STEM-S

LAST MINUTE REVISION

LMR NOTES

INI-SS Sample Notes

PRESENTED BY

Stem-S

Stomach

Blood Supply of Stomach

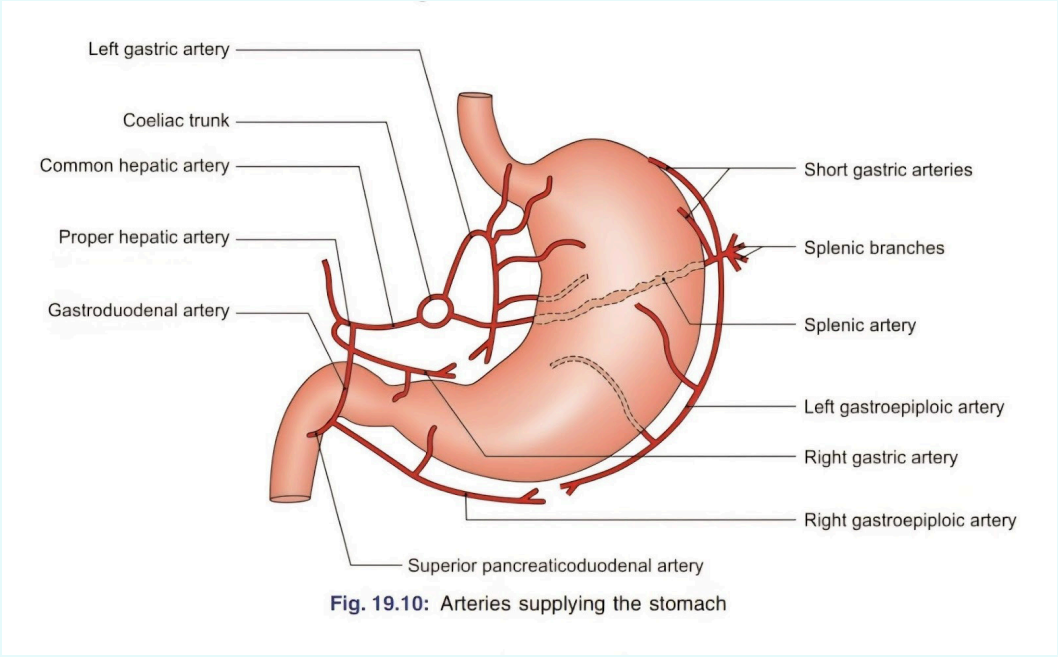


Fig. 19.10: Arteries supplying the stomach

Area of Stomach	Artery	Parent Vessel
Fundus	Short gastric arteries (5–7)	Splenic artery
Upper lesser curvature	Left gastric artery	Celiac trunk
Lower lesser curvature (near pylorus)	Right gastric artery	Proper hepatic artery
Upper greater curvature	Left gastroepiploic artery	Splenic artery
Lower greater curvature	Right gastroepiploic artery	Gastroduodenal artery
Pyloric region	Right gastric + Right gastroepiploic	Hepatic / GDA

Microscopy of Stomach –Oxyntic & Pyloric Cells

- Gastric mucosa contains gastric pits → gastric glands.
- Two main histologic regions:Oxyntic (fundic/body) glands and Pyloric glands.
- Oxyntic glands → acid & intrinsic factor secretion.
- Pyloric glands → mucus and gastrin secretion.

Oxyntic (Fundic) Glands

Location:Fundus & Body of stomach

Major cells present:

- Parietal (Oxyntic) cells
- Chief cells
- Mucous neck cells
- Enteroendocrine cells

Parietal Cells (Oxyntic Cells)

5 Hydrochloric acid (HCl)

- Activates pepsinogen → pepsin
- Helps protein digestion
- Provides antimicrobial environment in stomach

9 Intrinsic Factor

- Glycoprotein secreted by parietal cells
- Binds Vitamin B12
- Required for Vitamin B12 absorption in terminal ileum
- Deficiency → Pernicious anemia

Chief Cells

Secretion- Pepsinogen & Gastric lipase

Pyloric Glands

Location:Pyloric antrum

Features

- Deep gastric pits
- Mainly mucus-secreting cells

Pyloric Cells- Mucus - Protects mucosa from acid

Enteroendocrine Cells in Pyloric Region

Cell	Hormone	Function
G cells	Gastrin	↑ Acid secretion
D cells	Somatostatin	↓ Gastrin
EC cells	Serotonin	Motility

