8 LECTURES

- Gastro-esophageal reflux disease
- Peptic Ulcer Disease
- Diarrhea
- Malabsorption
- Inflammatory bowel disease-1
- Inflammatory bowel disease-2
- Colonic polyps and carcinoma-1
- Colonic polyps and carcinoma-2

8 LECTURES

Gastro-esophageal reflux disease

Peptic Ulcer Disease

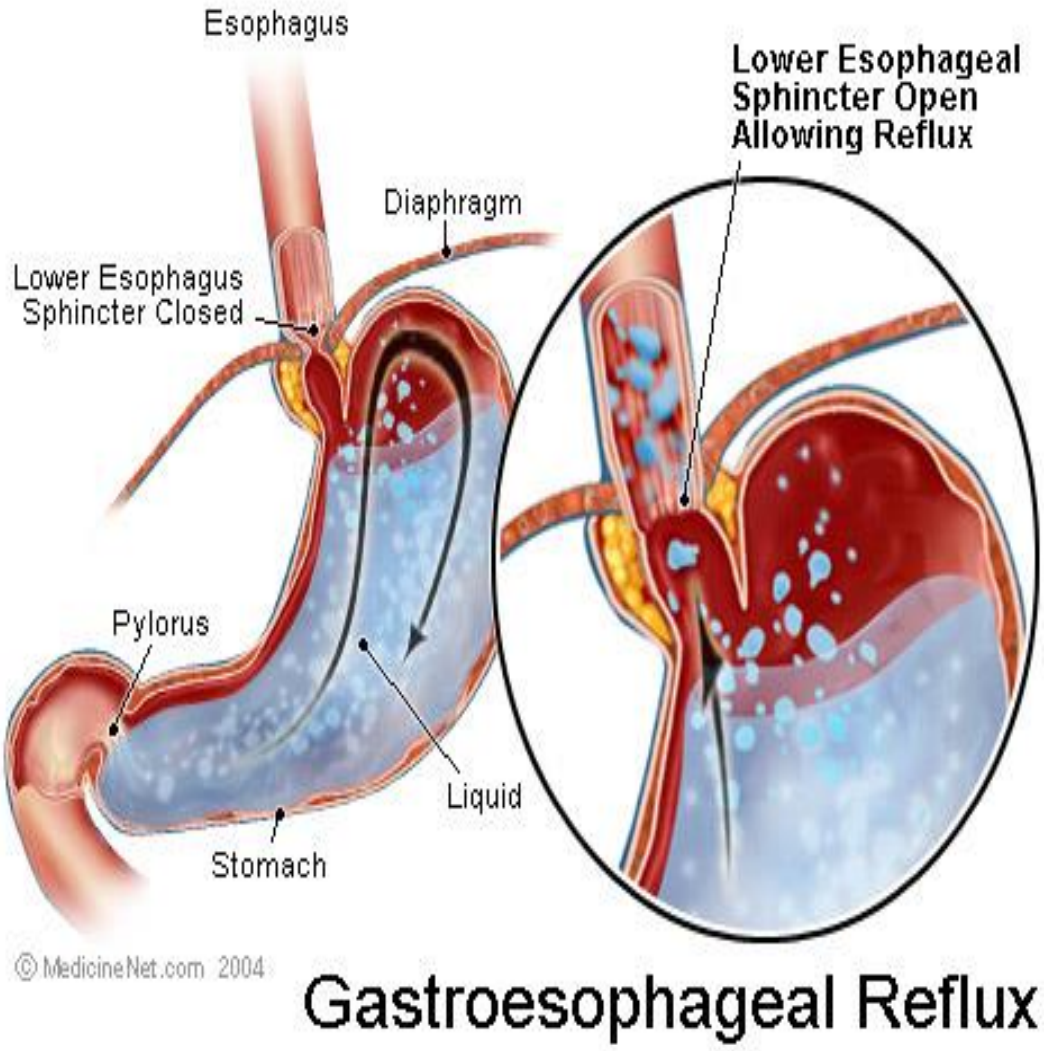
Revision

Objectives

Upon completion of this lecture the students will :

1. Define gastroesophageal reflux disease
2. Understand the Pathophysiology of reflux esophagitis.
3. Know clinical features of reflux esophagitis
4. Describe the pathological features of

Figure 2 Anatomic radiographic landmarks of the lower esophageal sphincter (LES).

