Peptic Ulcer Disease: PUD

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Objectives

Upon completion of this lecture the students will :

A] Understand the Pathophysiology of acute and chronic peptic ulcer

B] Know the possible causes of gastric and duodenal ulcers with emphasis on most common causes (H pylori and drugs)

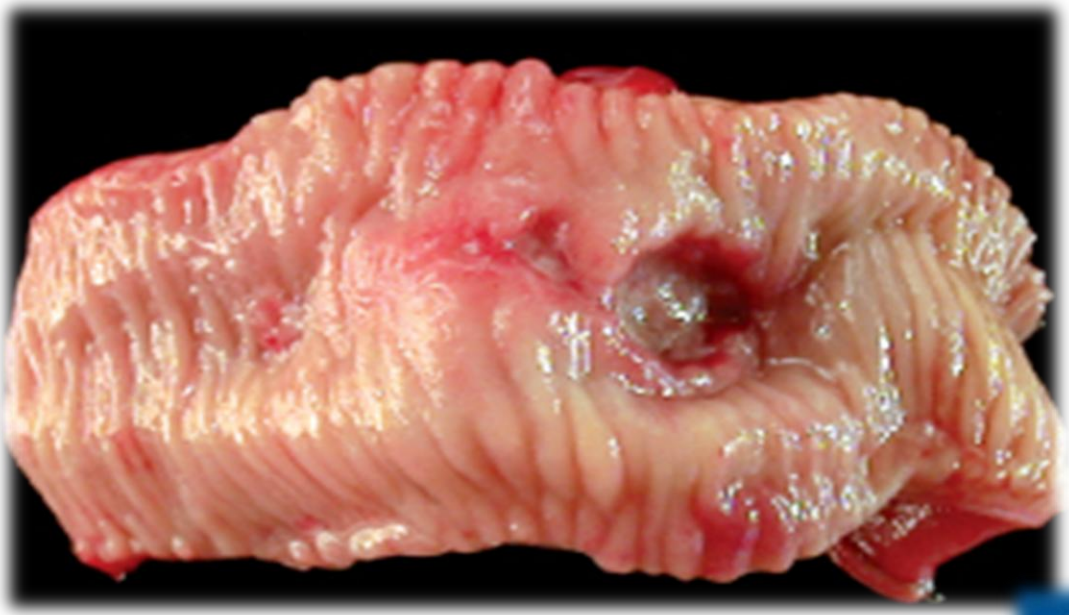
C] Recognize the gross and microscopic features of peptic ulcer (Morphology)

D] Recognize the clinical features and consequences of acute and chronic peptic ulcer (complications)

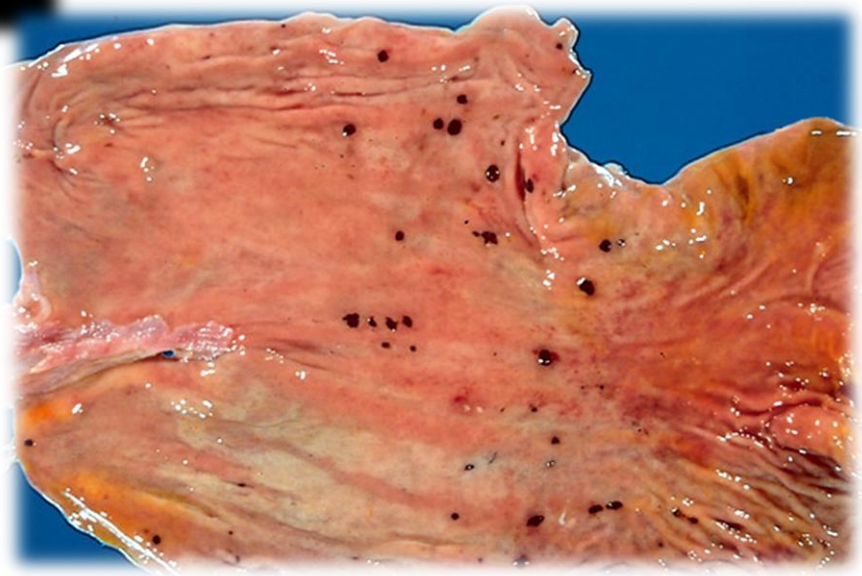
Ulcer

Ulcer: a breach in the mucosa of the alimentary tract extending through muscularis mucosa into submucosa or deeper.

