PEPTIC ULCER
(Pathophysiology & Therapy)

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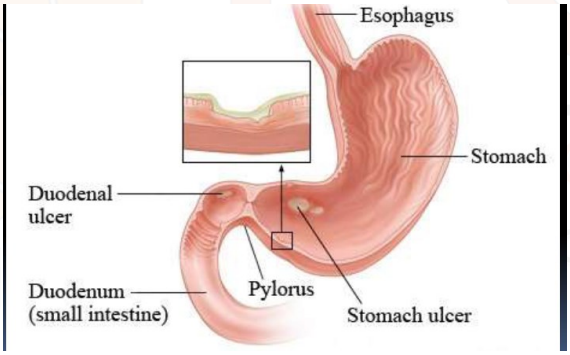
Introduction

- **Peptic Ulcer**, is a lesion (wounds) in the lining (mucosa) of the digestive tract, typically in the stomach or duodenum (upper part of small intestine), caused by the digestive action of pepsin and gastric acid.
- Peptic ulcer comes under the **GI tract disorders**



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Peptic Ulcers

Ulcer: wounds is body membrane

- Peptic Ulcer
- Mouth ulcer
- Diabetic foot ulcer
- Genital ulcer
- Corneal ulcer
- Ulcerative sarcoidosis
- Ulcerative colitis

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Peptic Ulcers

Peptic Ulcer/Peptic Ulcer Disease (PUD)

- Peptic Ulcer is a lesion in the lining (mucosa) of the digestive tract (Stomach and Duodenum), caused by the digestive action of pepsin and stomach acid.

Types:

- **A. Acute Peptic Ulcer**

1. Cushing peptic ulcer: Gastric, duodenal or esophageal ulcer arising in patients with intercranial injury or operation
2. Curling peptic ulcer: Occuring mosty in the proximal duodenum and associated with severe burns and trauma

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Peptic Ulcers

- **B. Chronic Peptic Ulcer**
  1. **Duodenal peptic ulcer (75-80 %)**
  2. **Gastric peptic ulcer (15-20 %)**
  3. Esophageal ulcer: Mostly occur in the lower end of esophagus; associated with bad case of chronic gastro esophageal reflux disease or GERD
  4. Bleeding ulcer: Internal bleeding is caused by a peptic ulcer which has been left untreated; most dangerous.
  5. Refractory ulcer: These are simply peptic ulcers that have not healed after at least 3 months of treatment

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Peptic Ulcers

Gastric vs Duodenal ulcer

	Duodenal Ulcer	Gastric ulcer
Age	Any age, more common in 30-40 y	50-60 year
Sex	Male>Female	Female>Male
Etiology	Helicobecter pylori infection, damaging of mucosal wall	Helicobecter pylori infection, NSAIDs (aspirin, ibuprofen, diclofenac)
Pain	Epigastric	Epigastric can radiate to back
Onset	2-3 h after eating and midnight	Immediately after eating
Agg. by	Hungar	Eating

Peptic Ulcers

Gastric vs Duodenal ulcer

	Duodenal Ulcer	Gastric ulcer
Relived by	eating	Lying down and Vomiting
Vomiting	Uncommon	Common (to relieve pain)
Wight	No weight loss	Weight loss
Hematemesis	40%	60%
Melena	60%	40%

Clinical Symptoms of PUD

- **B. Abdominal pain:** Located in epigastric area, Burning sensation
- Heart burn
- Nausea
- Perforations
- Blotting and abdominal fullness
- Loss of appetite (because of pain): In gastric ulcer
- Weight loss: In gastric ulcer
- Hematemesis
- Melena

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Diagnosis

- Stool examination for fecal occult blood.
- Complete blood count (CBC) for decrease in blood cells.
- Esophago-gastro-deuodeno-scopy (EGD)

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Eitopathogensis

- Peptic ulcer occurs due to imbalance between aggressive and defensive factors that can lead to tissue damages.

Aggressive Factors	Defensive Factors
HCl	Bicarbonate
Pepsin	Mucous
H. Pylori	Prostaglandins (PGE2)
Gastrin	Mucosal blood flow
Bile Acids	

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Eitopathogensis

- **Life Style: Smoking**, acidic drink, junk foods, high alcohol consumption

→ Increase the aggressive factors (pepsin, HCl)

- **H. pylori infection**: →H. pylori bacteria commonly live in mucous layer that covers and protects the tissue. it can cause inflammation and produce ulcer. It can be spread by close contacts. It may cause increase in gastrin and pepsinogen levels.

