Gastrointestinal System

Peptic Ulcer Disease (PUD)

Introduction & Epidemiology

- Peptic Ulcer Disease is a gastrointestinal disorder characterized by mucosal erosions or ulcers ≥5 mm in diameter in the stomach (gastric ulcer) or proximal duodenum (duodenal ulcer) resulting from the corrosive action of gastric acid and pepsin.
- Global Prevalence: ~5–10% of adults worldwide at some point in life.
- Higher incidence in:
    - Males (duodenal ulcers)
    - Older adults (gastric ulcers)
    - Smokers, NSAID users
- H. pylori and NSAIDs are the two most common causes.

Etiopathogenesis

Causative Factors:

- **Helicobacter pylori infection** (~70-90% of duodenal and 50–70% of gastric ulcers)
- **NSAIDs** (e.g., Aspirin, Ibuprofen)
- **Stress-related mucosal damage** (critical illness, trauma)
- **Hypersecretory conditions** (e.g., Zollinger-Ellison Syndrome)
- **Lifestyle** (alcohol, caffeine, tobacco, spicy food – contributory but not direct causes)

Pathophysiology:

- Ulcer formation is due to imbalance between aggressive and protective factors:

Aggressive Factors	Protective Factors
Gastric acid (HCl)	Mucus-bicarbonate layer
Pepsin	Prostaglandins
H. pylori	Surface epithelial cells
NSAIDs	Adequate blood flow

Mechanisms:

- H. pylori: causes inflammation, weakens mucosal barrier, increases acid
- NSAIDs: inhibit COX-1 → ↓ prostaglandins → ↓ mucus + bicarbonate

Clinical Features / Symptoms

Duodenal Ulcers

- Pain 2–3 hours after meals or at night
- Relieved by food or antacids
- May present with weight gain

Gastric Ulcers

- Pain worsens with food
- Associated with weight loss
- Nausea, bloating, early satiety

Alarm Symptoms (Red flags)

- Hematemesis (vomiting blood)
- Melena (black tarry stools)
- Anemia
- Sudden severe abdominal pain → perforation
- Dysphagia or persistent vomiting → obstruction

Diagnosis & Investigations

Initial Approach

- Clinical symptoms + risk factor history (NSAID, H. pylori)

Tests for H. pylori

- **Non-invasive:**
  - Urea breath test (gold standard)
  - Stool antigen test
  - Serology (not preferred for active infection)
- **Invasive (if endoscopy planned):**
  - Rapid urease test (CLO test)
  - Histology
  - Culture/PCR

Endoscopy (EGD)

- For >55 years, alarm features, or non-responsive to therapy
- Helps to:
  - Confirm ulcer
  - Biopsy for H. pylori and malignancy

Treatment Goals / Therapeutic Objectives

- Eradicate H. pylori (if present)
- Relieving pain and symptoms
- Heal ulcer completely
- Prevent recurrence
- Avoid complications (bleeding, perforation, obstruction)
- Prevent NSAID-induced ulcers in high-risk patients

Non-Pharmacological Therapy

- Avoid NSAIDs, alcohol, caffeine, spicy foods
- Smoking cessation
- Avoid eating 2–3 hrs before bed
- Reduce psychological stress
- Adequate sleep and dietary hygiene

Pharmacological Therapy

First-line (based on etiology)

1. H. pylori-positive ulcers (10–14 days therapy)

A. Standard Triple Therapy:

- PPI (e.g., omeprazole 20 mg BID)
- Clarithromycin 500 mg BID
- Amoxicillin 1 g BID *(or Metronidazole 500 mg BID if penicillin allergic)*

B. Quadruple Therapy:

- PPI BID
- Bismuth subsalicylate 525 mg QID
- Tetracycline 500 mg QID
- Metronidazole 500 mg TID

C. Concomitant Therapy:

- PPI + Clarithromycin + Amoxicillin + Metronidazole
- Duration: 10–14 days; Eradication should be confirmed.