1. *Peptic ulcer*



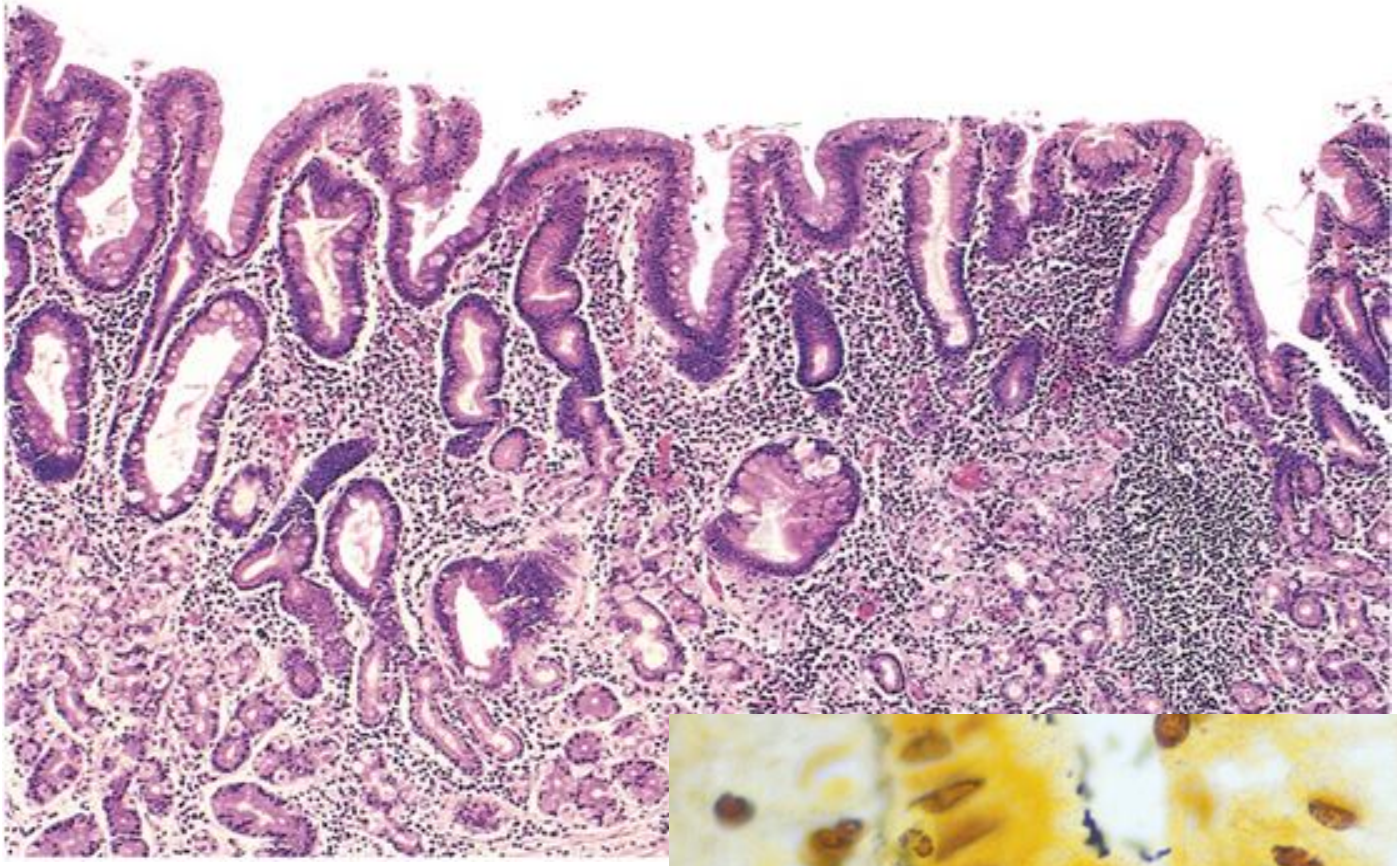
1. *Stress ulcers (acute gastric ulcers)*



Normally, there should be BALANCE

Causes of Peptic Ulcers

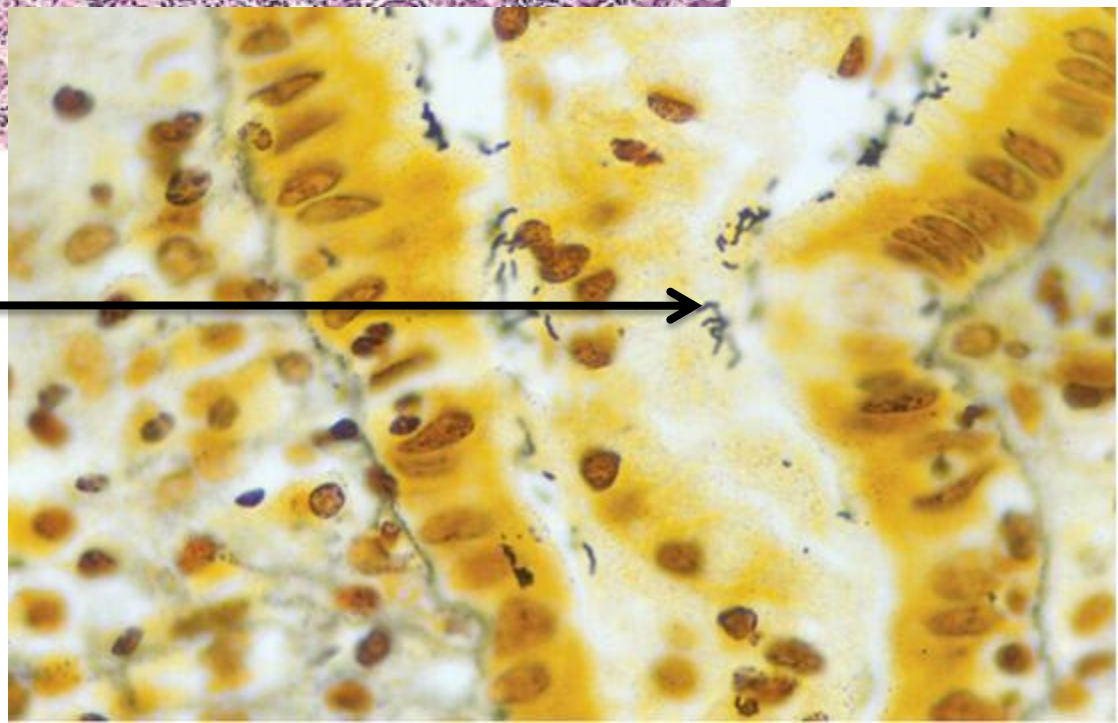
Defensive Factors	Aggressive Factors
	imbalance
Mucus	H. pylori
bicarbonate	Drugs (NSAIDs)
Blood flow	Acid
cell renewal	pepsin
Prostaglandins	Bile salts
Phospholipid	



