

# PEPTIC ULCER DISEASES

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# Definitions

- **Ulcer:**

A lesion on an epithelial surface (skin or mucous membrane) caused by superficial loss of tissue

- **Erosion:**

A lesion on an epithelial surface (skin or mucous membrane) caused by superficial loss of tissue, limited to the mucosa

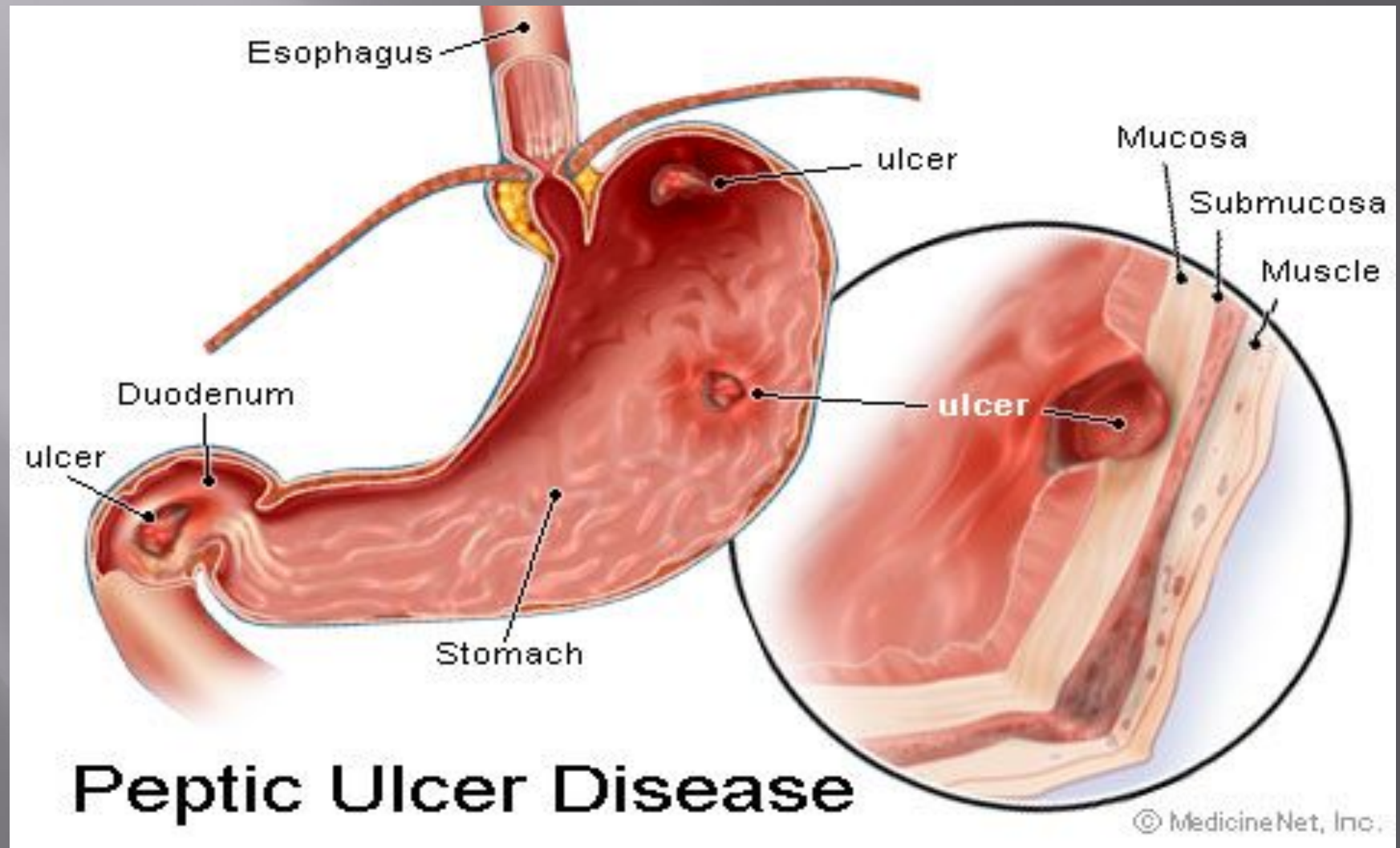
# Definitions

## ■ **Peptic Ulcer**

An ulcer of the alimentary tract mucosa, usually in the stomach or duodenum, & rarely in the lower esophagus, where the mucosa is exposed to the acid gastric secretion

- *It has to be deep enough to penetrate the muscularis mucosa*

# Peptic Ulcer Disease



# Pathophysiology

- A peptic ulcer is a mucosal break, 3 mm or greater in size with depth, that can involve mainly the stomach or duodenum.

