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:**

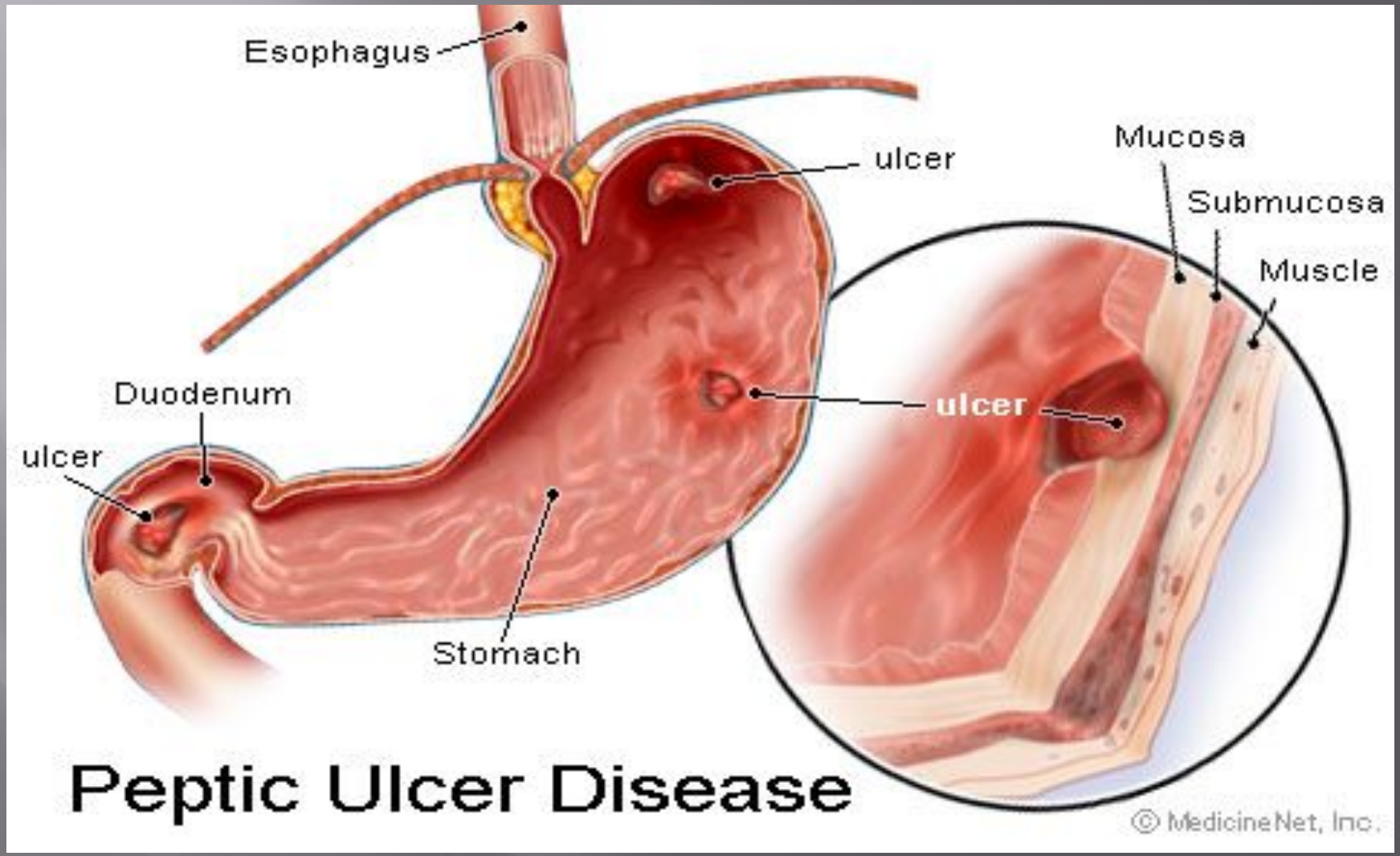
  A lesion on an epithelial surface (skin or mucous membrane) caused by superficial loss of tissue, limited to the mucosa

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) HCO3 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