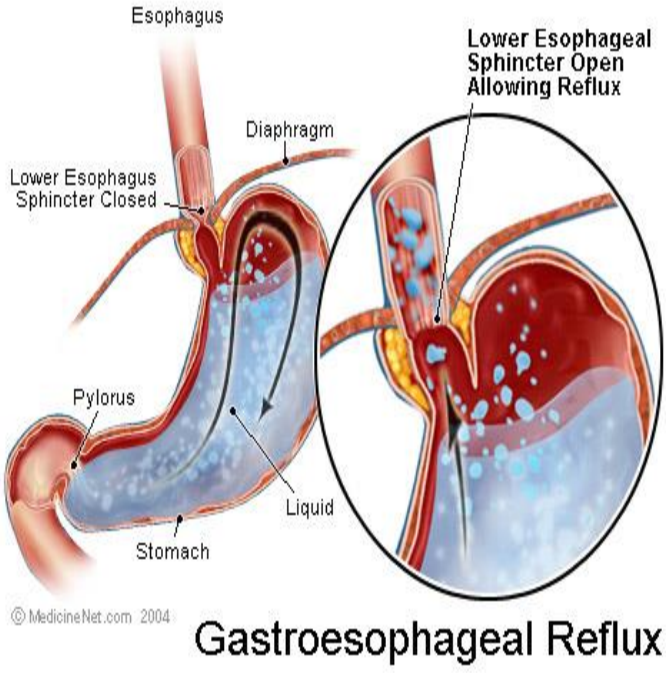


Gastroesophageal Reflux Disease (GERD)

- Gastroesophageal reflux is a normal physiologic phenomenon experienced intermittently by most people, particularly after a meal.
- Gastroesophageal reflux disease (GERD) occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury.

Definition

- American College of Gastroenterology (ACG)
  - Symptoms OR mucosal damage produced by the abnormal reflux of gastric contents into the esophagus
  - Often chronic and relapsing
  - May see complications of GERD in patients who lack typical symptoms



Gastroesophageal Reflux

Physiologic vs Pathologic

- Physiologic GERD
  - Postprandial
  - Short lived
  - Asymptomatic
  - No nocturnal symptomes
- Pathologic GERD
  - Symptoms
  - Mucosal injury
  - Nocturnal symptomes

Epidemiology

- About 44% of the US adult population have heartburn at least once a month
- 14% of Americans have symptoms weekly
- 7% have symptoms daily

GERD
Pathophysiology

- Abnormal lower esophageal sphincter
- or
- Increase abdominal pressure

GERD

Pathophysiology

A. Abnormal lower esophageal sphincter

1. Functional (frequent transient LES relaxation)
2. Mechanical (hypotensive LES)
3. Foods (eg, coffee, alcohol),
4. Medications (eg, calcium channel blockers),
5. Location ..... hiatal hernia

The most common cause of (GERD).

decrease the pressure of the LES.