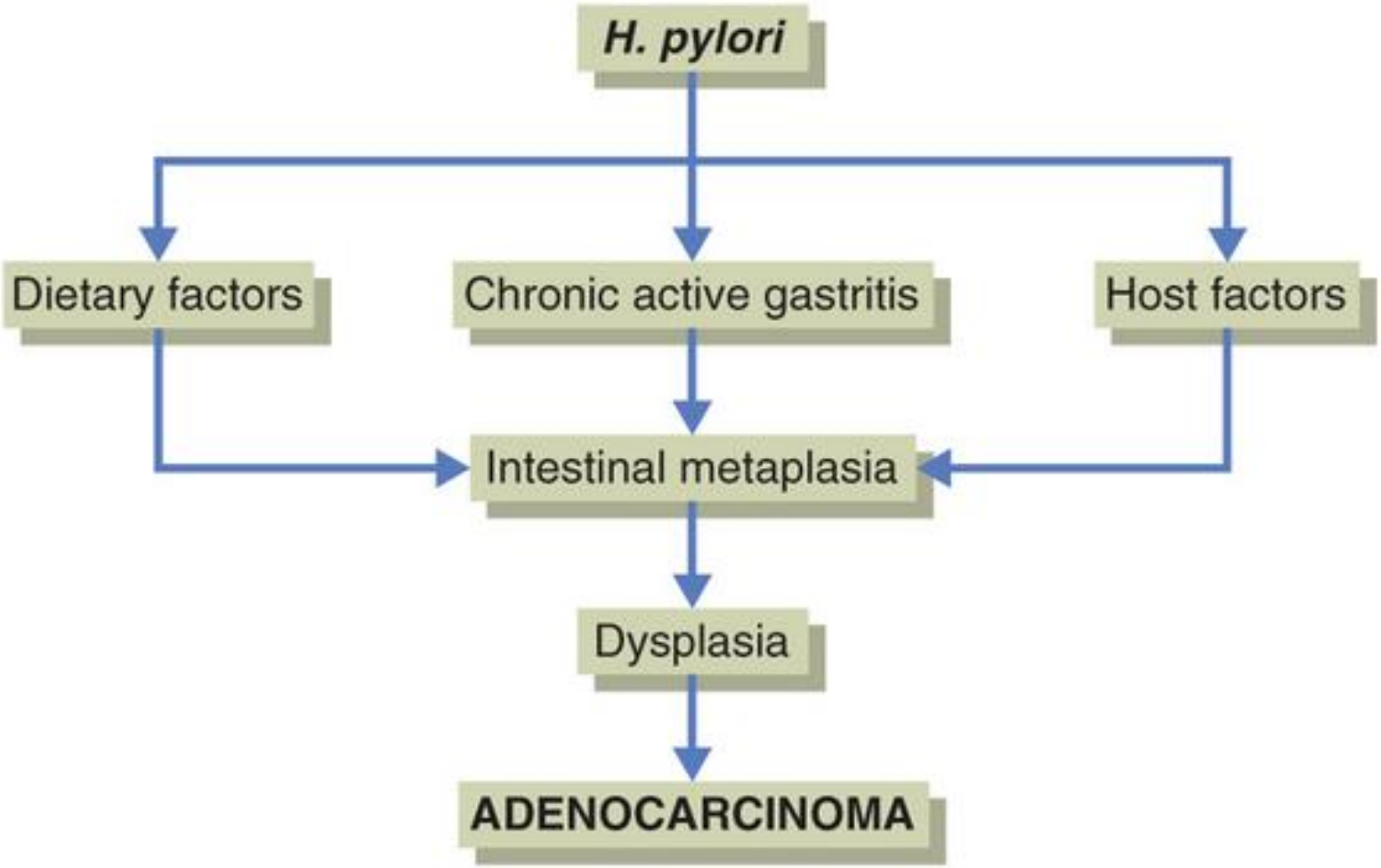
H. pylori causes:

chronic active gastritis

A chronic disease but is always active.



Pathophysiology of Peptic Ulcers



Pathophysiology

1- Acute peptic ulcers

- As part of an acute *gastritis*

  acute response to an irritant 'chemical' injury by drugs or alcohol

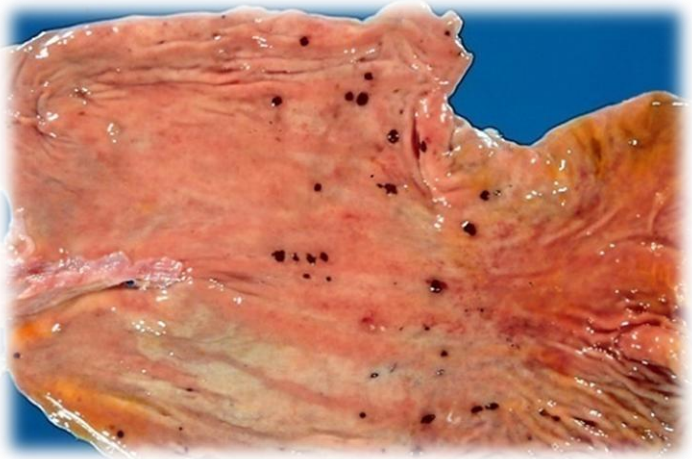
- As a complication of a severe *stress response*

  severe burns (Curling's ulcer)

  major trauma or cerebrovascular accidents(c)

- As a result of extreme *hyperacidity*

  Zollinger-Ellison syndrome.

