🩺 Gastrointestinal Pathology

Stomach

I. 👶 Gastroschisis

📌 Definition

- Congenital defect of the anterior abdominal wall
- Abdominal contents protrude outside the body

⚙️ Pathogenesis (Flowchart)

Failure of proper abdominal wall formation → Defect develops beside umbilicus → Intestines herniate outside

abdominal cavity → Exposure to amniotic fluid → Inflamed/damaged bowel

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🔍 Key Feature

- Herniated bowel is NOT covered by peritoneum or amnion ⚠️

⚖️ Gastroschisis vs Omphalocele

Feature	Gastroschisis	Omphalocele
Covering membrane	❌ Absent	✅ Present
Location	Beside umbilicus	Through umbilicus
Cause	Abdominal wall defect	Failure of bowel return

🎯 Exam Focus

- No covering membrane = classic clue
- Exposure to amniotic fluid causes bowel irritation

II. 🧩 Omphalocele

📌 Definition

- Persistent herniation of bowel into umbilical cord

⚙️ Embryologic Mechanism (Flowchart)

During development:

Midgut normally herniates into umbilical cord → Later returns to abdominal cavity

Failure of return → Persistent herniation → Omphalocele

🔍 Key Feature

Contents are covered by:

- Peritoneum
- Amnion

⚠️ Important distinction from gastroschisis

🧠 Concept Insight

Physiologic herniation is normally temporary during fetal development.

👉 Failure of return = omphalocele.

🎯 Exam Focus

- Membrane-covered herniation through umbilicus
- Embryology is frequently tested

III. 🤮 Pyloric Stenosis

📌 Definition

- Congenital hypertrophy of pyloric smooth muscle
- Causes gastric outlet obstruction

👶 Epidemiology

- More common in males

⚙️ Pathogenesis (Flowchart)

Hypertrophy of pyloric muscle → Narrowing of pyloric channel → Gastric outlet obstruction → Forceful gastric contractions → Projectile vomiting

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🔍 Classic Presentation

Usually appears around:

📍 2 weeks after birth

📊 Classic Clinical Triad

Feature	Explanation
Projectile nonbilious vomiting 🤮	Obstruction proximal to duodenum
Visible peristalsis	Strong gastric contractions

Olive-like abdominal mass	Hypertrophied pylorus

🧠 Concept Insight

Why is vomiting nonbilious?

Obstruction occurs:

- Before bile enters GI tract

👉 Therefore bile is absent.

🛠️ Treatment

- Myotomy (surgical splitting of muscle)

🎯 Exam Focus

- Projectile nonbilious vomiting = classic buzzword ⭐
- Olive-shaped mass is highly tested

IV. 🔥 Acute Gastritis

📌 Definition

- Acute inflammation due to acidic injury of gastric mucosa

⚖️ Normal Gastric Defense Mechanisms

Defense	Function
Mucin layer	Protects epithelium

Bicarbonate secretion	Neutralizes acid
Adequate blood flow	Removes leaked acid + supplies nutrients

⚙️ Pathogenesis (Flowchart)

Imbalance between acid and mucosal defenses → Mucosal injury → Inflammation → Erosion or ulcer formation

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🚨 Major Risk Factors

1. 🔥 Curling Ulcer

Associated with severe burns

Burns → Hypovolemia → Reduced gastric blood flow → Mucosal ischemia → Acute gastritis/ulceration

2. 💊 NSAIDs

NSAIDs → ↓ Prostaglandin synthesis → ↓ Mucus + bicarbonate secretion → Increased acid injury

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3. 🍺 Alcohol

- Direct mucosal irritation

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4. 💉 Chemotherapy

- Damages rapidly dividing mucosal cells

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5. 🧠 Cushing Ulcer

Associated with increased intracranial pressure

↑ Intracranial pressure → Vagal stimulation → ↑ Acid secretion → Gastric ulceration

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6. 🚑 Shock / ICU Stress Ulcers

Shock → Ischemia → Mucosal injury → Multiple stress ulcers

🔬 Morphologic Changes

Lesion	Definition
Inflammation	Superficial injury
Erosion	Loss of superficial epithelium

Ulcer	Loss extending into mucosa

🎯 Exam Focus

- Curling ulcer = burns
- Cushing ulcer = CNS injury
- NSAIDs decrease prostaglandins ⚠️

V. 🧪 Chronic Gastritis

📌 Definition

- Chronic inflammation of gastric mucosa

📊 Types of Chronic Gastritis

Type	Main Cause
Autoimmune gastritis	Autoimmune destruction
H. pylori gastritis	Infection

A. 🧬 Chronic Autoimmune Gastritis

📌 Definition

Autoimmune destruction of:

- Gastric parietal cells

📍 Mainly in:

- Body
- Fundus

⚙️ Pathogenesis (Flowchart)

Autoimmune T-cell attack → Destruction of parietal cells → ↓ HCl secretion → Achlorhydria → ↑ Gastrin secretion → G-cell hyperplasia