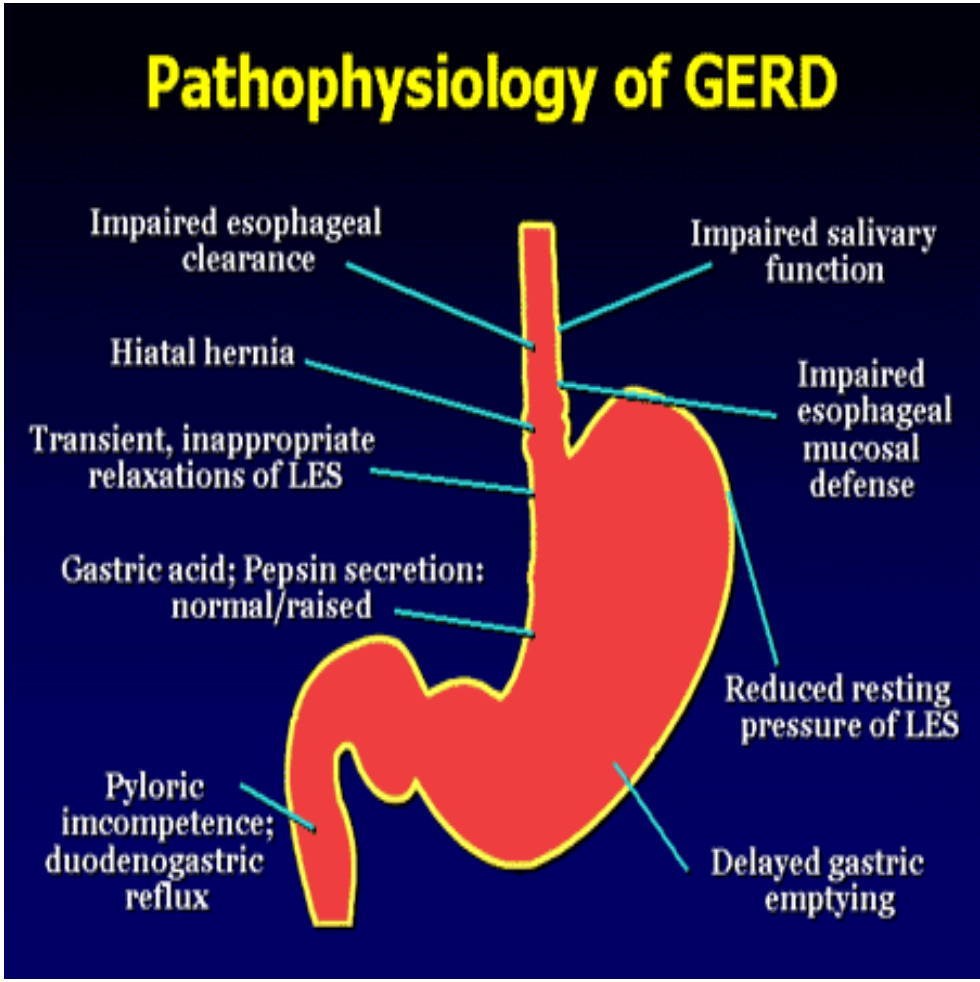
- or

B. Increase abdominal pressure

obesity
Pregnancy
increased gastric volume

Pathophysiology

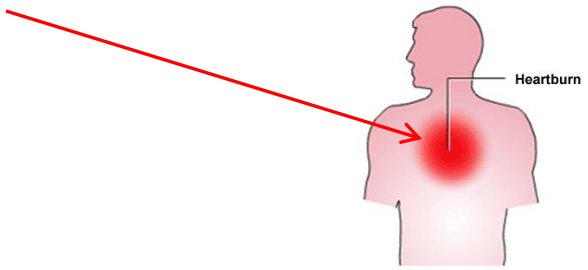
- Primary barrier to gastroesophageal reflux is the lower esophageal sphincter
- LES normally works in conjunction with the diaphragm
- If barrier disrupted, acid goes from stomach to esophagus



Clinical Manisfestations

- Most common symptoms
  - Heartburn—retrosternal burning discomfort
  - Regurgitation—effortless return of gastric contents into the pharynx without nausea, retching, or abdominal contractions

Atypical symptoms….coughing, chest pain, and wheezing.