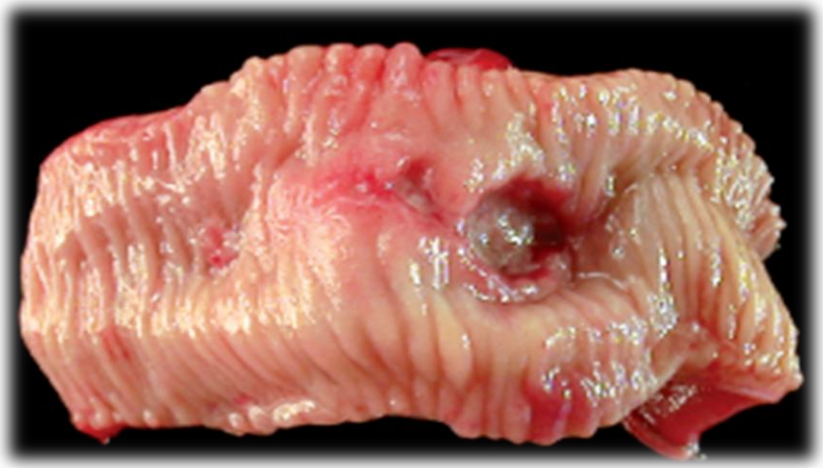


Pathophysiology

2- Chronic peptic ulcers

- **May occur in any portion of the GI tract if exposed to acidic gastric juices**
- **MAINLY (98%) in 1st part of duodenum. THEN in stomach. (4:1)**
- **THIRD LOCATION: Esophagus - as a result of GERD or acid secretion by ectopic gastric mucosa.**

Mickel's Diverticulum:
a small bulge in the small intestine present at birth (congenital malformation).
Gastric mucosa within it, can result in "Peptic Ulcerations" of adjacent mucosa.



Pathophysiology

Peptic Ulcer Disease

1) Gastric ulcers

The mucosal defenses against acid attack consist of:

1. Mucus-bicarbonate barrier

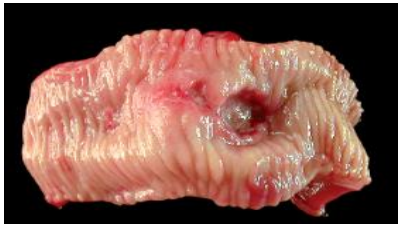
⇨ Duodeno-gastric reflux (bile reflex)

2. The surface epithelium.

⇨
1. NSAIDs (blocking the synthesis of the prostaglandins)
2. *H. pylori* infection, (cytotoxins and ammonia)

Thus, in **Gastric** ulcers, Factors causing breakdown of mucosal defenses, are of **more damage** than "excessive acid production" .

Peptic Ulcer Disease



2) Duodenal ulcers

In **Duodenal Ulcers**, Increased production of acid is of **most damage** here.

H. pylori-infected individuals:
secrete 2-6 times much acid than normal

Helicobacter Pylori does not colonize normal duodenal epithelium
BUT:
It is involved in duodenal ulceration because there is **gastric metaplasia** (in response to excess acid). Gastric metaplasia facilitates the way for colonization by Helicobacter

Increased production of acid + *H. Pylori* = Duodenal ulcers

Terminology

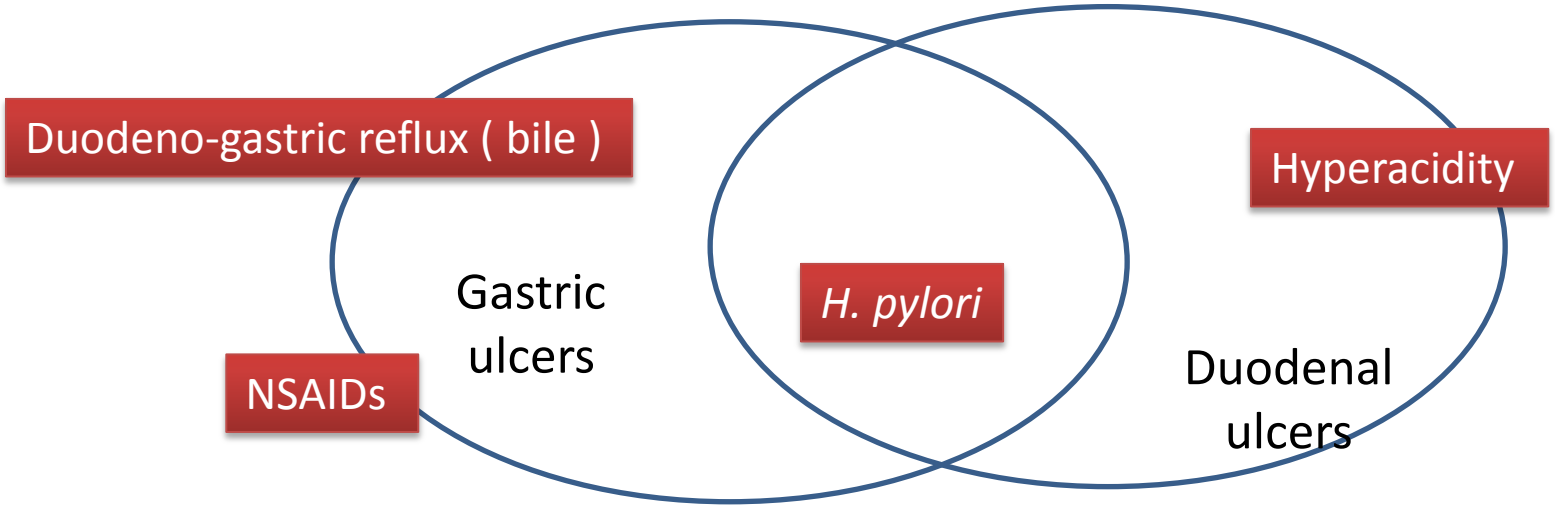
- Metaplasia: Transformation of cells from a normal to an abnormal state