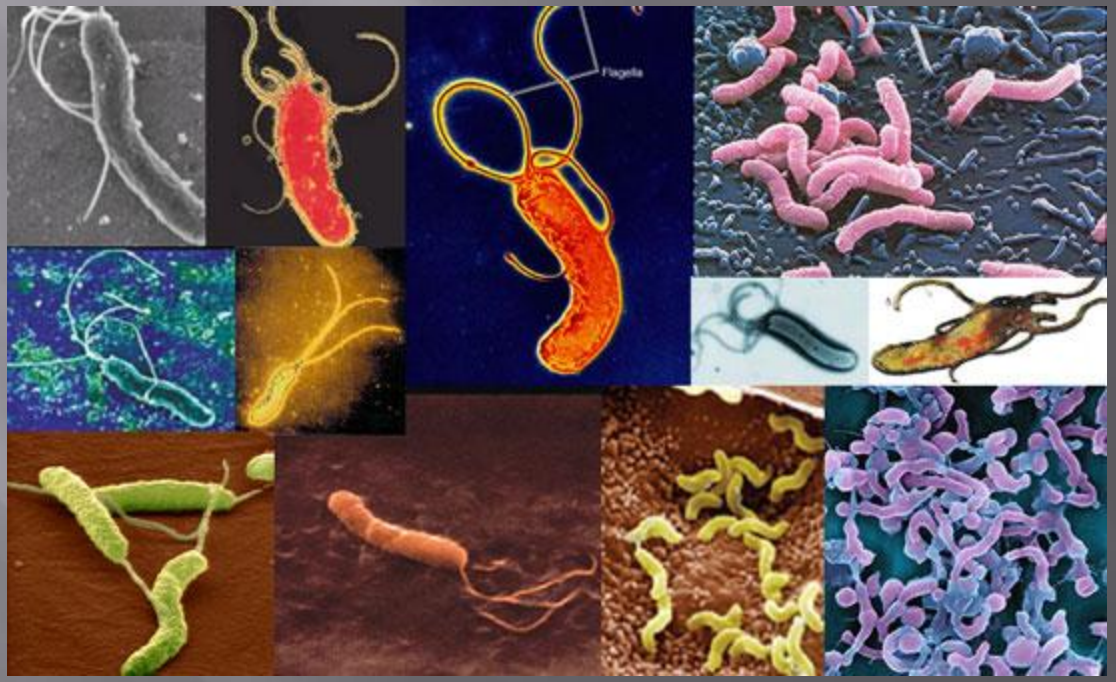
Etiology

- 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*



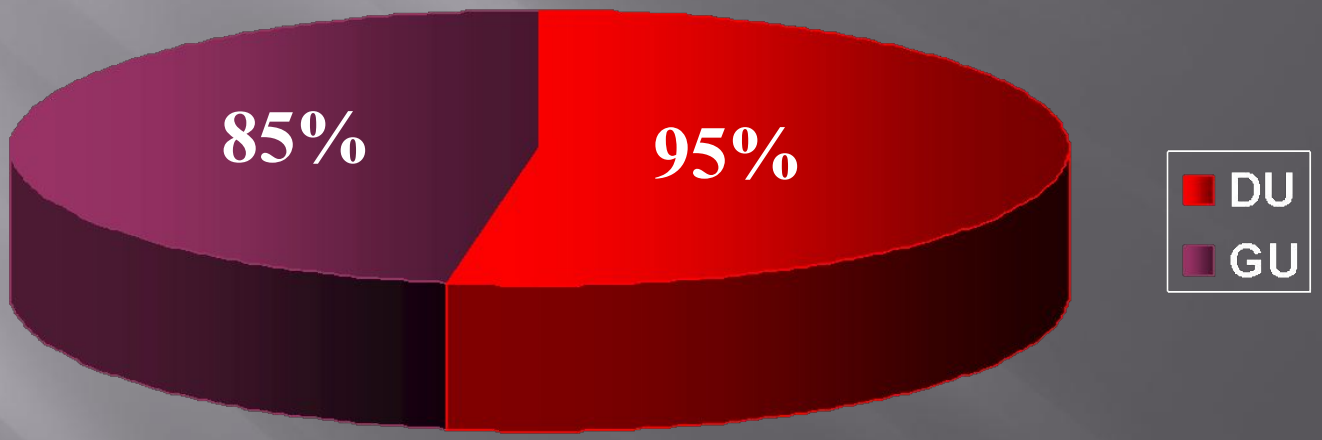
H.pylori Epidemiology

- 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 ≥ 3% in developing countries