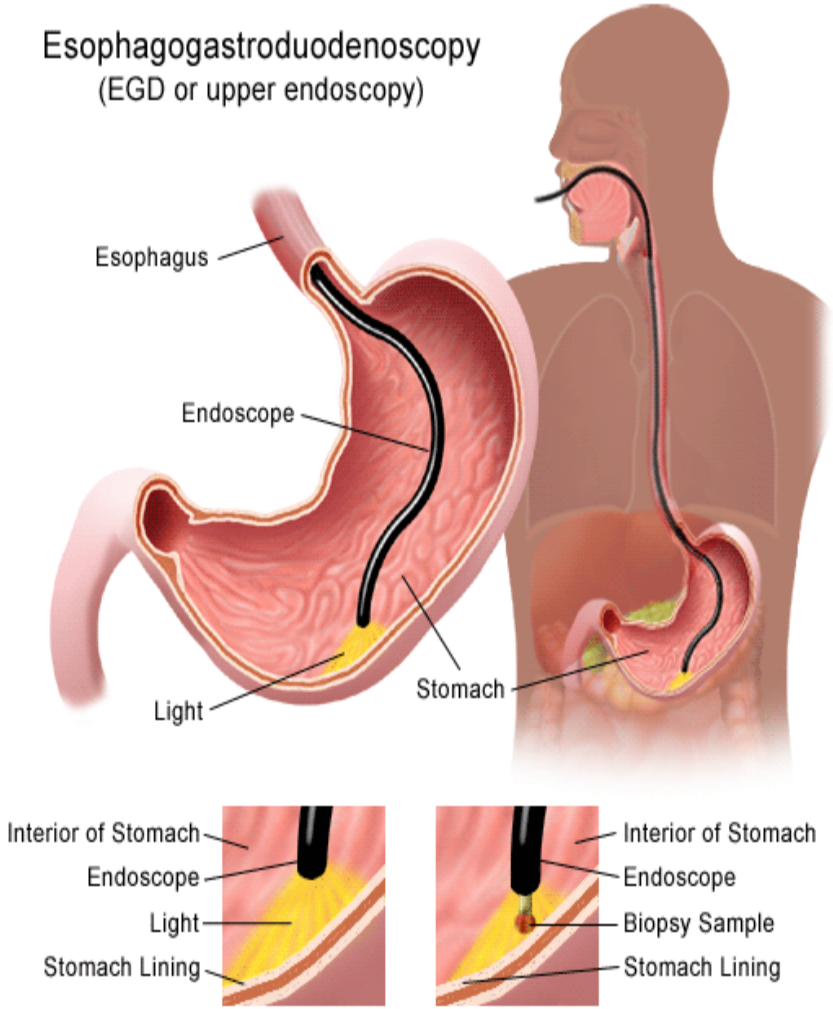


Diagnostic Evaluation

- If classic symptoms of heartburn and regurgitation exist in the absence of “alarm symptoms” the diagnosis of GERD can be made clinically and treatment can be initiated

Esophagogastrodudenoscopy

- Endoscopy (with biopsy if needed)
  - In patients with alarm signs/symptoms
  - Those who fail a medication trial
  - Those who require long-term tx



pH

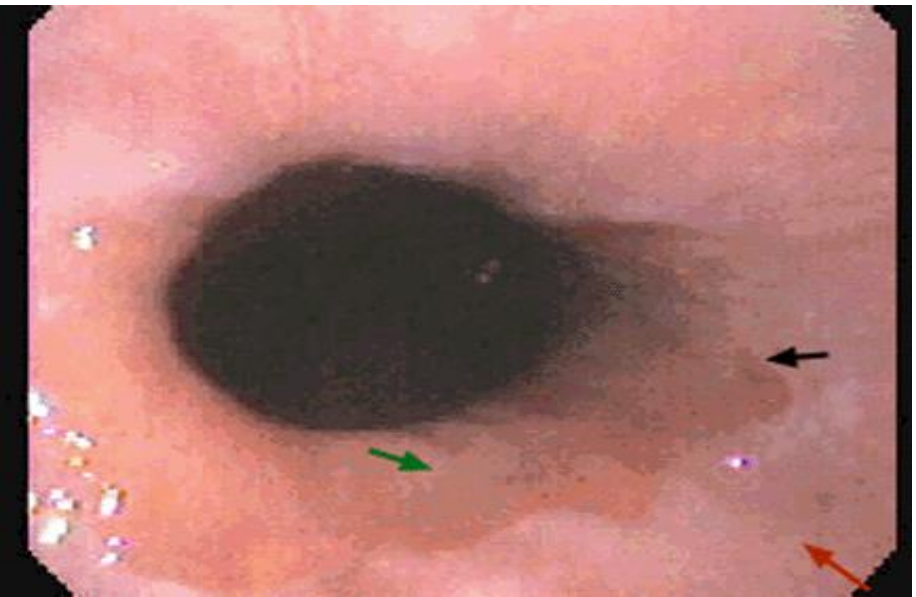
- 24-hour pH monitoring
  - Accepted standard for establishing or excluding presence of GERD for those patients who do not have mucosal changes
  - Trans-nasal catheter or a wireless capsule shaped device