Cell type is Changed

- Dysplasia: abnormality of development

The same type of cell

Statistics and Relations

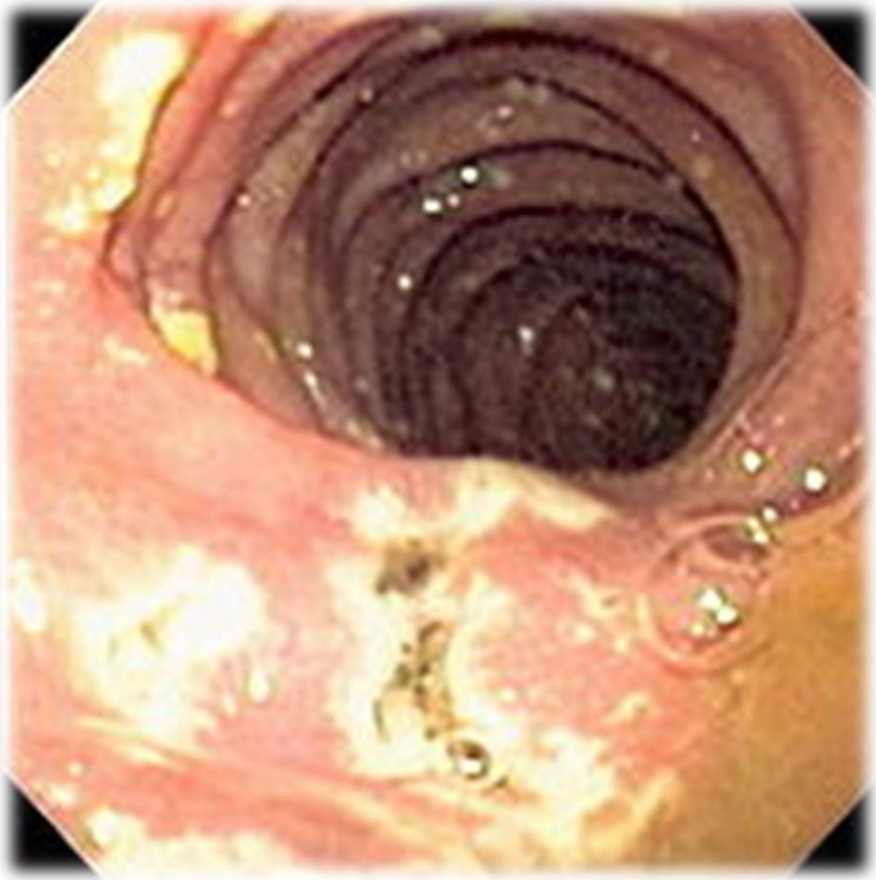


H pylori infection of the "pyloric antrum" →
is present in nearly all patients with **chronic duodenal ulcer** and approximately 75% of patients with **chronic gastric ulcer.**

20% of patients with H.Pylori will develop **Peptic Ulcer**
While 70% of patients with **Peptic Ulcer**, commonly have H.pylori infection.

Zollinger-Ellison syndrome

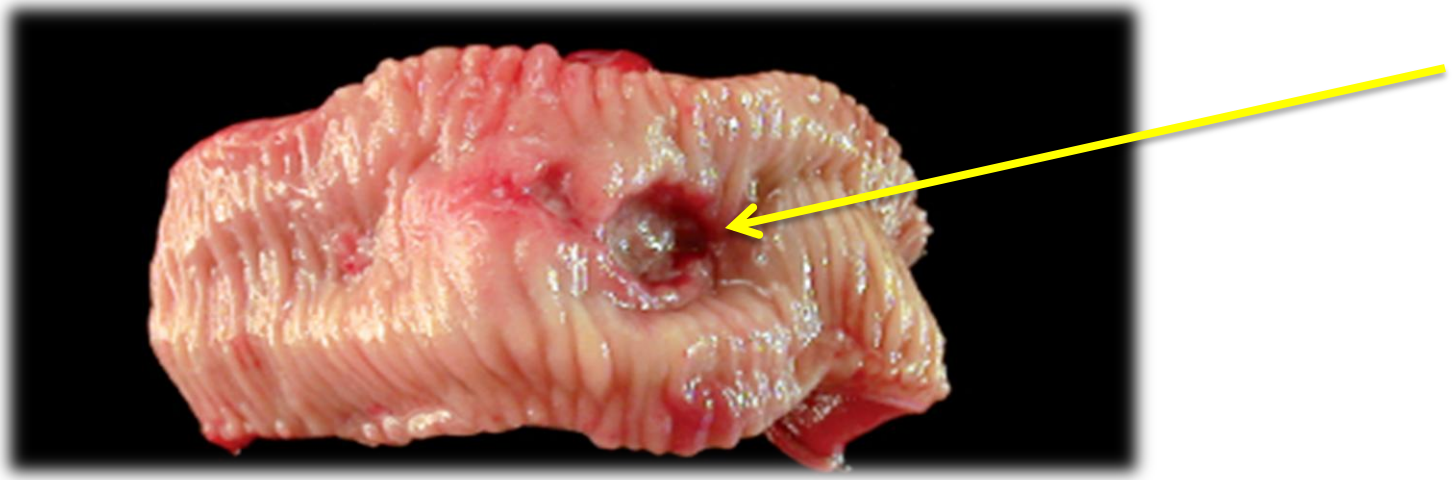
Gastrin-secreting tumor of the pancreas or small intestine