# Pathophysiology

Two major variants in peptic ulcers are commonly encountered in the clinical practice:

- 1) *Duodenal Ulcer* (DU)
- 2) *Gastric Ulcer* (GU)

# Pathophysiology

DU result from increased acid load to the duodenum due to:

- 1) Increased acid secretion because of:
  - A. Increased parietal cell mass
  - B. Increased gastrin secretion (e.g. Zollinger-Ellison syndrome, alcohol & spicy food)
- 2) Decreased inhibition of acid secretion, possibly by *H. pylori* damaging somatostatin-producing cells in the antrum

# Pathophysiology

DU result from increased acid load to the duodenum due to:

- 3) Smoking impairing gastric mucosal healing
- 4) Genetic susceptibility may play a role (more in blood gp. O)
- 5)  $\text{HCO}_3$  secretion is decreased in the duodenum by *H. pylori* inflammation



# Pathophysiology

GU results from the break down of gastric mucosa:

- 1) Associated with gastritis affecting the body & the antrum
- 2) The local epithelial damage occurs because of cytokines released from *H. pylori* & because of abnormal mucus production
- 3) Parietal cell damage occur so that acid production is normal or low

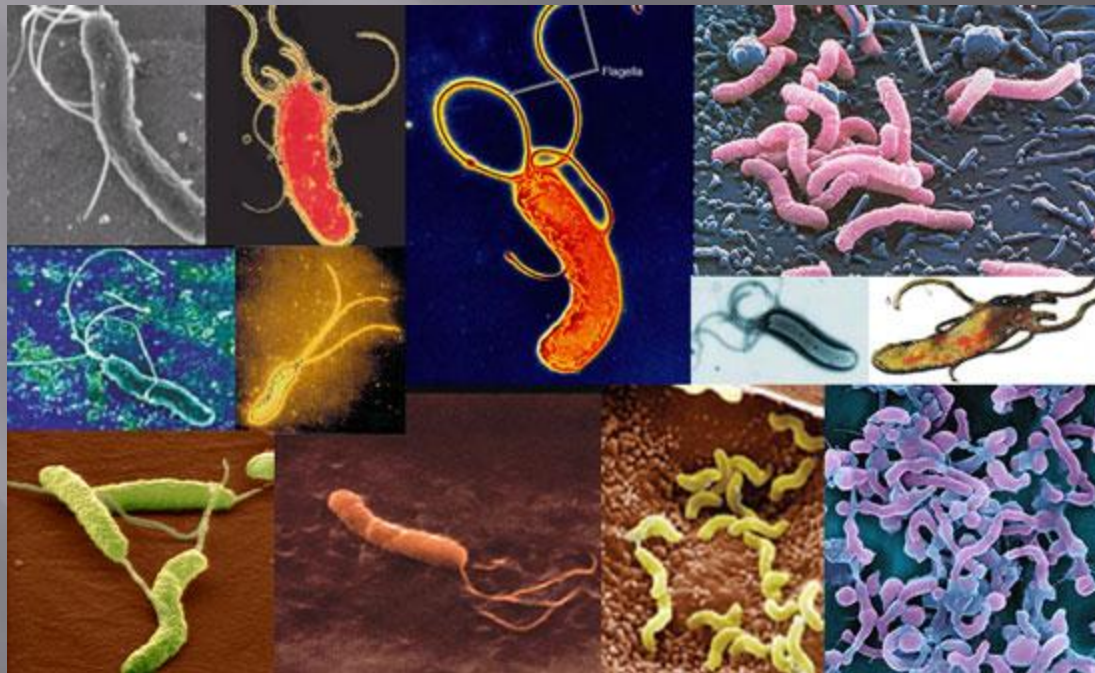
# Etiology

- The two most common causes of PUD are:
  - *Helicobacter pylori* infection ( 70-80%)
  - Non-steroidal anti-inflammatory drugs (NSAIDS)

# Etiology

- ▣ Other uncommon causes include:
  - Gastrinoma (Gastrin secreting tumor)
  - Stress ulceration (trauma, burns, critical illness)
  - Viral infections
  - Vascular insufficiency