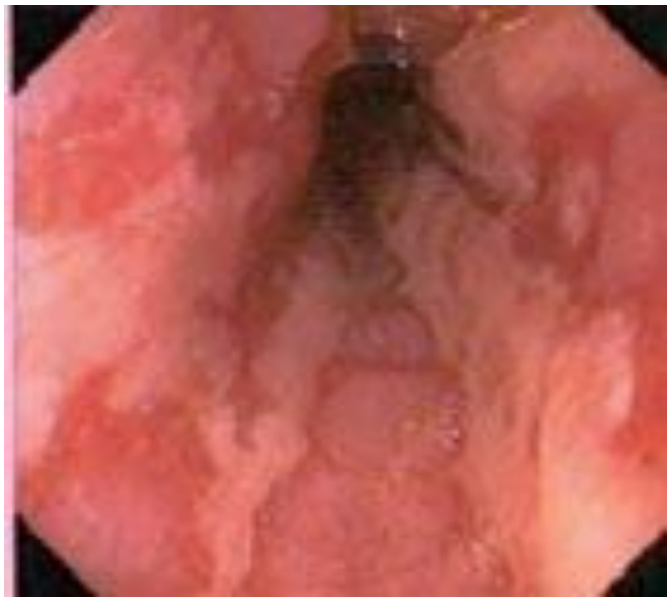
Complications

- Erosive esophagitis
- Stricture
- Barrett’s esophagus

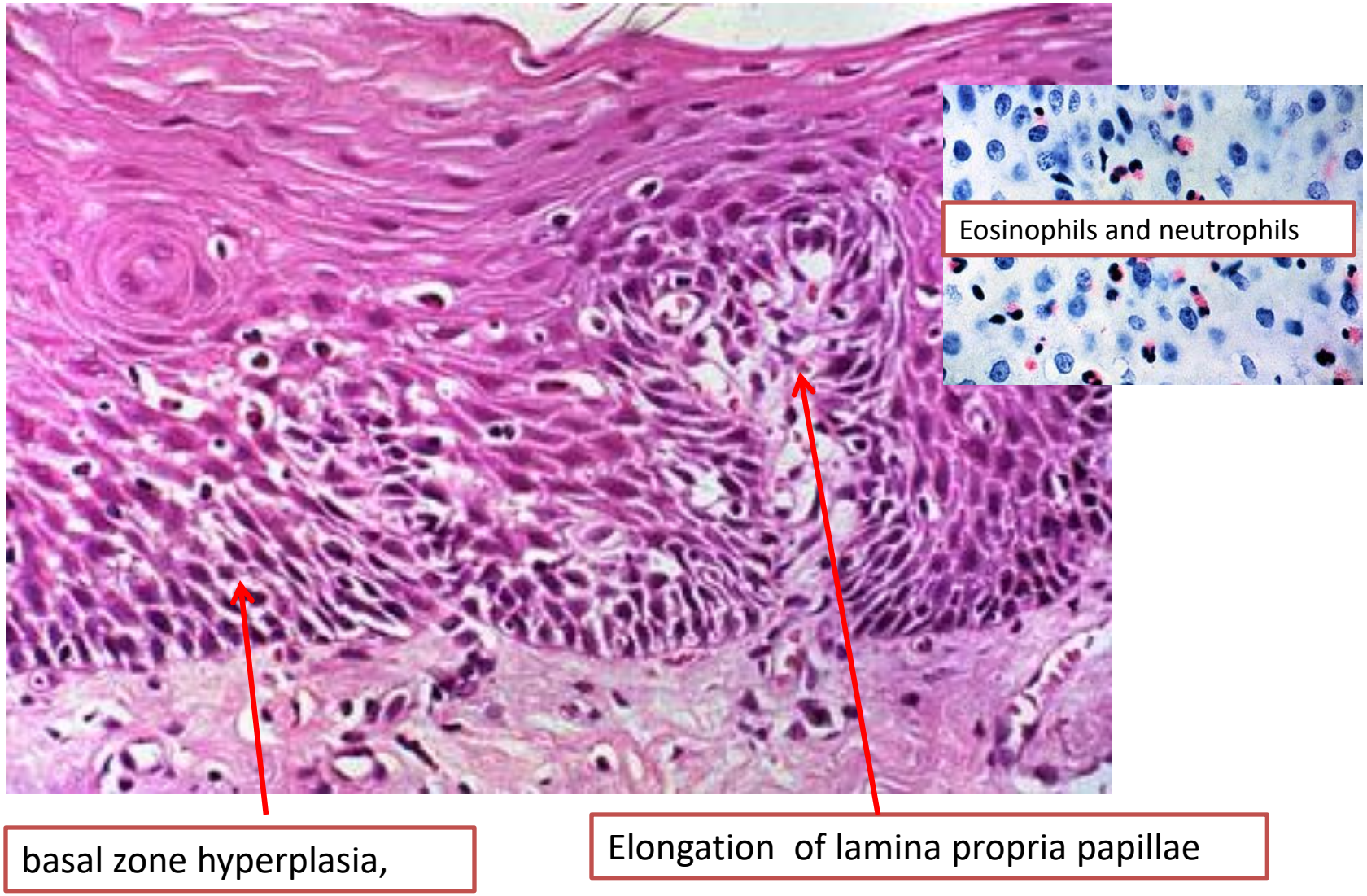
Complications

- Erosive esophagitis
  - Responsible for 40-60% of GERD symptoms
  - Severity of symptoms often fail to match severity of erosive esophagitis



