



PEPTIC ULCER

Introduction

- ▶ An ulcer is any discontinuity or breakdown in a bodily membrane that impedes the organ of which that membrane is a part from continuing its normal functions.

PEPTIC ULCER

- ▶ Also known as “peptic ulcer disease”(PUD)
- ▶ Peptic ulcer is a break in the lining of the stomach, small stomach, first part of the small intestine or occasionally the lower esophagus.

Sites of Peptic ulcer

- ▶ Duodenum 80%
- ▶ Stomach 19%
- ▶ Duodenum & Stomach 4%
- ▶ Other sites (1%)
Ex. GE junction, Meckel's diverticulum

Symptoms

- ▶ Abdominal pain : located in epigastric area, burning sensation, occurs in empty stomach 2-4 hours after meal or at night(nocturnal pain)
- ▶ Nausea and vomiting
- ▶ Heartburn
- ▶ Abdominal fullness
- ▶ Loss of appetite
- ▶ Melena

Types of Peptic Ulcer

- ▶ **Acute Ulcer** : Arises generally after any injuries, trauma or operations of GI parts.
- ▶ **Chronic ulcer**
 - Duodenal ulcer (on upper part of SI)
 - Gastric ulcer (inside the stomach)
 - Esophageal ulcer (lining of stomach)
 - Bleeding Ulcer
 - Refractory Ulcer

Etiology

- ▶ No single cause
- ▶ Most common is due *Helicobacter pylori* infection.
- ▶ Use of **NSAIDs drugs** like aspirin, naproxen, ibuprofen
- ▶ Excess acid production
- ▶ Alcoholism, Smoking and Tobaccos
- ▶ Serious illness and stress conditions

PATHOGENESIS OF PEPTIC ULCER



H. pylori INDUCED ULCER

Gram negative bacteria produced heat shock proteins



Cytokines, histamine, lipopolysaccharides, certain enzymes



Phospholipase



Urease, protease, fucosidase etc.

- Urease convert in acidic media urea into ammonia and carbon dioxide. Ammonia itself cause destruction of mucosal lining.

➤ Ammonia cause infection of mucosal lining and ultimately inflammatory mediators release.

➤ Cytokines → Leukocytes adhesion and inflammatory reactions starts

↓
Damage mucosa of GIT

↓
Ulcer occurs

DRUG INDUCED ULCER

Drugs for example NSAIDS as aspirin(non selectively inhibit cox1 and cox2 in human body

Arachidonic acid $\xrightarrow{\text{cox1,2}}$ Prostaglandins

Controls gastric juice secretions

Damage mucosal lining lead to ulcer

STRESS INDUCED ULCER

In stress energy consumption increase so increase glycolysis which is usually done by cortisol hormone



This hormone inhibit phospholipase A2



No arachidonic acid formation no prostaglandin
increase gastric juice secretions



Cause ulcer

STEROIDS INDUCED ULCER

Steroids acts on cell membrane (phospholipid)



Inhibit phospholipase



Inhibits arachidonic acid no prostaglandins and
damaging of mucosal lining

ULCER DUE TO GENETIC DEFECT

Rare genetics occurs some time having blood group O positive the size of parietal cell is increase



Increase cell demand as HCL secretions increase



Cause destruction of mucosal lining leading towards ulcer

TREATMENT AND PREVENTION

► Medications

PPIs - Omeprazole, Rabeprazole, Pantoprazole
etc .

Antibiotics - For H. pylori induced ulcers

Histamine receptor blockers : eg. ranitidine

Changing lifestyle

Surgery (if needed)

Minimizing use of NSAIDs

Focus on balanced diet and healthy foods