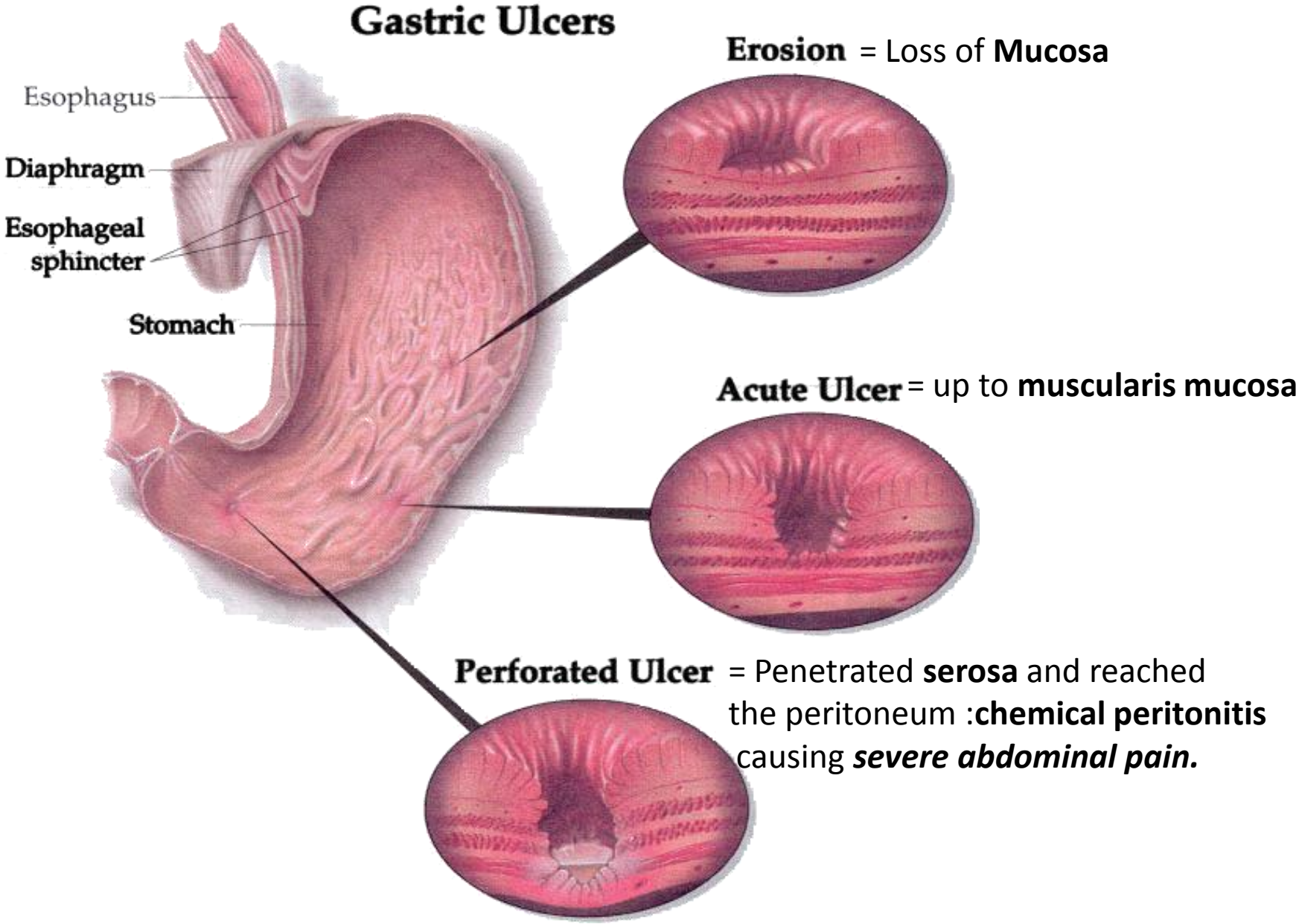


Morphology of Peptic Ulcers

Macroscopic Features - GROSS

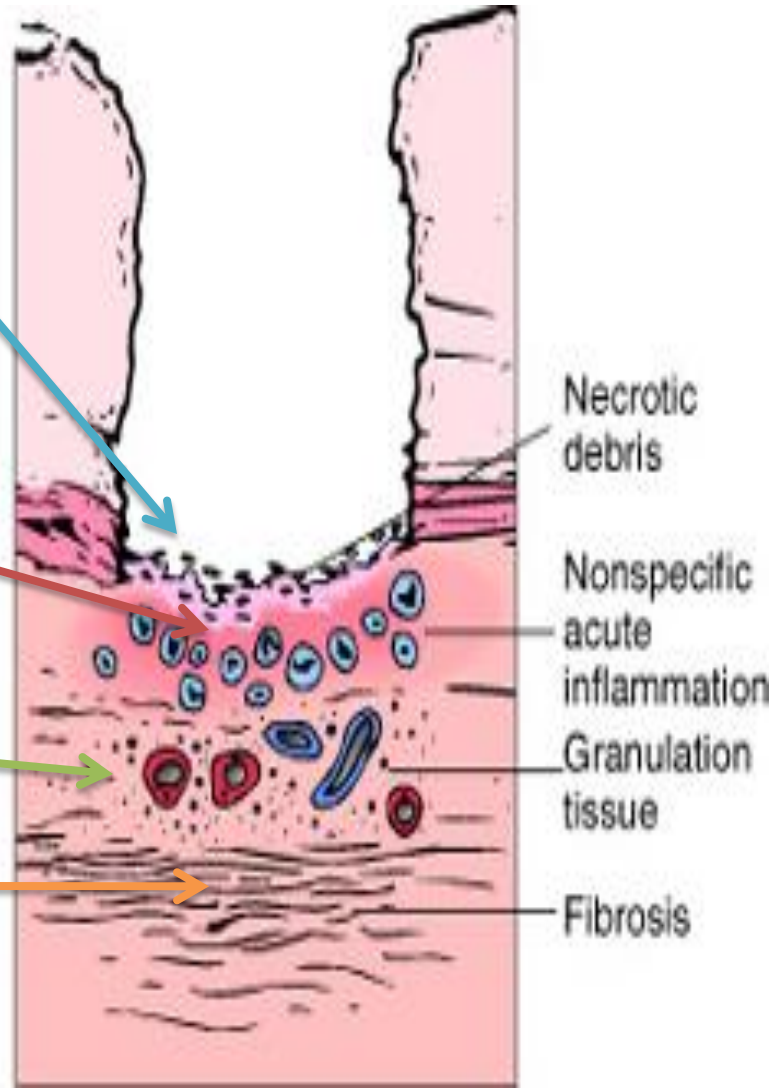
- usually less than 20 mm in diameter but they may be bigger than 100 mm in diameter.
- Sharply Punched out defect
- Round to oval in shape.
- Solitary (usually one in number).
  In acute-ulcers due to stress, they are many in number
- Its edges are smooth and low.
  In cancers they're heaped up.



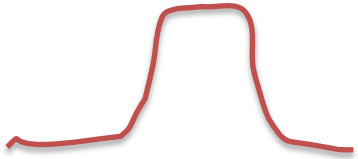

Gastric Ulcers
Esophagus
Diaphragm
Esophageal sphincter
Stomach
Erosion = Loss of Mucosa
Acute Ulcer = up to muscularis mucosa
Perforated Ulcer = Penetrated serosa and reached the peritoneum :chemical peritonitis causing severe abdominal pain.

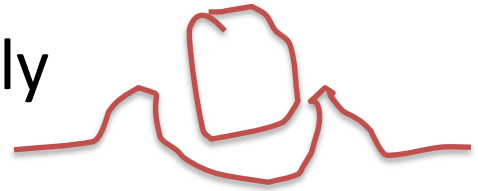
Macroscopic Features

- At base: **Necrotic tissue**
- Beneath base: **zone of inflammation + pleomorph exudate.**
- Beneath inflammation: **Granulation tissue and fibroblasts** (tissue trying to heal)
- Beneath granulation: **Mature Fibrous tissue (Scar)** protects from perforation