Anomalies of the Stomach and Duodenum

Stomach

Anomaly	Incidence	Age at Presentation	Symptoms / Signs	Treatment
Gastric / Antral / Pyloric Atresia	~3/100,000 (with webs)	Infancy	Non-bilious vomiting	Gastroduodenostomy / Gastrojejunostomy
Pyloric or Antral Membrane (Web)	Similar incidence	Any age	Failure to thrive, vomiting	Incision / excision, pyloroplasty
Microgastria	Rare	Infancy	Vomiting, malnutrition	Continuous drip feeds / jejunal reservoir pouch
Gastric Diverticulum	Rare	Any age	Usually asymptomatic	Usually no treatment required
Gastric Duplication	Rare (M:F = 1:2)	Any age	Abdominal mass, vomiting, hematemesis; rupture → peritonitis	Excision / partial gastrectomy
Gastric Teratoma	Rare	Any age	Upper abdominal mass	Surgical resection
Gastric Volvulus	Rare	Any age	Vomiting, refusal to feed	Reduction of volvulus + anterior gastropexy
Pyloric Stenosis (Infantile hypertrophic)	USA ~3/1000; M:F = 4:1	Infancy	Projectile non-bilious vomiting	Ramstedt pyloromyotomy
Congenital Absence of Pylorus	Rare	Childhood / adulthood	Dyspepsia (if symptomatic)	Usually none

Duodenum

Anomaly	Incidence	Age at Presentation	Symptoms / Signs	Treatment
Duodenal Atresia / Stenosis	~1/20,000	Newborn	Bilious vomiting, upper abdominal distention	Duodenojejunostomy / Gastrojejunostomy
Annular Pancreas	~1/10,000	Any age	Bilious vomiting, failure to thrive	Duodenojejunostomy
Duodenal Duplication Cyst	Rare	Any age	GI bleeding, abdominal pain	Excision
Malrotation with Midgut Volvulus	Rare	Any age	Bilious vomiting, upper abdominal distention	Reduction of volvulus + division of Ladd bands ± resection

Management

- Correct hypochloremic metabolic alkalosis
- Surgery only after metabolic correction.
- Definitive Treatment- Ramstedt pyloromyotomy (Longitudinal incision of hypertrophied pyloric muscle)

Acute Phlegmonous Gastritis

- Rare, severe bacterial infection of the gastric wall characterized by suppurative inflammation of the submucosa and muscularis of the stomach
- Often progresses rapidly → sepsis and high mortality if untreated

Etiology

Most commonly due to bacterial infection, especially:

- Streptococcus species (most common)
- Staphylococcus aureus
- Escherichia coli

Clinical Features

- Acute severe epigastric pain
- Fever
- Nausea ,vomiting and hematemesis
- Signs of sepsis or peritonitis

Characteristic sign:Severe epigastric tenderness

Diagnosis

Laboratory

- Leukocytosis
- Elevated inflammatory markers

Imaging

CT scan (most useful)

Findings:

- Diffuse gastric wall thickening and edema
- Intramural abscess

Culture

- Gastric aspirate or blood culture identifies organism

Medical Management

- Third-generation cephalosporin + metronidazole or Piperacillin–tazobactam

Surgical Management (Partial or total gastrectomy)

Indications:

- Failure of medical therapy
- Gastric necrosis
- Perforation
- Abscess formation

Emphysematous Gastritis

- Rare, life-threatening infection of the stomach characterized by gas formation within the gastric wall due to gas-forming organisms.
- Associated with severe systemic toxicity and high mortality.
- Important to differentiate from gastric emphysema (benign).

Etiology (Causative Organisms)

Common gas-forming bacteria:

- Clostridium species
- Escherichia coli
- Streptococcus
- Staphylococcus aureus
- Pseudomonas
- Sometimes polymicrobial infection.