# 1. Etiology – *Helicobacter pylori*



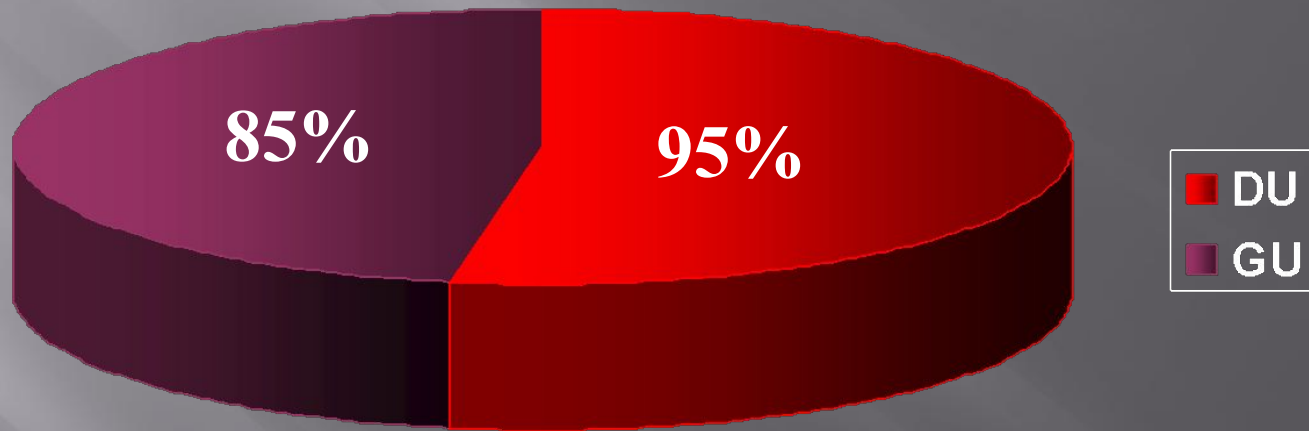
# ***H.pylori* Epidemiology**

- ▣ One half of world's population has *H.pylori* infection, with an estimated prevalence of 80-90 % in the developing world
- ▣ The annual incidence of new *H. pylori* infections in industrialized countries is 0.5% of the susceptible population compared with  $\geq 3\%$  in developing countries