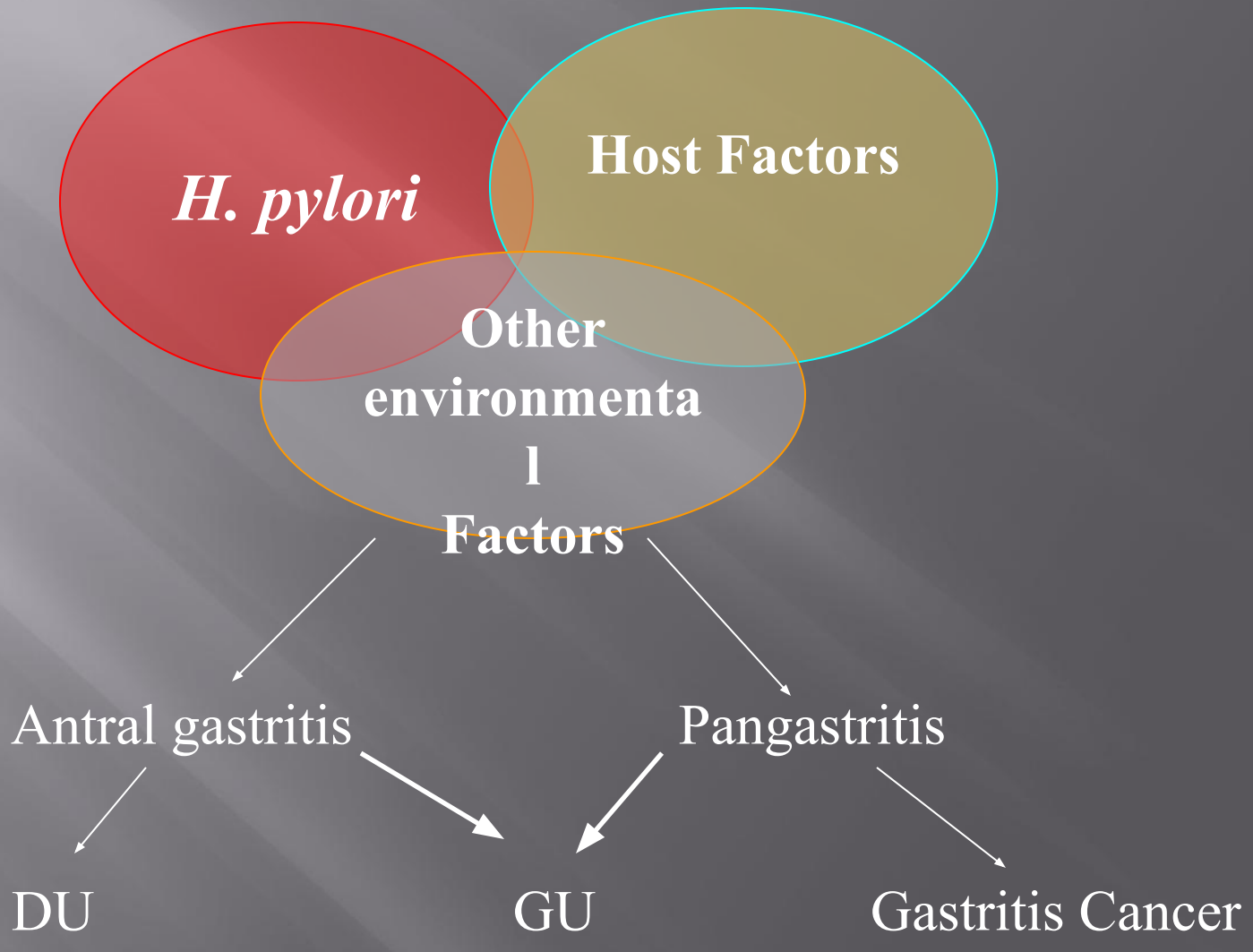
H.pylori as a cause of PUD

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

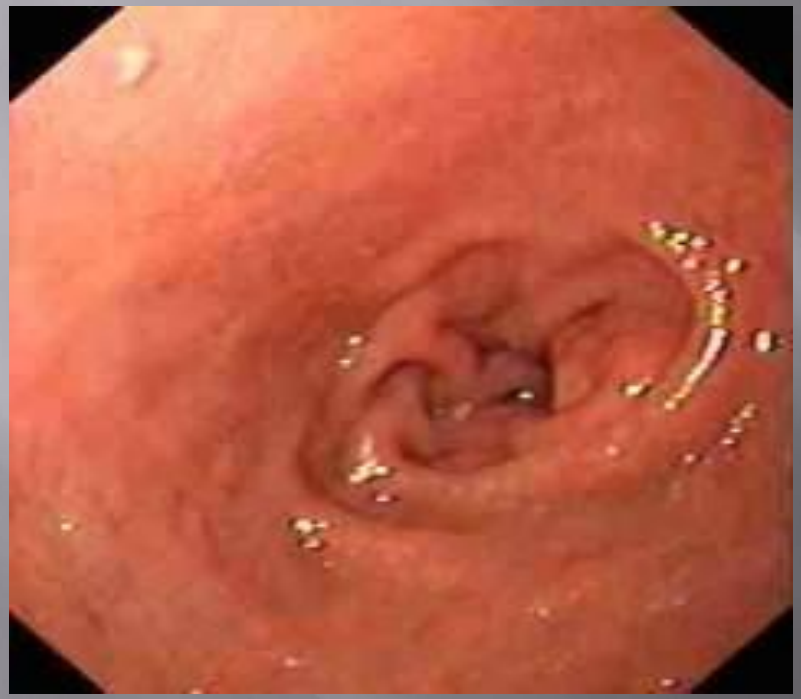
Diagnosis:

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

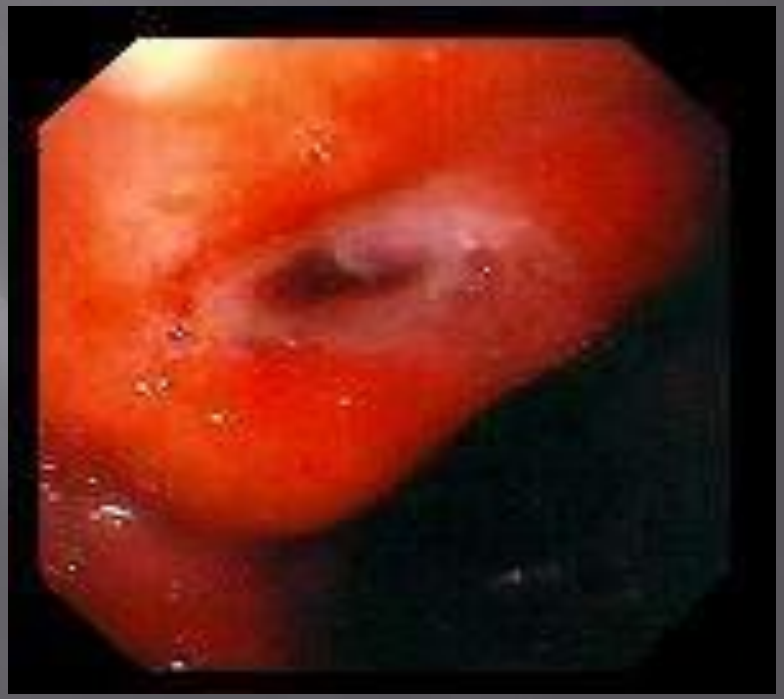
Diagnosis of PUD

Diagnosis of PUD depends mainly on endoscopic and radiographic confirmation

Doudenal Ulcer on Endoscopy

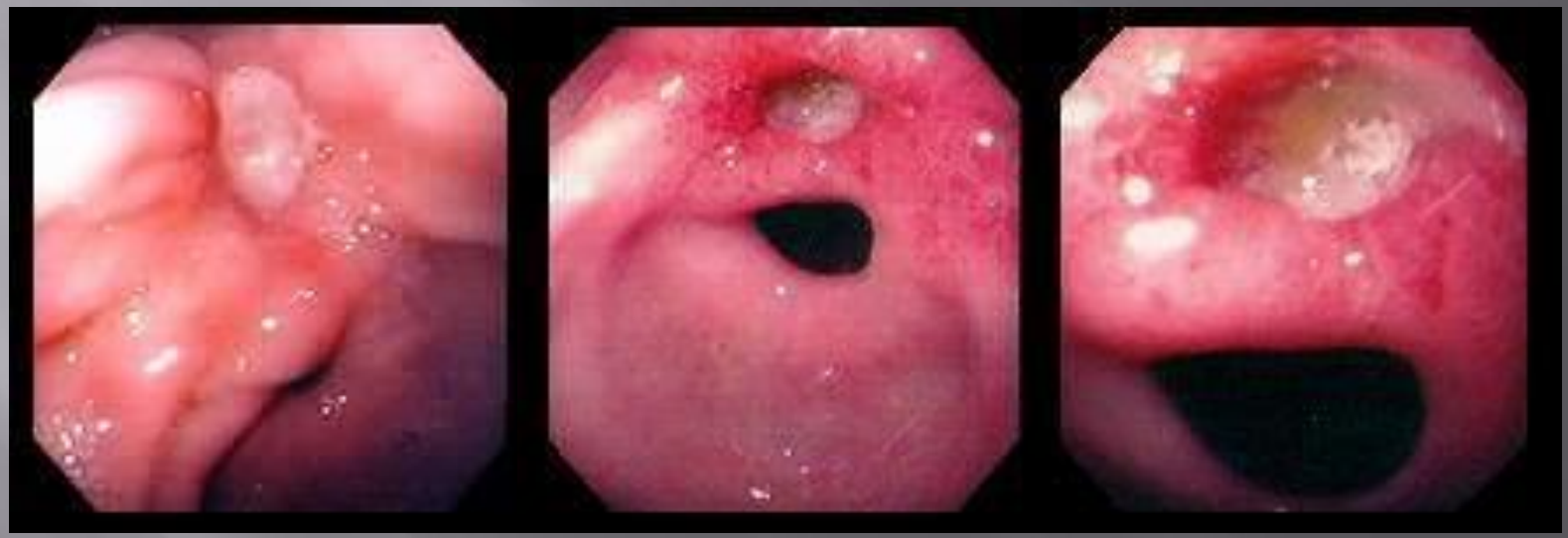


Normal doudenal bulb



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*

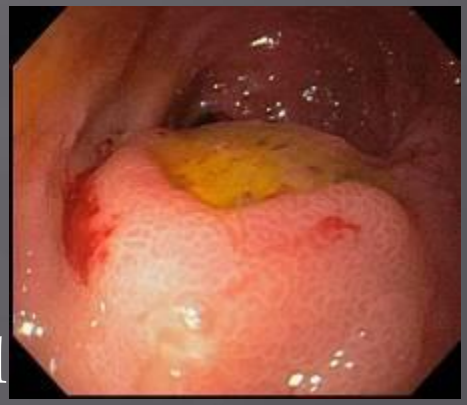
