

Definition, etiopathogenesis, clinical manifestations, nonpharmacological and pharmacological management of the diseases associated with Gastrointestinal Disorders System

Gastroesophageal Reflux Disease (GERD)

Definition:

- GERD is a chronic condition where stomach contents reflux into the esophagus, causing symptoms and/or complications due to mucosal damage from gastric acid.

Etiopathogenesis:

Pathophysiological Mechanisms:

- Lower Esophageal Sphincter (LES) dysfunction (transient relaxation or hypotension)
- **Hiatal hernia:** Weakening of diaphragm increases reflux
- Delayed gastric emptying
- Impaired esophageal clearance
- Reduced salivary bicarbonate (neutralizing effect lost)

Risk Factors:

- Obesity
- Pregnancy
- Smoking
- Alcohol
- Caffeine
- Certain medications (anticholinergics, NSAIDs, CCBs)
- Diet (fatty, spicy, citrus foods)

Clinical Manifestations:

Typical symptoms:

- Heartburn (burning chest discomfort)
- Regurgitation
- Sour or bitter taste in the mouth
- Chest pain (may mimic angina)

- Worsens after meals or lying down

Atypical/extralaryngeal symptoms:

- Chronic cough
- Hoarseness
- Asthma-like symptoms
- Dental erosion

Complications:

- Esophagitis
- Strictures
- Barrett’s esophagus (precancerous)
- Esophageal adenocarcinoma

Non-Pharmacological Management:

- Weight loss if overweight
- Elevate head of bed by 6–8 inches
- Avoid large meals and late-night eating
- Avoid trigger foods (chocolate, citrus, caffeine, spicy/fatty foods)
- Quit smoking and alcohol
- Avoid tight-fitting clothes
- Chewing gum (stimulates saliva, aids clearance)

Pharmacological Management:

- **Antacids** (e.g., calcium carbonate): Symptom relief
- **H2-receptor blockers** (e.g., ranitidine, famotidine): Reduce acid secretion
- **Proton pump inhibitors (PPIs)** (e.g., omeprazole, esomeprazole): First-line for moderate-severe GERD, healing esophagitis
- **Prokinetics** (e.g., metoclopramide, domperidone): Enhance LES tone and gastric emptying (used rarely)
- **Maintenance therapy** for chronic or severe cases
- **Surgical option:** Fundoplication (Nissen procedure) in refractory cases

Peptic Ulcer Disease (PUD)

Definition:

- PUD refers to ulceration in the gastric or duodenal mucosa that penetrates the muscularis mucosae, often due to the imbalance between mucosal protective factors and aggressive factors like acid and pepsin.

Etiopathogenesis:

Major causes:

- **Helicobacter pylori** infection: Gram-negative bacterium that damages mucosa and increases acid
- **NSAIDs**: Inhibit prostaglandin synthesis → ↓ mucosal defense
- Stress ulcers (burns, trauma, ICU patients)
- Smoking, alcohol, corticosteroids

Pathophysiology:

- Breakdown of mucosal barrier
- Acid and pepsin penetrate deeper layers → ulceration
- H. pylori produces urease → ammonia → mucosal injury
- NSAIDs reduce mucus, bicarbonate secretion

Clinical Manifestations:

Gastric ulcer:

- Epigastric pain worsened by food
- Nausea, vomiting
- Weight loss

Duodenal ulcer:

- Epigastric pain relieved by food but returns after 2–3 hours
- Night-time pain
- Weight gain (eating relieves pain)

Complications:

- GI bleeding (hematemesis, melena)
- Perforation (rigid abdomen, severe pain)
- Gastric outlet obstruction
- Increased risk of gastric cancer (especially with H. pylori)

Non-Pharmacological Management:

- Avoid NSAIDs and aspirin if possible
- Stop smoking and alcohol
- Avoid caffeine and spicy foods
- Eat small, frequent meals
- Stress management
- Eradicate H. pylori (if present)

Pharmacological Management:

1. H. pylori eradication (Triple therapy):

- PPI + Clarithromycin + Amoxicillin (or metronidazole)
- **Duration:** 10–14 days

2. Proton Pump Inhibitors (PPIs):

- Omeprazole, pantoprazole: Promote healing and reduce acid

3. H₂-receptor antagonists:

- Ranitidine, famotidine (less effective than PPIs)

4. Antacids:

- Temporary relief

5. Cytoprotective agents:

- **Sucralfate:** Coats ulcer
- **Misoprostol:** Prostaglandin analog (especially for NSAID ulcers)

Alcoholic Liver Disease (ALD)

Definition:

- ALD encompasses a spectrum of liver injuries due to chronic alcohol intake, ranging from simple steatosis (fatty liver) to alcoholic hepatitis, and ultimately to cirrhosis.

Etiopathogenesis:

- **Excessive alcohol consumption** impairs liver metabolism through:
 - Acetaldehyde accumulation (toxic metabolite)
 - Generation of reactive oxygen species (ROS)



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