Lecture :2

Peptic Ulcer Disease

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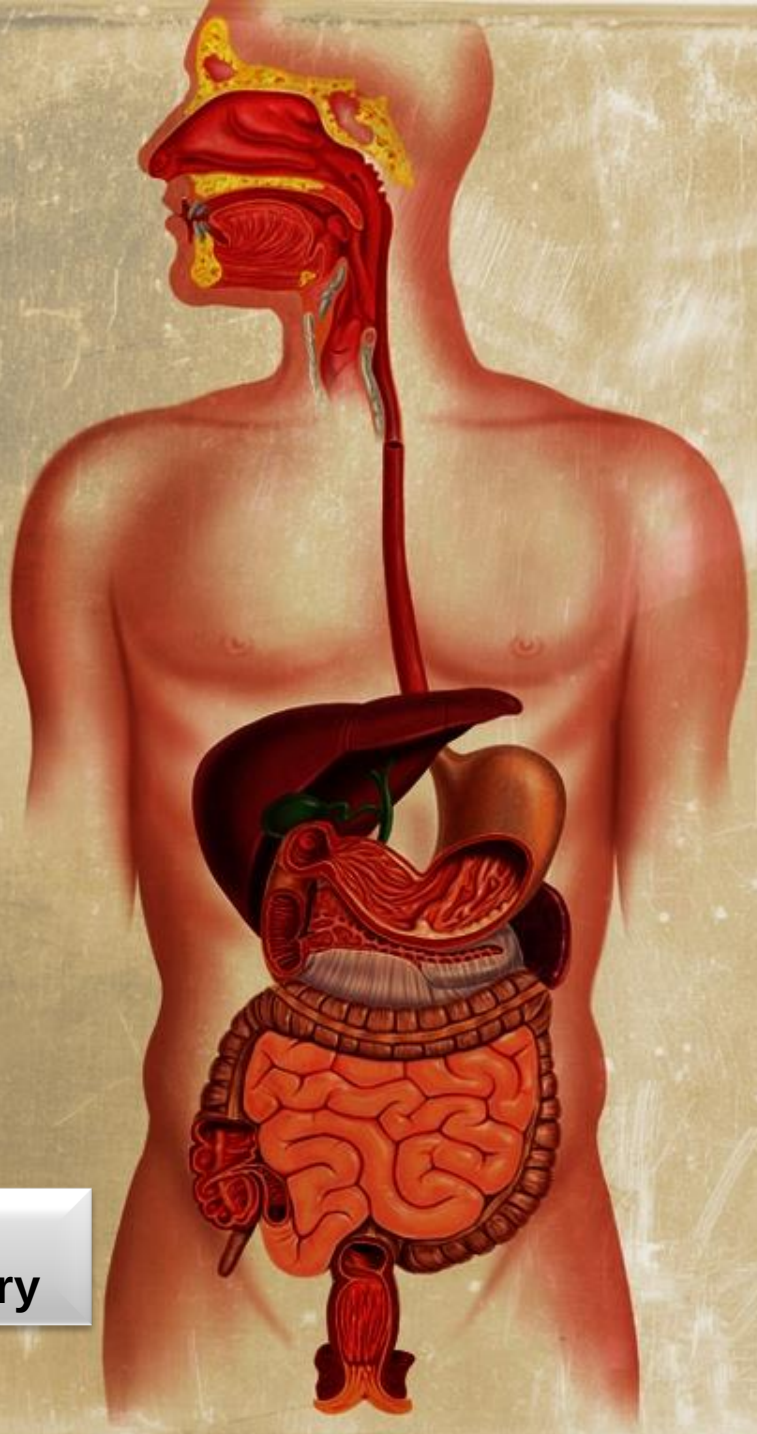

Objectives

Here is some videos to help you understand:

-1Peptic Ulcer (05:43) >> **Highly recommended**

-2Peptic Ulcer Disease Explained ((13:38

-3Peptic Ulcer Disease Pathophysiology ((29:53

Upon completion of this lecture the students will :

1. Understand the Pathophysiology of acute and chronic peptic ulcer
2. Know the possible causes of gastric and duodenal ulcers with emphasis on most common causes (H pylori and drugs)
3. Recognize the gross and microscopic features of peptic ulcer
4. Recognize the clinical features and consequences of acute and chronic peptic ulcer