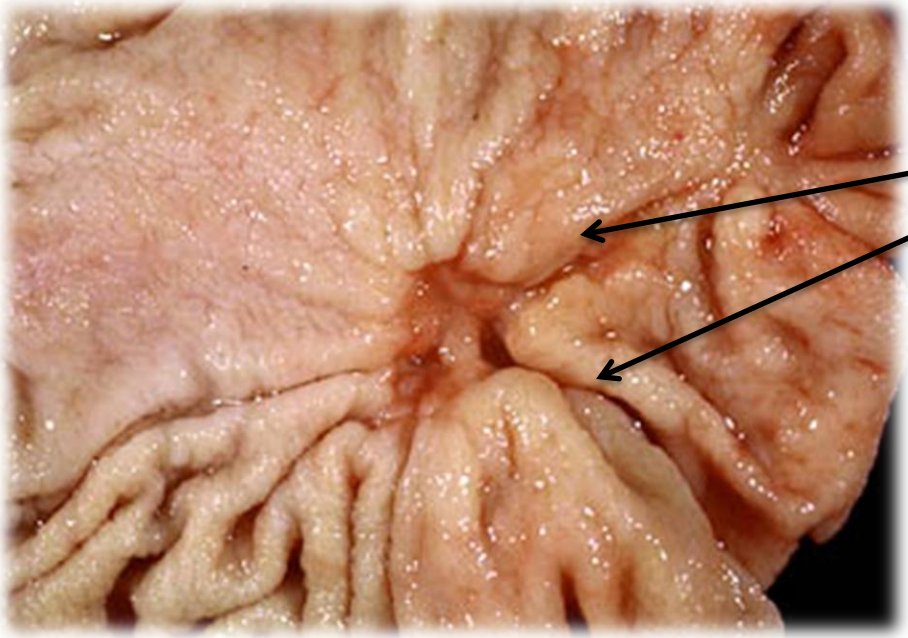


To differentiate from Malignant Ulcer



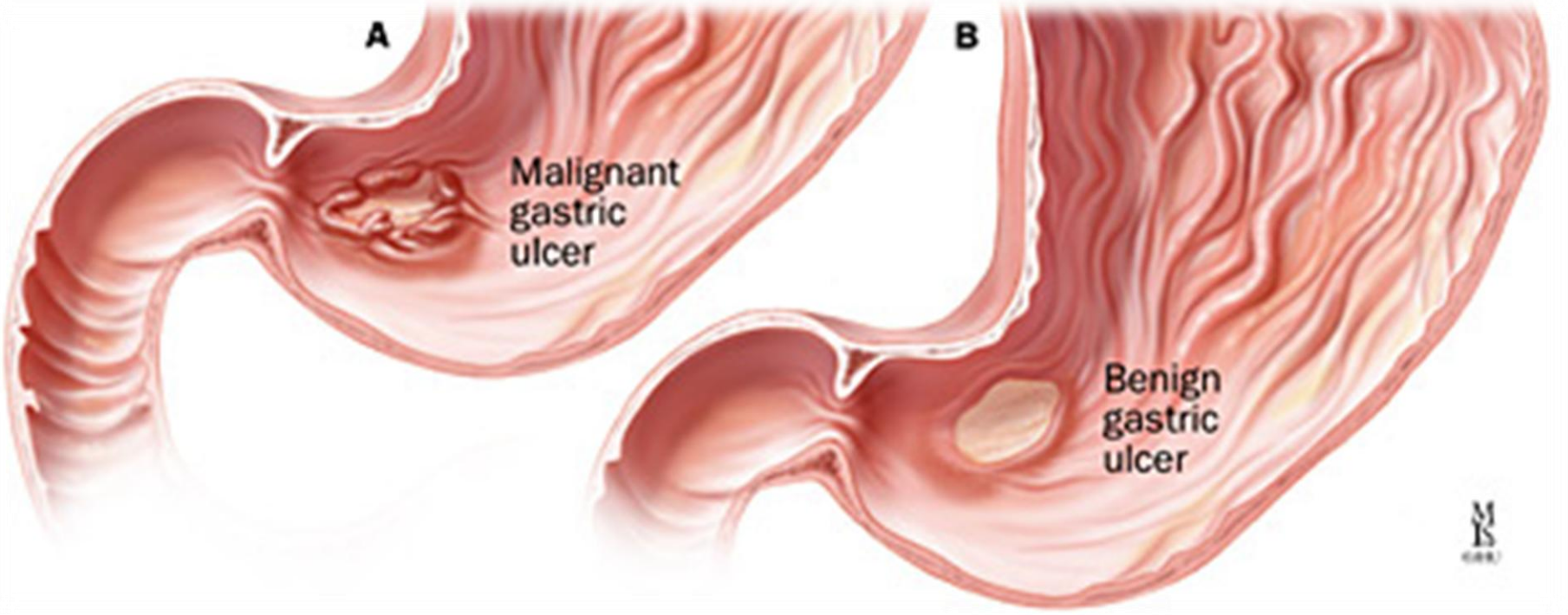


1. A tumor forms
2. Gets necroted due to low blood supply
3. Gets stuffed away (falls out)
4. It has **raised and inverted edges,** just like:



See how gastric rugae (folds) are **radiating** from the ulcer.
(***only in malignant*** ones)

Cancer is a complication of ulcers

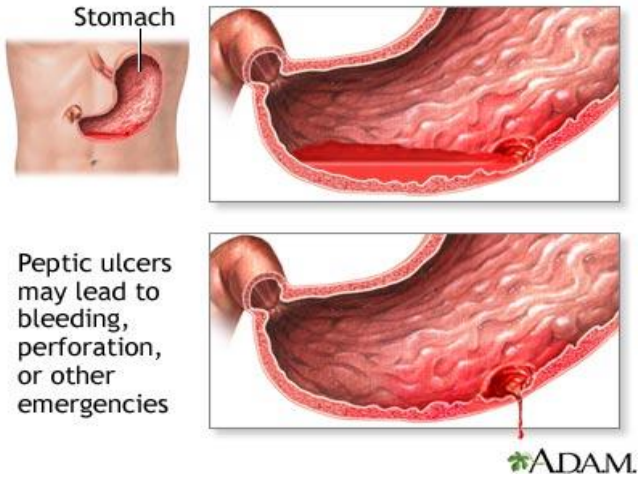


Clinical features

- Epigastric pain (the most common symptom)
  - Gnawing or burning sensation
  - Occurs 2-3 hours after meals
  - Relieved by food or anti-acids (antacids)
  - Patient awakens with pain <u>at night</u> when stomach is empty.

Some present with complications such as *iron deficiency anemia*, *frank hemorrhage*, or *perforation*.

Complications



- **Hemorrhage**.
- **Penetration**: The ulcer penetrates the full thickness of the stomach or duodenal wall, progressing into adherent underlying tissue, particularly the pancreas or liver. Penetration of the pancreas often manifests clinically as severe back pain.
- **Perforation** : This leads to peritonitis.
- **Fibrous stricture** : This is seen in peptic ulcer of the esophagus and the stomach (pyloric stenosis)
- **Malignant change** : This is extremely uncommon.

Therapy

Current therapies for PUD are aimed at

1. ***H. pylori* eradication**

2. Acid suppression :

a) Proton pump inhibitors (PPIs)
b) H2 blockers