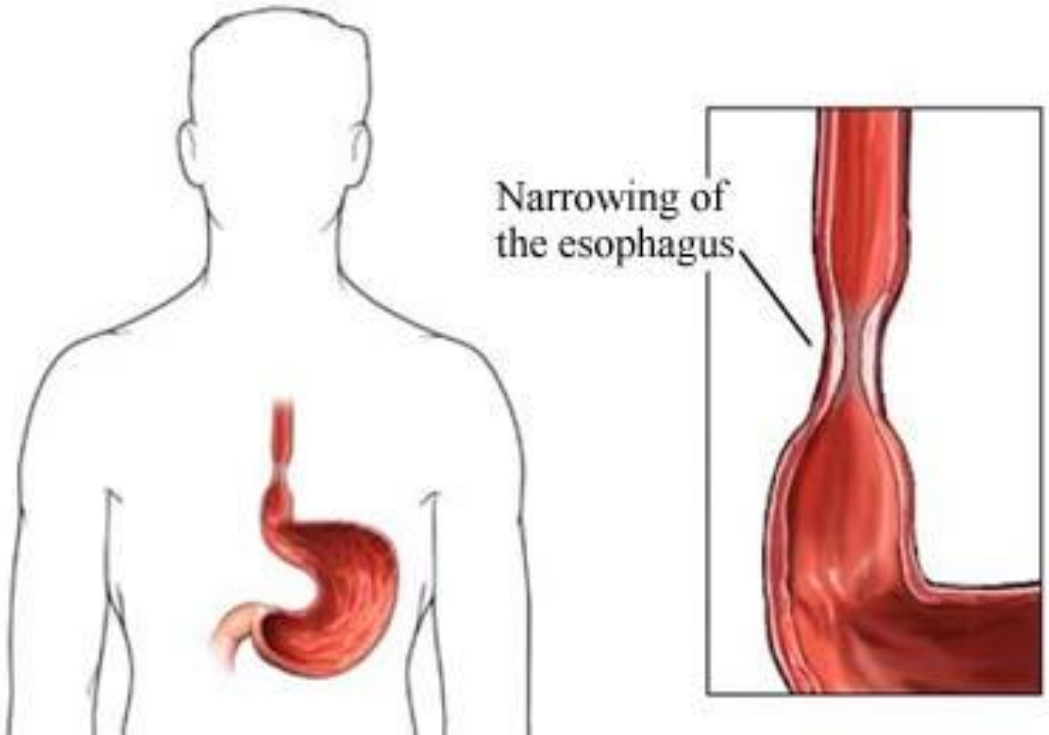


Esophagitis

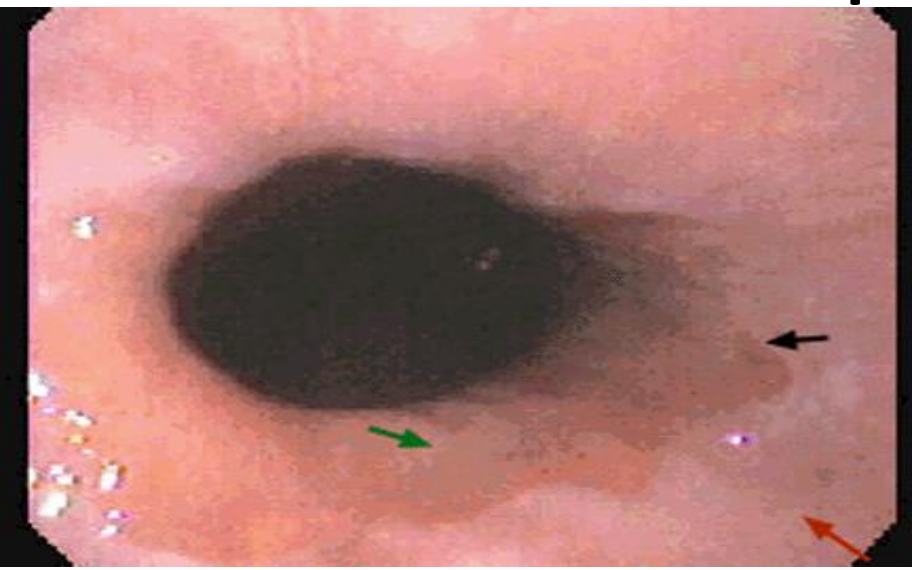


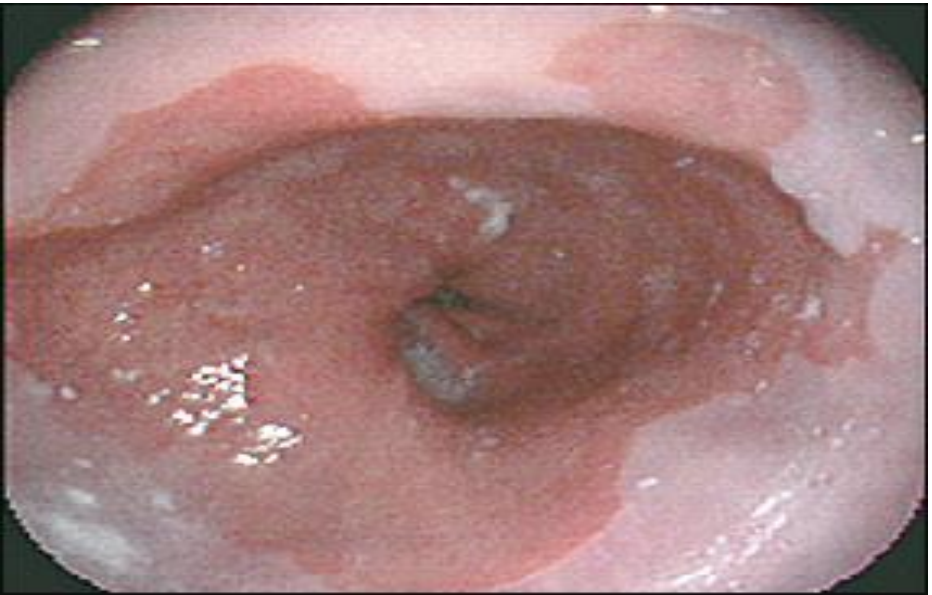
Complications

- Esophageal stricture
  - Result of healing of erosive esophagitis
  - May need dilation