Non-invasive

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

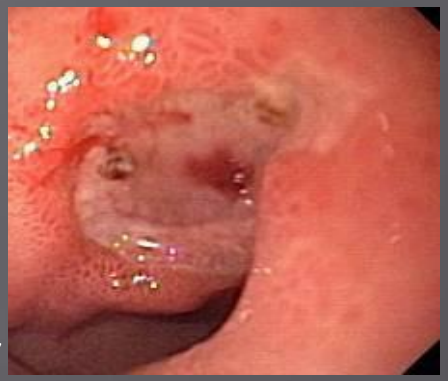
Diagnosis of *H. pylori*

Invasive

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



Diagnosis of *H. pylori*



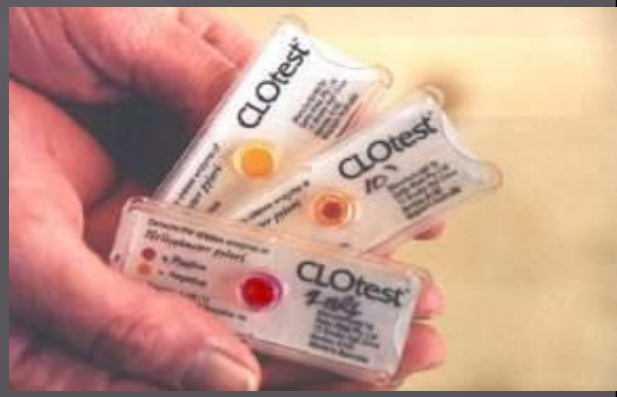
Invasive (endoscopy)

- 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)