AND

↓ Intrinsic factor → Vitamin B12 deficiency → Pernicious anemia

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🧠 Concept Insight

Important Antibodies

- Anti-parietal cell antibodies
- Anti-intrinsic factor antibodies

⚠️ But actual tissue injury is: T-cell mediated (Type IV hypersensitivity)

📊 Clinical Features

Feature	Mechanism
Achlorhydria	Loss of parietal cells
↑ Gastrin	Compensatory response
Pernicious anemia	↓ Intrinsic factor
Intestinal metaplasia	Chronic injury

🔬 Histologic Feature

- Intestinal metaplasia

⚠️ Complication

- Increased risk of:
    - Gastric adenocarcinoma (intestinal type)

🎯 Exam Focus

- Body/fundus involvement
- Pernicious anemia association
- Type IV hypersensitivity

B. 🦠 Chronic H. pylori Gastritis

📌 Definition

- Chronic gastritis caused by Helicobacter pylori

🌍 Importance

- Most common form of gastritis (~90%)

📍 Most Common Site

- Antrum

⚙️ Pathogenesis (Flowchart)

H. pylori colonization → Urease + protease production → Weakening of mucosal defenses → Acute + chronic inflammation → Gastritis

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🧠 Concept Insight

Why is urease important?

Urease converts:

- Urea → ammonia

👉 Ammonia neutralizes acid locally, helping bacteria survive.

🔍 Clinical Features

- Epigastric pain

⚠️ Major Complications

H. pylori gastritis → Peptic ulcer disease → Intestinal metaplasia → Gastric adenocarcinoma

OR

Chronic lymphoid stimulation → MALT lymphoma

💊 Treatment

Triple therapy:

- Proton pump inhibitor
- Two antibiotics

🧪 Confirmation of Eradication

Test	Meaning
Negative urea breath test	Eradication confirmed
Negative stool antigen	Eradication confirmed

⚖️ Autoimmune vs H. pylori Gastritis

Feature	Autoimmune Gastritis	H. pylori Gastritis
Site	Body/fundus	Antrum
Mechanism	Autoimmune	Infection
Acid level	↓ Acid	Variable
Gastrin	↑	Usually normal/increased
Pernicious anemia	Common	Rare
Cancer risk	Adenocarcinoma	Adenocarcinoma + MALT lymphoma

VI. 🔥 Peptic Ulcer Disease (PUD)

📌 Definition

- Solitary mucosal ulcer involving:
  - Proximal duodenum (90%)
  - Distal stomach (10%)

⚙️ General Pathogenesis

Imbalance between:

- Gastric acid/pepsin ⚔️

  AND

- Mucosal defenses 🛡️

→ Mucosal injury → Ulcer formation

A. 🍽️ Duodenal Ulcer

📌 Definition

- Peptic ulcer occurring in proximal duodenum

🦠 Major Cause

- Helicobacter pylori (>95%)

Rare cause:

- Zollinger-Ellison syndrome

⚙️ Pathogenesis (Flowchart)

H. pylori infection → Increased acid exposure to duodenum → Mucosal injury → Chronic inflammation → Duodenal ulcer

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🔍 Clinical Feature

Pain improves with meals 🍞

Reason:

Food temporarily buffers gastric acid.

🧪 Diagnostic Finding

Endoscopic biopsy:

- Ulcer
- Hypertrophy of Brunner glands

🧠 Concept Insight

Role of Brunner Glands

Brunner glands:

- Located in duodenum
- Secrete alkaline mucus

👉 Hypertrophy occurs as protection against excess acid.

📍 Common Site

- Usually anterior duodenum

⚠️ Posterior Duodenal Ulcer Complications

Posterior ulcer penetration → Erosion into

gastroduodenal artery → Massive bleeding 🩸

OR

Penetration into pancreas → Acute pancreatitis

🎯 Exam Focus

- Pain relieved by meals = duodenal ulcer
- Posterior ulcer → gastroduodenal artery bleed ⚠️
- Brunner gland hypertrophy is highly tested

B. 🍔 Gastric Ulcer

📌 Major Causes

Cause	Frequency
H. pylori	Most common (~75%)
NSAIDs	Common
Bile reflux	Less common

⚙️ Pathogenesis (Flowchart)

Mucosal defense damage → Acid injury to stomach lining → Ulcer formation

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🔍 Clinical Feature

Pain worsens with meals 🍽️

Reason:

Food stimulates acid secretion.

📍 Common Location

- Lesser curvature of antrum

⚠️ Complication

Ulcer erosion → Left gastric artery involvement → Bleeding 🩸

🎯 Exam Focus

- Pain aggravated by meals = gastric ulcer
- Lesser curvature is classic site

⚖️ Duodenal vs Gastric Ulcer

Feature	Duodenal Ulcer	Gastric Ulcer
Common site	Proximal duodenum	Lesser curvature
Pain with meals	Improves	Worsens

Main cause	H. pylori	H. pylori/NSAIDs
Cancer association	Rarely malignant	May represent carcinoma ⚠️

D. 🔬 Ulcer vs Carcinoma

📌 Important Principle

Gastric ulcers may mimic carcinoma ⚠️

Therefore:

👉 Biopsy is essential.