H. Pylori (Gram –ve Bac)→ produce heat shock proteins→ Cytokines, Histamines, certain enzymes→ Phospholipase, **urease**, protease → in acidic media urease converts urea into **Ammonia** and CO2→ Ammonia can damage the mucosal lining → **Ulcer**

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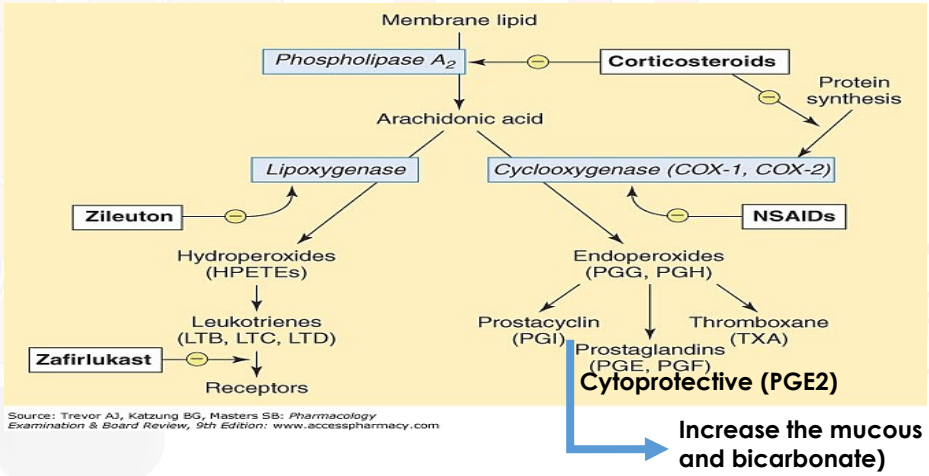
Eitopathogensis

- **NSAIDS:** Aspirin, ibuprofen, diclofenac, etc

→ Decrease the production defensive factors (Prostaglandin)



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Eitopathogensis

- **Chronic Stress may worsen the ulcer**

Stress→ increase energy consumption (glycolysis)→that occurs due to Cortisol hormone → inhibit the PLA2 → inhibit prostaglandin → increase acid secretion and inhibit mucous and bicarbonate secretion → ULCER

- **Zollinger Ellison Syndrome (ZES)**

Tumor of goblet cell→ increase gastrin activity → Increase Acid secretion → ULCER

- Genetic
- Other medications: SSRIs (Fluoxetine), Steroids, Antibiotics, etc

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Physiology of Acid Secretion

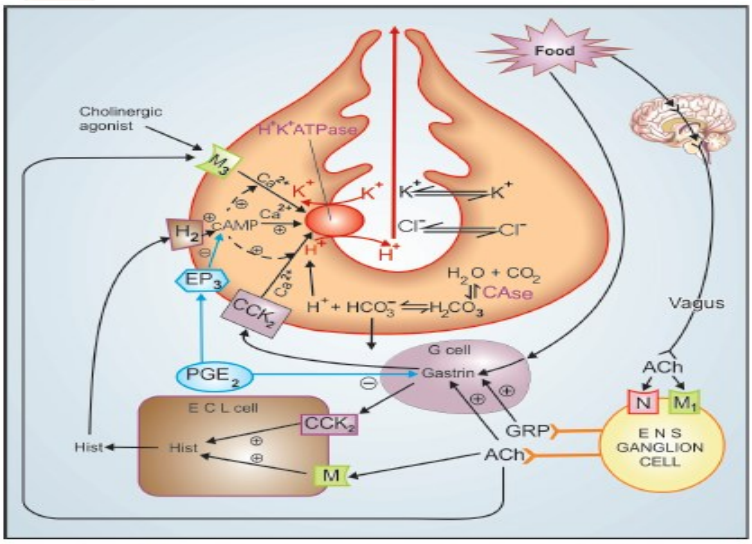

Figure 46.1: Secretion of HCl by gastric parietal cell and its regulation
C.Ase.—Carbonic anhydrase; Hist.—Histamine; ACh.—Acetylcholine; CCK_2—Gastrin cholecystokinin receptor; M.—Muscarinic receptor; N—Nicotinic receptor; H_2—Histamine H_2 receptor; EP_3—Prostaglandin receptor; ENS—Enteric nervous system; ECL cell—Enterochromaffin-like cell; GRP—Gastrin releasing peptide; + Stimulation; – Inhibition.

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Therapeutic Management

Therapeutic Goal:

- Relief of pain
- Ulcer healing
- Prevention of complications (bleeding, perforation)
- Prevention of relapse.

Approaches for the treatment of peptic ulcer are:

1. Reduction of gastric acid secretion

(a) **H2 antihistamines**: Cimetidine, Ranitidine, Famotidine

(b) **Proton pump inhibitors**: Omeprazole, Esomeprazole, Pantoprazole, Rabeprazole,

(c) **Anticholinergic drugs**: Pirenzepine, Propantheline, Oxyphenonium

(d) **Prostaglandin analogue**: Misoprostol

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Therapeutic Managment

2. Neutralization of gastric acid (Antacids)

(a) **Systemic:** Sodium bicarbonate, Sod. citrate

(b) **Nonsystemic:** Magnesium hydroxide, Mag. trisilicate, Aluminium hydroxide gel, Magaldrate,

3. **Ulcer protectives:** Sucralfate, Colloidal bismuth subcitrate (CBS)
4. **Anti-H. pylori drugs:** Amoxicillin, Clarithromycin, Metronidazole,

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