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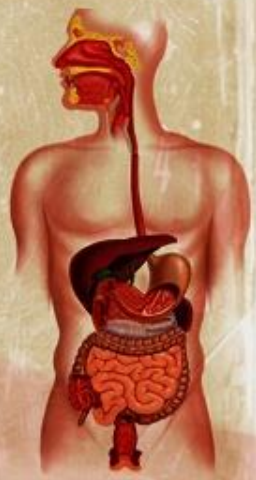


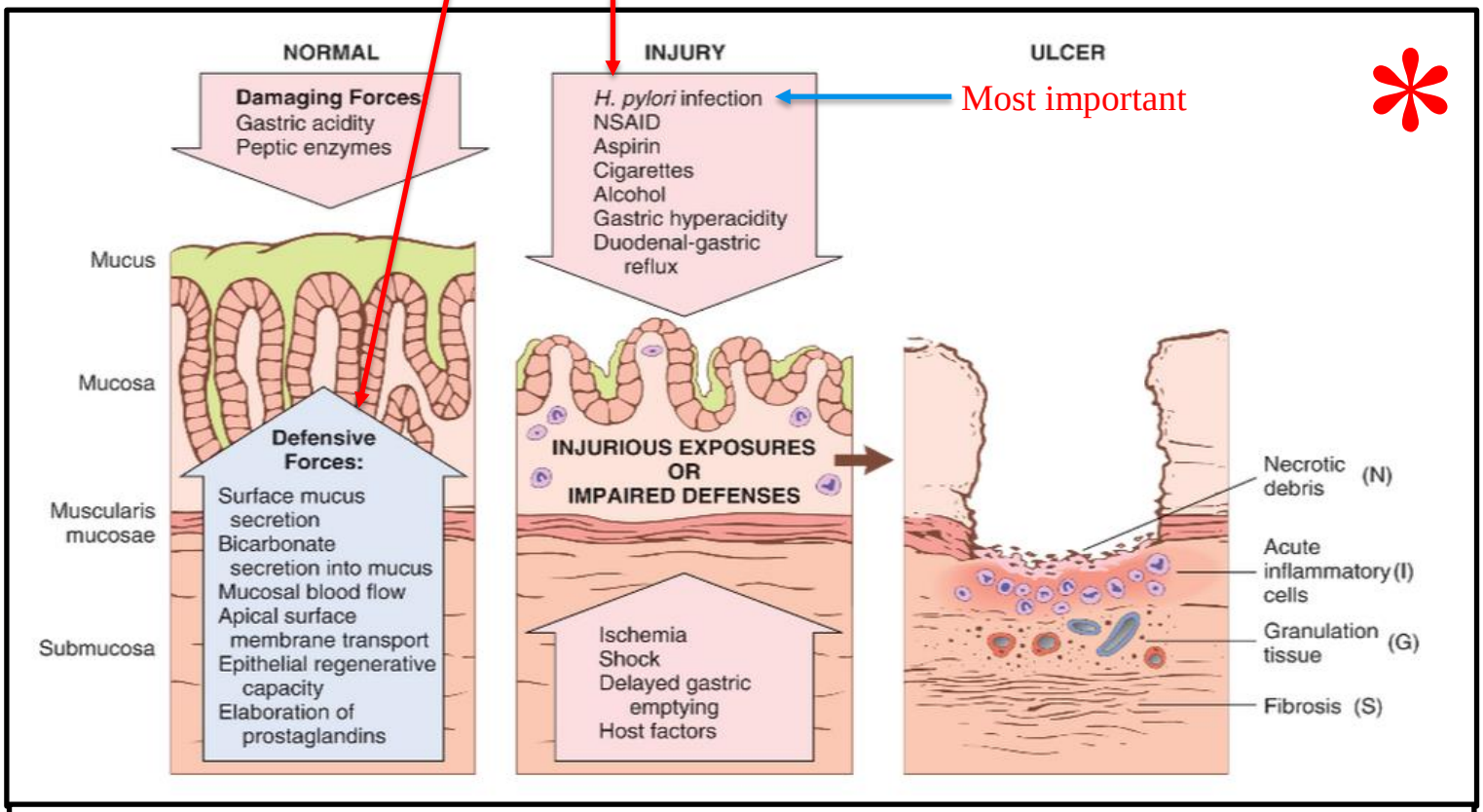
Ulcer

Definition of ulcer:

A breach (breakdown) in the mucosa of the alimentary tract extending through muscularis mucosa into submucosa or deeper

Pathophysiology of ulcer:

Imbalance between defensive and aggressive factors



Mechanisms of gastric injury and protection. This diagram illustrates the progression from more mild forms of injury to ulceration that may occur with acute or chronic gastritis. Ulcers include layers of necrotic debris *(N)*,inflammation *(I)*, and granulation tissue*(G)*; a fibrotic scar *(S)*, which develops over time, is present only in chronic lesions.