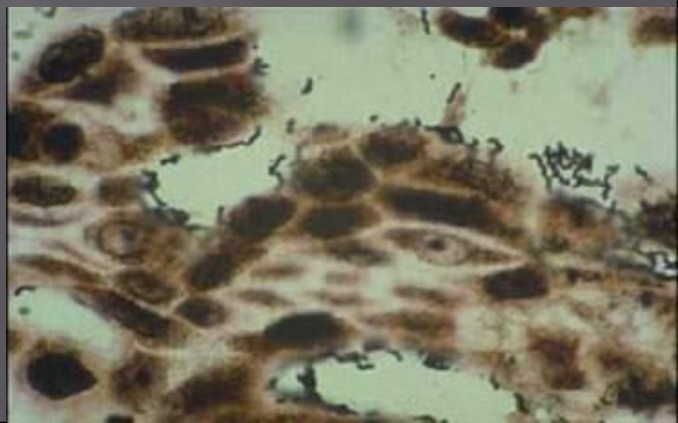
- **Rapid urease test (RUT)**
  - Considered the endoscopic diagnostic test of choice
  - Gastric biopsy specimens are placed in the rapid urease test kit. If *H pylori* are present, bacterial urease converts urea to ammonia, which changes pH and produces a **COLOR** change



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*

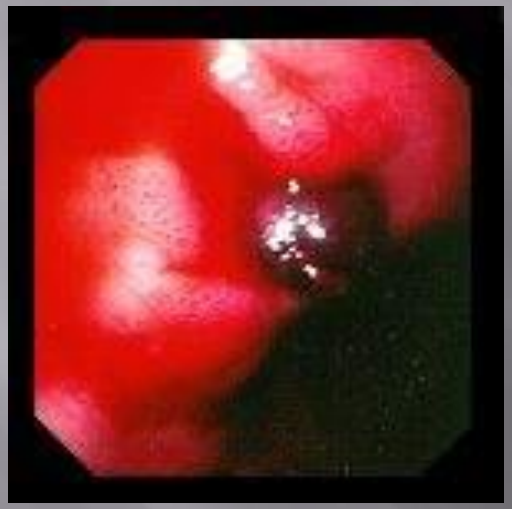
Invasive

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

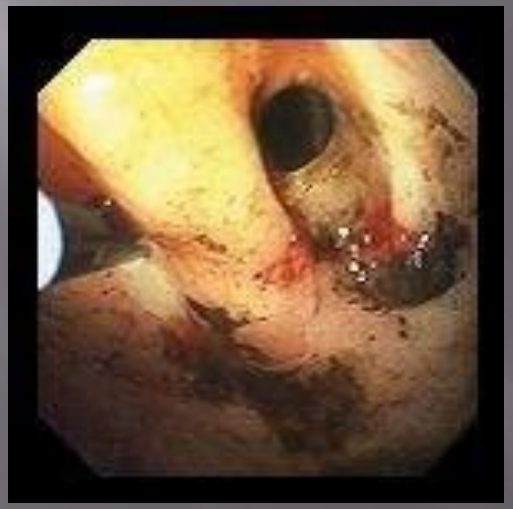
PUD – Complications

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

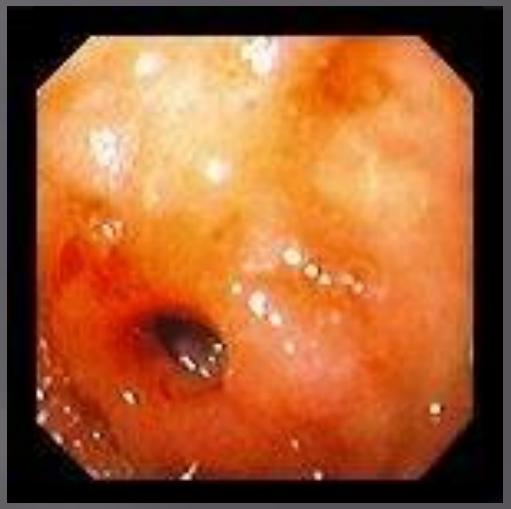
Complications of PUD on Endoscopy



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

- H2-Receptors antagonists
- Proton pump inhibitors
- Cyto-protective agents
- Prostaglandin agonists
- Antacids
- Antibiotics for *H. pylori* eradication

Thank U