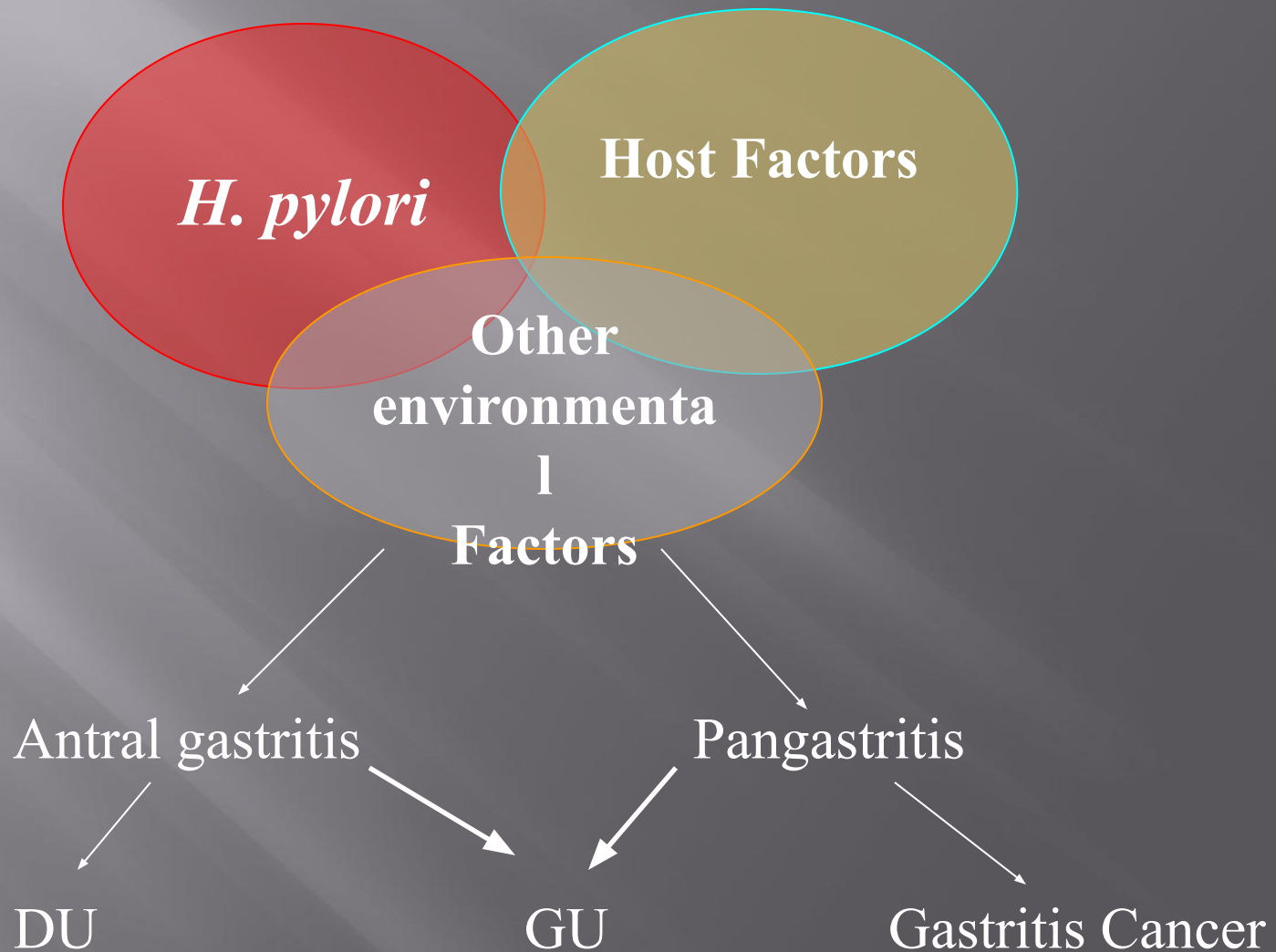
## ***H.pylori* as a cause of PUD**

**The majority of PUD patients are  
*H. pylori* infected**

## *H.pylori* as a cause of PUD



# Carcinogenic effect of *H. pylori*





# Type of NSAID & Risk of Ulcer

Risk Group	Drug	Relative Risk
Low	Ibuprofen	2.0
	Diclofenac	4.2
Medium	Naproxen	9.1
	Indomethacin	11.3
	Piroxicam	13.7
High	Ketoprofen	23.7
	Azapropazone	31.5

# Clinical Presentation

- Recurrent epigastric pain (the most common symptom)
  - Burning
  - Occurs 1-3 hours after meals
  - Relieved by food □ DU
  - Precipitated by food □ GU
  - Relieved by antacids
  - Radiate to back (consider penetration)
  - Pain may be absent or less characteristic in one-third of patients especially in elderly patients on NSAIDs

# Clinical Presentation

- ▣ Nausea, Vomiting
- ▣ Dyspepsia, fatty food intolerance
- ▣ Chest discomfort
- ▣ Anorexia, weight loss especially in GU
- ▣ Hematemesis or melena resulting from gastrointestinal bleeding

# Diagnosis of PUD

# Peptic Ulcer Disease

## Diagnosis:

- 1) Diagnosis of ulcer
- 2) Diagnosis of *H. pylori*

# Diagnosis of PUD

Diagnosis of PUD depends mainly on endoscopic and radiographic confirmation

# Doudenal Ulcer on Endoscopy



**Normal doudenal bulb**



**Doudenal Ulcer**

# Gastric Ulcer on Endoscopy



**Chronic Gastric Ulcers**



# Diagnosis of *H. pylori*

## □ Non-invasive

- $C^{13}$  or  $C^{14}$  Urea Breath Test
- Stool antigen test
- *H. pylori* IgG titer (serology)

## □ Invasive

- Gastric mucosal biopsy
- Rapid Urease test

# Diagnosis of *H. pylori*

## Non-invasive

### 1. C13 or C14 Urea Breath Test

**The best test for the detection  
of an active infection**

# Diagnosis of *H. pylori*

## Non-invasive

- 1) Serology for *H pylori*
  - a. Serum Antibodies (IgG) to *H pylori* (Not for active infection)
  - b. Fecal antigen testing (Test for active HP)