1.Acute gastric Ulcers.

Pathophysiology

1- As part of an acute gastritis :

Acute response to an irritant 'chemical' injury by drugs e.g. NSAID* or alcohol.

NSAID-induced ulcers are caused by direct chemical irritation as well as cyclooxygenase inhibition, which prevents prostaglandin synthesis (which has protective function).

2- As a complication of a severe stress response :

- Severe burns (Curling's ulcer):

  loss of high amount of fluids > Hypovolemia > decreased blood flow to the stomach (hypo-perfusion) > decrease protective mechanism ulceration.

- Major trauma (Stress ulcer)

  bleeding > hypotension > hypo-perfusion > decrease protective mechanism > ulceration.

- Cerebrovascular accidents (Cushing ulcer) :

  increase in intracranial pressure which causes direct stimulation of vagal nuclei, which causes gastric acid hyper-secretion, which lead to the formation ulcer.

3- As a result of extreme hyperacidity:

Zollinger-Ellison syndrome (Multiple peptic ulcerations in the stomach, duodenum, and even jejunum, is caused by uncontrolled release of gastrin by a tumor and the resulting massive acid production).

Prognosis:

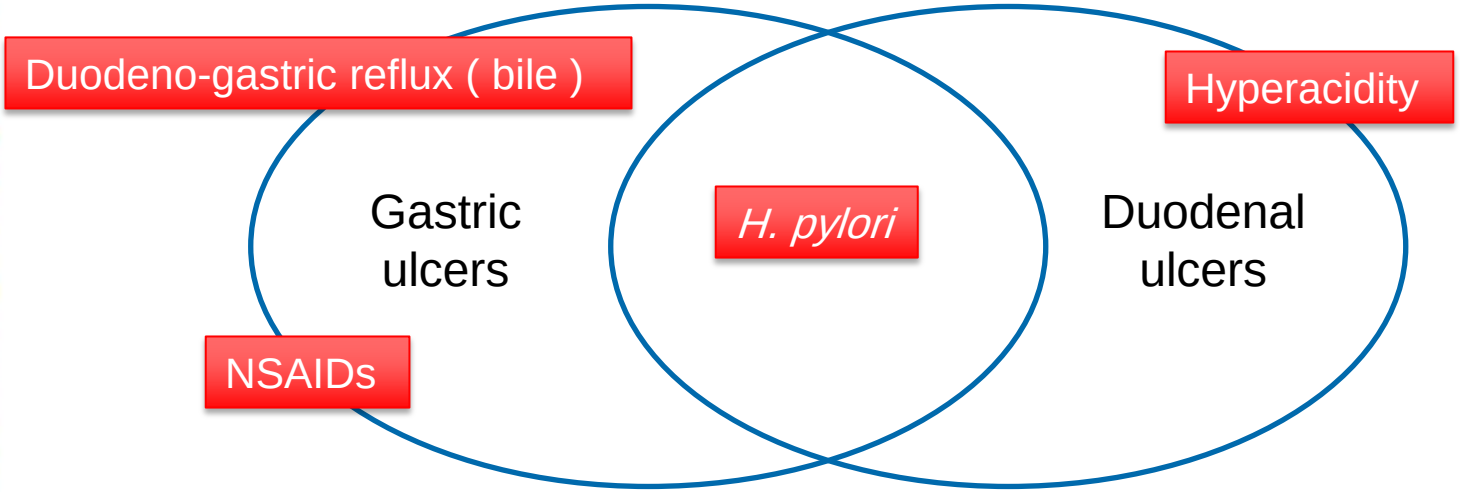
The gastric mucosa can recover completely if the person does not die from the primary disease