Pathogenesis

Mucosal injury→ Bacterial invasion of gastric wall→ Gas production in submucosa/muscularis→ Gastric wall necrosis→ Sepsis

Clinical Features

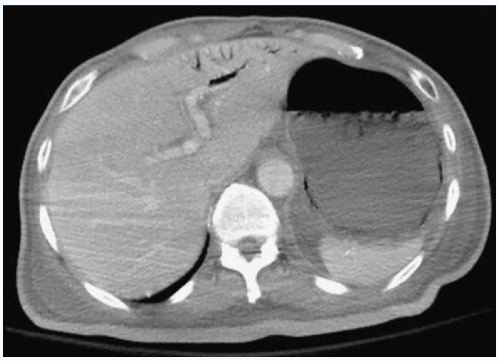
- Severe abdominal pain
- Nausea and vomiting
- Hematemesis
- Fever
- Abdominal distension
- Signs of sepsis or shock

Physical findings:

- Epigastric tenderness

Diagnosis

CT scan – Investigation of choice



CT of emphysematous gastritis showing curvilinear air in the posterior wall of the fluid-filled stomach, as well as portal venous gas.

4. Pathogenetic Factors of H. pylori

Factor	Function / Role in Pathogenesis
Urease enzyme	Converts urea → ammonia + CO_2; neutralizes gastric acid and enables survival
Flagella	Motility through gastric mucus to reach epithelium
Adhesins (BabA, SabA)	Allow adhesion to gastric epithelial cells
CagA protein (Cytotoxin-associated gene A)	Injected into host cells → inflammation, epithelial damage, ↑ cancer risk
VacA toxin (Vacuolating cytotoxin)	Causes epithelial cell injury and vacuolation
Lipopolysaccharide (LPS)	Stimulates immune response and inflammation
Proteases & phospholipases	Damage gastric mucus barrier
Mucinase	Degrades mucus layer facilitating colonization

5. Pathophysiologic Outcomes

Pattern of Infection	Result
Antral predominant gastritis	↑ Gastrin → ↑ acid → Duodenal ulcer
Corpus predominant gastritis	Gland atrophy → ↓ acid → Gastric carcinoma risk
Chronic inflammation	MALT lymphoma

INI-SS High-Yield Pearls

- Urease is the key survival enzyme of H. pylori.
- CagA-positive strains → higher risk of gastric cancer.
- VacA toxin → epithelial cell damage.
- Antral infection → hyperacidity → duodenal ulcer.
- Corpus infection → atrophic gastritis → gastric carcinoma.

Tests for Helicobacter pylori Infection – High-Yield Table

Endoscopic Tests

Test	Advantages	Disadvantages
Biopsy Urease Test (Rapid urease test)	Rapid results; Accurate if patient not on PPIs or antibiotics; No extra pathology cost	Requires endoscopy; Less accurate after treatment or with PPI use
Histology	Excellent sensitivity & specificity; Special stains improve detection; Provides information on gastric mucosa (gastritis, atrophy, dysplasia)	Expensive (endoscopy + pathology); Interobserver variability; Accuracy affected by PPI or antibiotics
Culture	Specificity ≈100%; Allows antibiotic sensitivity testing	Difficult culture technique; Not widely available; Expensive

Non-Endoscopic Tests

Test	Advantages	Disadvantages
Serology (IgG antibody)	Widely available; Inexpensive; Good negative predictive value (NPV)	Poor positive predictive value (PPV) when prevalence is low; Not useful after treatment (antibodies persist)
Urea Breath Test (^{13}C / ^{14}C)	Detects active infection; High sensitivity & specificity; Useful before and after treatment	Limited availability in some settings; Accuracy affected by PPI/antibiotics; Small radiation exposure with ^{14}C
Stool Antigen Test	Detects active infection; Useful before and after treatment; Accuracy not affected by prevalence	Fewer validation data; Accuracy affected by PPI/antibiotics

INI-SS High-Yield Pearls

- Best non-invasive test for active infection → Urea breath test
- Best test to confirm eradication → Urea breath test or stool antigen
- Serology cannot distinguish active vs past infection
- Culture useful for antibiotic resistance testing
- PPIs reduce sensitivity of urease test, breath test, and stool antigen test

Quick Recall

- Active infection tests → Urea breath test, Stool antigen
- Past exposure test → Serology
- Endoscopic rapid test → Rapid urease test (CLO test)
- Resistance testing → Culture