# Diagnosis of *H. pylori*

## Invasive

- Upper GI endoscopy
  - Highly sensitive test
  - Patient needs sedation
  - Has both **diagnostic** & **therapeutic** role



# Diagnosis of *H. pylori*

## Invasive (endoscopy)

- Diagnostic:
  - Detect the site and the size of the ulcer, and superficial ulcer can be detected
  - Detect source of bleeding
  - Biopsies can be taken for rapid urease test, histopathology & culture



# Diagnosis of *H. pylori*

## Invasive (endoscopy)

### ■ Rapid urease test ( RUT)

- Considered the endoscopic diagnostic test of choice
- Gastric biopsy specimens are placed in the rapid urease test kit. If *H. pylori* are present, bacterial urease converts urea to ammonia, which changes pH and produces a **COLOR** change



# Diagnosis of *H. pylori*

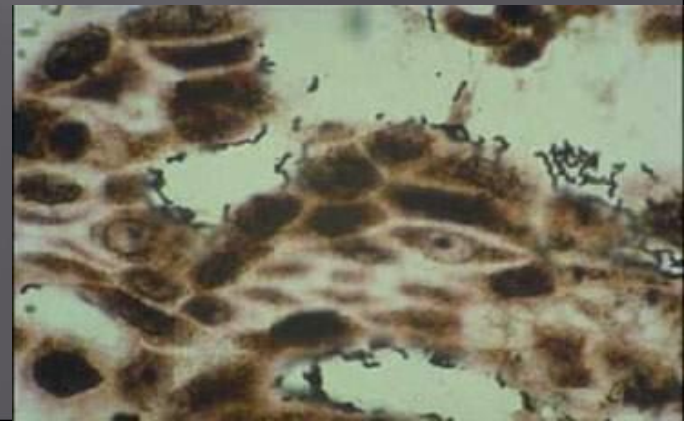
## Invasive (endoscopy)

### \* Histopathology

- Done if the rapid urease test result is negative

### \* Culture

- Used in research studies and is not available routinely for clinical use



# Diagnostic Tests for *Helicobacter pylori*

## Invasive

<u>Test</u>	<u>Sensitivity</u> (%)	<u>Specificity</u> (%)	<u>Usefulness</u>
<b>Endoscopy with biopsy</b>			Diagnostic strategy of choice in children with persistent or severe upper abdominal symptoms
<b>Histology</b>	> 95	100	Sensitivity reduced by PPIs, antibiotics, & bismuth-containing compounds
<b>Urease activity</b>	93 to 97	> 95	Sensitivity reduced by PPIs, antibiotics, bismuth-containing compounds, & active bleeding
<b>Culture</b>	70 to 80	100	Technically demanding



# PUD – Complications

- ▣ Bleeding
- ▣ Perforation
- ▣ Gastric outlet or duodenal obstruction
- ▣ Chronic anemia

# Complications of PUD on Endoscopy



**Bleeding DU**



**Perforated GU**



**Duodenal stricture**

# PUD Treatment

# Treatment Goals

- ▣ Rapid relief of symptoms
- ▣ Healing of ulcer
- ▣ Preventing ulcer recurrences
- ▣ Reducing ulcer-related complications
- ▣ Reduce the morbidity (including the need for endoscopic therapy or surgery)
- ▣ Reduce the mortality

# General Strategy

- ▣ Treat complications aggressively if present
- ▣ Determine the etiology of ulcer
- ▣ Discontinue NSAID use if possible
- ▣ Eradicate *H. pylori* infection if present or strongly suspected, even if other risk factors (e.g., NSAID use) are also present;
- ▣ Use antisecretory therapy to heal the ulcer if *H. pylori* infection is not present

# General Strategy

- ▣ Smoking cessation should be encouraged
- ▣ If DU is diagnosed by endoscopy, RU testing of endoscopically obtained gastric biopsy sample, with or without histologic examination should establish presence or absence of *H. pylori*
- ▣ If DU is diagnosed by x-ray , then a serologic , UBT, or fecal antigen test to diagnose *H. pylori* infection is recommended before treating the patient for *H. pylori*

# Drugs Therapy

- ▣ H<sub>2</sub>-Receptors antagonists
- ▣ Proton pump inhibitors
- ▣ Cyto-protective agents
- ▣ Prostaglandin agonists
- ▣ Antacids
- ▣ Antibiotics for *H. pylori* eradication