*Most common cause of acute gastric ulcer

2.Chronic Peptic Ulcer

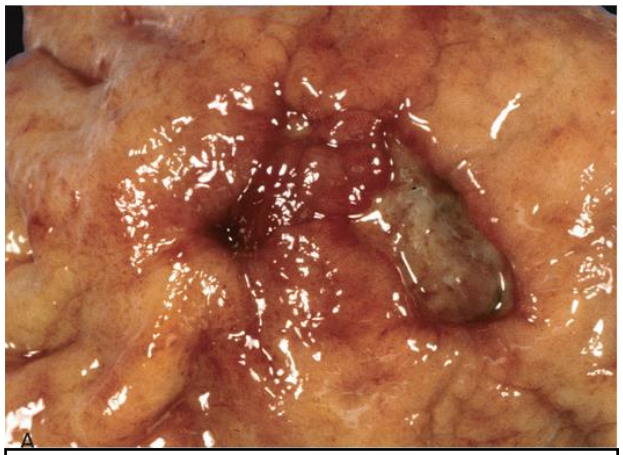
- ✓ Usually call it Peptic ulcer disease
- ✓ May occur in any portion of the GI tract exposed to acidic gastric juices
- ✓ Usually Occur in duodenum or stomach, Ratio = 4:1
- ✓ Usually caused by H.pylori infection
- ✓ Gastric ulcer, due to breakdown of mucosal defense rather than excessive acid production.
- ✓ May occur in Esophagus as a result of GERD or acid secretion by ectopic gastric mucosa.
- ✓ May occur in small intestine secondary to Meckel's diverticulum (Embryo lecture)

Causes:

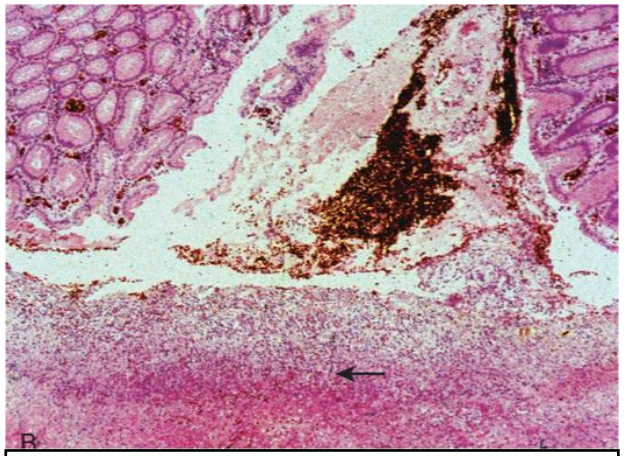


N.B: H pylori infection of the pyloric antrum is present in nearly all patients with chronic duodenal ulcer and approximately 75% of patients with chronic gastric ulcer.

Morphology:



Mucosal defect with clean edges.



The necrotic ulcer base *(arrow)* is composed of granulation tissue

Clinical Features:

Epigastric Pain (the most common symptom):

- Gnawing (burning sensation).

Occurs 2-3 hours after meals or before meal (duodenal ulcer).

And if it occurs during meal or after 10 Minute (gastric ulcer)

- Relieved by food or antacids
- Patient awakens with pain at night.
- Nausea, vomiting, bloating انتفاخ, and belching تجشؤ may be present.

*Some patients present with complications such as **iron deficiency anaemia, frank haemorrhage (most common)**, or **perforation ثقب lead to peritonitis.**

بعض المرضى لا يأتي للعيادة بأعراض المرض و إنما بمضاعفات المرض

Summary from Robbins

SUMMARY

Acute and Chronic Gastritis

- The spectrum of *acute gastritis* ranges from asymptomatic disease to mild epigastric pain, nausea, and vomiting. Causative factors include any agent or disease that interferes with gastric mucosal protection. Acute gastritis can progress to *acute gastric ulceration*.
- The most common cause of chronic gastritis is *H. pylori infection*; most remaining cases are caused by *autoimmune gastritis*.
- *H. pylori* gastritis typically affects the antrum and is associated with increased gastric acid production. The induced mucosa-associated lymphoid tissue (MALT) can transform into lymphoma.
- *Autoimmune gastritis* causes atrophy of the gastric body oxyntic glands, which results in decreased gastric acid production, antral G cell hyperplasia, achlorhydria, and vitamin B_{12} deficiency. Anti-parietal cell and anti–intrinsic factor antibodies typically are present.
- *Intestinal metaplasia* develops in both forms of chronic gastritis and is a risk factor for development of gastric adenocarcinoma.
- Peptic ulcer disease can be caused by *H. pylori* chronic gastritis and the resultant hyperchlorhydria or NSAID use. Ulcers can develop in the stomach or duodenum and usually heal after suppression of gastric acid production and, if present, eradication of the *H. pylori*.