🧠 Concept Insight

Duodenal ulcers are almost never malignant

Because:

- Primary duodenal carcinoma is extremely rare

📊 Benign vs Malignant Gastric Ulcer

Feature	Benign Ulcer	Malignant Ulcer
Size	Small (<3 cm)	Large
Margins	Sharp, punched-out	Irregular, heaped-up
Surrounding folds	Radiating folds	Distorted/disrupted
Diagnosis	Biopsy confirms	Biopsy confirms

🎯 Exam Focus

- "Punched-out ulcer" = benign

- "Heaped-up margins" = malignancy ⚠️

VII. ⚠️ Gastric Carcinoma

📌 Definition

- Gastric adenocarcinoma arising from surface epithelium

📊 Types

Type	Key Feature
Intestinal type	Gland-forming mass/ulcer
Diffuse type	Signet ring cells + linitis plastica

A. 🧪 Intestinal Type Gastric Carcinoma

📌 Features

- More common type
- Appears as:
  - Large irregular ulcer
  - Heaped-up margins

📍 Common Site

- Lesser curvature of antrum

⚙️ Pathogenesis (Flowchart)

Chronic gastritis → Intestinal metaplasia → Dysplasia → Adenocarcinoma

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⚠️ Risk Factors

Risk Factor	Mechanism
H. pylori	Chronic inflammation
Autoimmune gastritis	Intestinal metaplasia
Nitrosamines 🍖	Carcinogenic compounds
Blood group A	Increased association

🧠 Concept Insight

Nitrosamines

Found in:

- Smoked foods
- Preserved foods

Strong association in:

- Japan 🇯🇵

🎯 Exam Focus

- Intestinal metaplasia is the major precursor lesion
- H. pylori strongly associated

B. 🧬 Diffuse Type Gastric Carcinoma

📌 Definition

- Diffuse infiltration of stomach wall by:
    - Signet ring cells

🔬 Signet Ring Cells

Contain:

- Intracytoplasmic mucin

Mucin pushes nucleus to periphery

→ "Signet ring" appearance 💍

⚙️ Pathogenesis (Flowchart)

Diffuse infiltration of gastric wall → Desmoplastic reaction → Thickened rigid stomach wall → Linitis plastica ("leather bottle stomach")

🧠 Concept Insight

Desmoplasia

Fibrotic stromal reaction around tumor:

- Makes stomach hard and rigid

⚠️ Important Association

Diffuse type is NOT associated with:

- H. pylori
- Intestinal metaplasia
- Nitrosamines

🎯 Exam Focus

- Signet ring cells = hallmark ⭐

- Linitis plastica is very high-yield

🚨 Clinical Features of Gastric Carcinoma

📌 Usually Presents Late → Poor prognosis 😔

🔍 Clinical Features

Feature	Explanation
Weight loss ⚖️	Cancer cachexia
Abdominal pain	Tumor invasion
Anemia	Chronic bleeding
Early satiety	Reduced stomach capacity

🌑 Paraneoplastic Signs

1. Acanthosis Nigricans
    - Velvety hyperpigmented skin
2. Leser-Trélat Sign
    - Sudden appearance of multiple seborrheic keratoses

🧬 Lymphatic Spread

📍 Virchow Node

- Left supraclavicular lymph node

⚙️ Spread Pathway

Gastric carcinoma → Thoracic duct drainage → Left supraclavicular node → Enlarged Virchow node

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🌍 Distant Metastases

1. 🔵 Sister Mary Joseph Nodule

- Periumbilical metastasis
- More common with intestinal type

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2. 🥚 Krukenberg Tumor

- Bilateral ovarian metastases
- Classically associated with diffuse type

⚙️ Diffuse Type Metastasis Flowchart

Diffuse gastric carcinoma → Spread of signet ring cells → Bilateral ovarian involvement → Krukenberg tumor

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⚖️ Intestinal vs Diffuse Gastric Carcinoma

Feature	Intestinal Type	Diffuse Type
Morphology	Ulcer/mass	Diffuse infiltration
Histology	Glands	Signet ring cells
H. pylori association	Strong	Weak/none
Intestinal metaplasia	Present	Absent
Gross appearance	Irregular ulcer	Linitis plastica

Common metastasis	Sister Mary Joseph nodule	Krukenberg tumor

🗒️ Revision Summary

- Gastroschisis = no membrane covering
- Omphalocele = membrane-covered herniation
- Pyloric stenosis = projectile nonbilious vomiting
- Acute gastritis = acid injury > defenses
- Curling ulcer = burns
- Cushing ulcer = CNS injury
- Autoimmune gastritis → pernicious anemia
- H. pylori = most common gastritis + ulcers + MALT lymphoma
- Duodenal ulcer pain improves with meals
- Gastric ulcer pain worsens with meals

- Posterior duodenal ulcer → gastroduodenal artery bleed
- Benign ulcer = punched-out appearance
- Intestinal gastric carcinoma linked to H. pylori
- Diffuse carcinoma → signet ring cells + linitis plastica
- Virchow node = left supraclavicular metastasis
- Krukenberg tumor = ovarian metastasis

-> The End <-