2. NSAID-Induced Ulcers

- Discontinue NSAID if possible
- PPI (e.g., Pantoprazole 40 mg OD) for 4–8 weeks

- Misoprostol (PGE1 analog): 200 mcg QID
- H2RAs (e.g., Ranitidine 150 mg BID): less effective
- COX-2 selective inhibitors if NSAID is necessary

Alternative drugs

- **Sucralfate**: Mucosal protectant; 1 g QID before meals
- **Antacids**: Symptom relief; not ulcer healing

Individualized Therapeutic Plan

Patient Factor	Therapeutic Consideration
Elderly	Avoid NSAIDs; consider H. pylori eradication even if asymptomatic
Pregnancy	Avoid tetracyclines, misoprostol; PPI and amoxicillin are safe
Renal Impairment	Avoid high-dose aluminum or magnesium antacids
Hepatic Impairment	Adjust PPI dose if severe liver disease
Allergies	Clarithromycin or amoxicillin alternatives needed

Controversies / Special Considerations

- **Antibiotic resistance** in H. pylori: clarithromycin resistance rising; necessitates local antibiogram-based regimens
- **Test of cure**: Controversy over timing (≥4 weeks post-therapy)
- **Long-term PPI use risks**: Bone fractures, infections (C. difficile), B12 deficiency

Latest Guidelines:

- Maastricht VI (2022): Emphasizes bismuth-based quadruple therapy as first-line in areas with high resistance
- ACG 2022: Advocates for tailored therapy based on local resistance

Monitoring Therapy

Parameter	Monitoring Tool	Time Frame
Symptom relief	Patient history	1–2 weeks
H. pylori eradication	Urea breath test / Stool antigen	≥4 weeks after therapy
Ulcer healing	Endoscopy (only if complications or recurrence)	6–8 weeks
Adverse effects	Liver enzymes, GI symptoms	Throughout treatment

Adverse Effects & Management

Drug	Common Side Effects	Management
PPIs	Headache, diarrhea, B12 deficiency (long-term)	Supplement B12, limit prolonged use
Clarithromycin	Taste disturbance, QT prolongation	Monitor ECG in cardiac patients
Metronidazole	Metallic taste, disulfiram reaction	Avoid alcohol
Amoxicillin	Rash, diarrhea	Switch if allergic
Misoprostol	Diarrhea, uterine cramps	Contraindicated in pregnancy

Follow-up and Patient Counseling

Follow-up:

- Confirm eradication (breath/stool test after 4 weeks)
- Re-endoscopy only if:
  - Alarm features
  - Refractory ulcer
  - Suspected malignancy (gastric ulcer)

Counseling Points:

- Complete full course of therapy
- Avoid self-medication with NSAIDs
- Diet modification: frequent small meals, avoid irritants
- Report signs of GI bleeding: black stools, coffee-ground vomitus
- Importance of follow-up tests
- Emphasize medication adherence for eradication success

Gastroesophageal Reflux Disease (GERD)

Introduction / Epidemiology

- Gastroesophageal Reflux Disease (GERD) is a chronic condition where stomach contents (acid, bile, enzymes) flow backward (reflux) into the esophagus, causing symptoms and/or complications due to mucosal damage.
- **Epidemiology:**
  - Affects 10–20% of adults in Western countries; lower prevalence in Asia (around 5%)
  - Increasing global trend, possibly due to obesity, dietary habits, and sedentary lifestyle
  - Equal distribution among genders; more common with advancing age

First Hope

FREE PREVIEW

Click here to visit our website

PHARMACY PREMIUM NOTES

Complete subject and semesters are available on FirstHope app only.

Scan or click here

Click here to get the app >

Available on Google Play

JOIN THE FIRSTHOPE COMMUNITY

Join our Telegram channel

Click here to join Telegram >

Scan or click here

@FirstHopeNotes

Instagram

@firsthope.official

Click here to follow >

YouTube channel

FirstHope-Pharmacy / Live streams

Click here to watch >

WhatsApp channel

Follow FirstHope on WhatsApp

Click here to join >

FirstHope website

firsthope.co.in

Click here to visit >

B.Pharm / D.Pharm / M.Pharm / Pharm.D

Preview notes. Complete subjects and semesters in the FirstHope app.