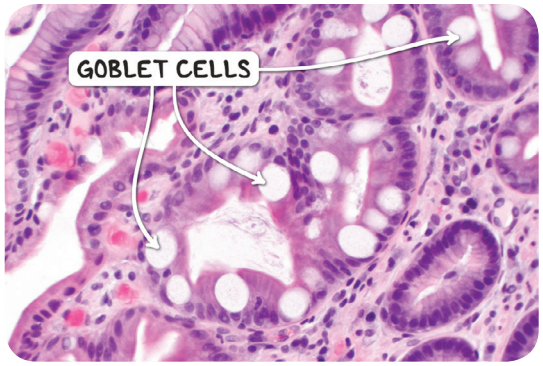

Figure 31.3 The histological appearance of intestinal metaplasia, characterized by the presence of goblet cells in the gastric mucosa.

TREATMENT

MEDICATIONS

Remove offending agents

- NSAIDs, acids/alkalis

Eradicate *H. pylori*

- Triple therapy
  - PPI + clarithromycin + amoxicillin (2 weeks)
- Quadruple therapy
  - PPI + bismuth + metronidazole + tetracycline (1 week)

Correct vitamin deficiencies

- For Autoimmune atrophic gastritis

CHRONIC ATROPHIC GASTRITIS OVERVIEW

	AUTOIMMUNE	INFECTIOUS
ETIOLOGY	$CD4^+$ cells vs. H^+/K^+ ATPase in gastric cells	*H. pylori* infection
AFFECTED AREAS	Body, fundus	Antrum
KEY MECHANISMS	Hypochloridia ↑ gastrin	Urease production by *H. pylori*
CLINICAL PRESENTATION	Mostly asymptomatic until severe Symptoms of vitamin deficiencies Malabsorption	Epigastric pain
HISTOLOGY	Lymphocytic infiltrate	Neutrophilic infiltrate
COMMON COMPLICATIONS	Pernicious anemia Iron deficiency anemia Carcinoid tumors	Peptic ulcers Mucosa-associated lymphoid tissue lymphomas (MALTomas)
COMMON ASSOCIATIONS	Autoimmune diseases	Low socioeconomic status

GASTROPARESIS

osms.it/gastroparesis

PATHOLOGY & CAUSES

- Delayed gastric emptying, no mechanical obstruction

CAUSES

- Most common cause
  - Idiopathic/diabetes
- Iatrogenic (post-surgical/medication side effect), post-viral
- More common among individuals with T1DM than T2DM secondary to neuropathy

SIGNS & SYMPTOMS

- Chronic nausea, vomiting
- Early satiety, bloating
- Abdominal pain

DIAGNOSIS

DIAGNOSTIC IMAGING

Endoscopy, CT scan, MRI

- Exclude mechanical obstruction

Gastric emptying scintigraphy

TREATMENT

MEDICATIONS

- Metoclopramide (gastrointestinal prokinetic)
- Remove medications that may delay gastric emptying

OTHER INTERVENTIONS

- Exercise; low fat diet

PEPTIC ULCER

osms.it/peptic-ulcer

PATHOLOGY & CAUSES

- Chronic mucosal ulceration of stomach/duodenum extends into muscularis mucosa.
- Most common cause of upper gastrointestinal bleeding; proximal duodenum/gastric antrum
- Associated with chronic gastritis
- ↑ acid secretion, ↓ protective mechanisms → mucosal damage → ulceration

RISK FACTORS

- *H. pylori* infection (most common)
  - ↑ gastric acid secretion, ↓ duodenal HCO3 secretion
- NSAID
  - Particularly low dose aspirin corticosteroids
- Physiologic stress
  - Cushing's ulcer (intracranial hypertension), Curling ulcer (severe burns)
- Psychological stress
- Hyperchlorydia
- Smoking
- Chronic obstructive pulmonary disease (COPD)
- Hypergastrinemia (Zollinger-Ellison syndrome)

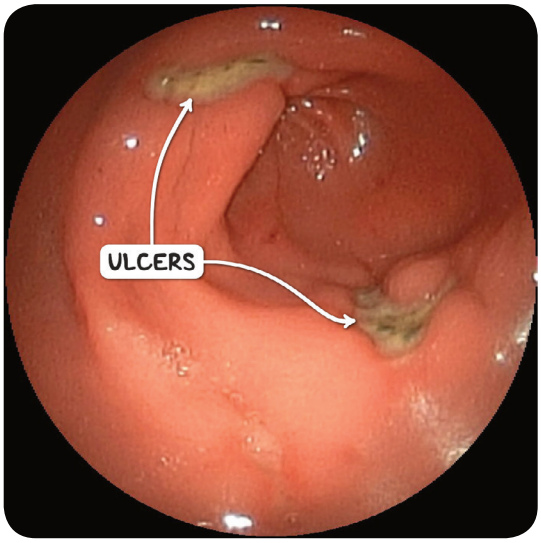

Figure 31.4 An endoscopic view of the gastric antrum which displays two discrete ulcers.

SIGNS & SYMPTOMS

- Up to 70% asymptomatic
- Epigastric burning pain; may mimic myocardial infarction
  - Usually occurs few hours after meal, worsens at night
  - Pain characteristically relieved by food/antacids
- Pain may radiate to back, chest, left/right upper abdominal quadrants
- Nausea, vomiting, coffee-ground emesis, bloating, weight loss

- Surgical emergency
  - Hematemesis, melena, positive guaiac test if slow bleed
  - Acute abdomen; abdominal guarding, peritonitis
  - GI obstruction
- Gastric outlet obstruction, fistula formation

DIAGNOSIS

DIAGNOSTIC IMAGING

Abdominal CT scan

Barium abdominal radiography

Endoscopy

- Diagnostic, therapeutic

TREATMENT

MEDICATIONS

- Discontinue NSAIDs, avoid smoking
- PPI

SURGERY

- Endoscopic ligation/coagulation of bleeding ulcers

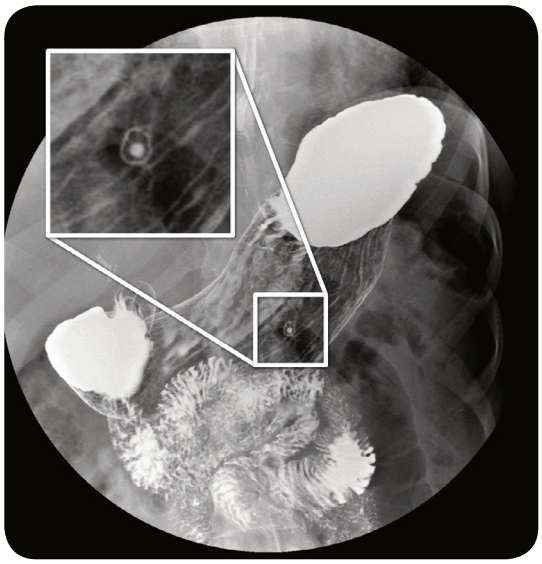

Figure 31.5 A barium study demonstrating the bullseye sign in a case of a gastric ulcer.