Complications



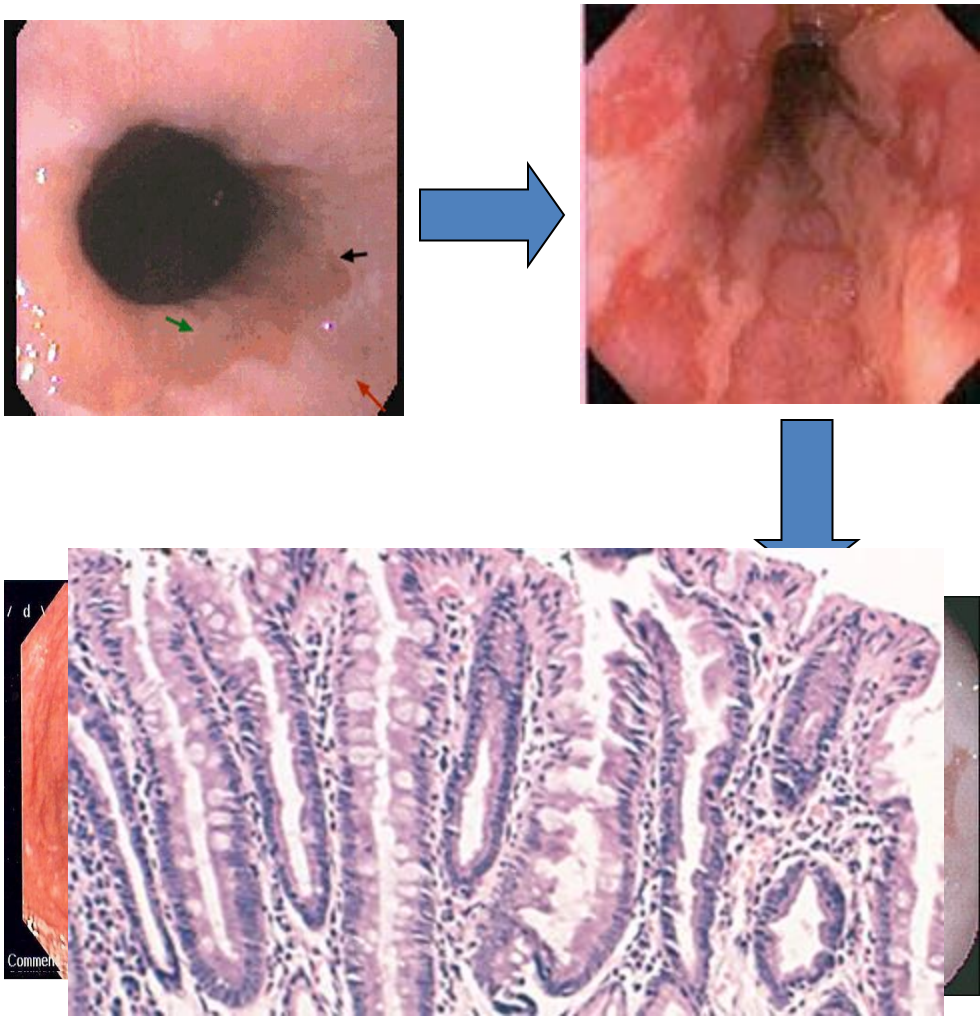


8-15%

- Barrett's Esophagus
  - Intestinal metaplasia of the esophagus
  - Associated with the development of adenocarcinoma

Complications

- Barrett's Esophagus
  - Acid damages lining of esophagus and causes chronic esophagitis
  - Damaged area heals in a metaplastic process and abnormal columnar cells replace squamous cells
  - This specialized intestinal metaplasia can progress to dysplasia and adenocarcinoma

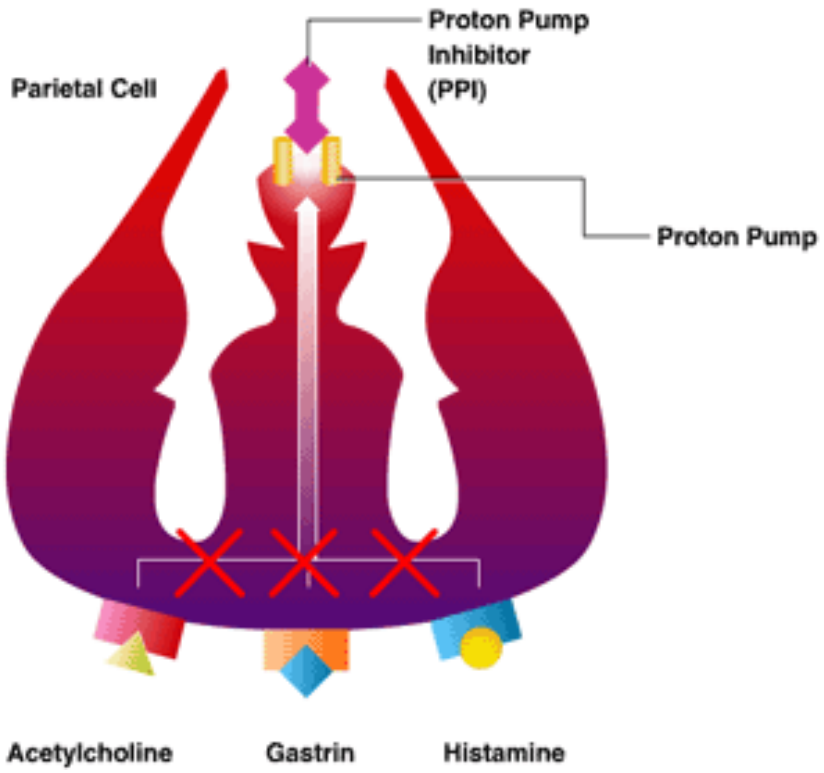


Many patients with Barrett's are asymptomatic

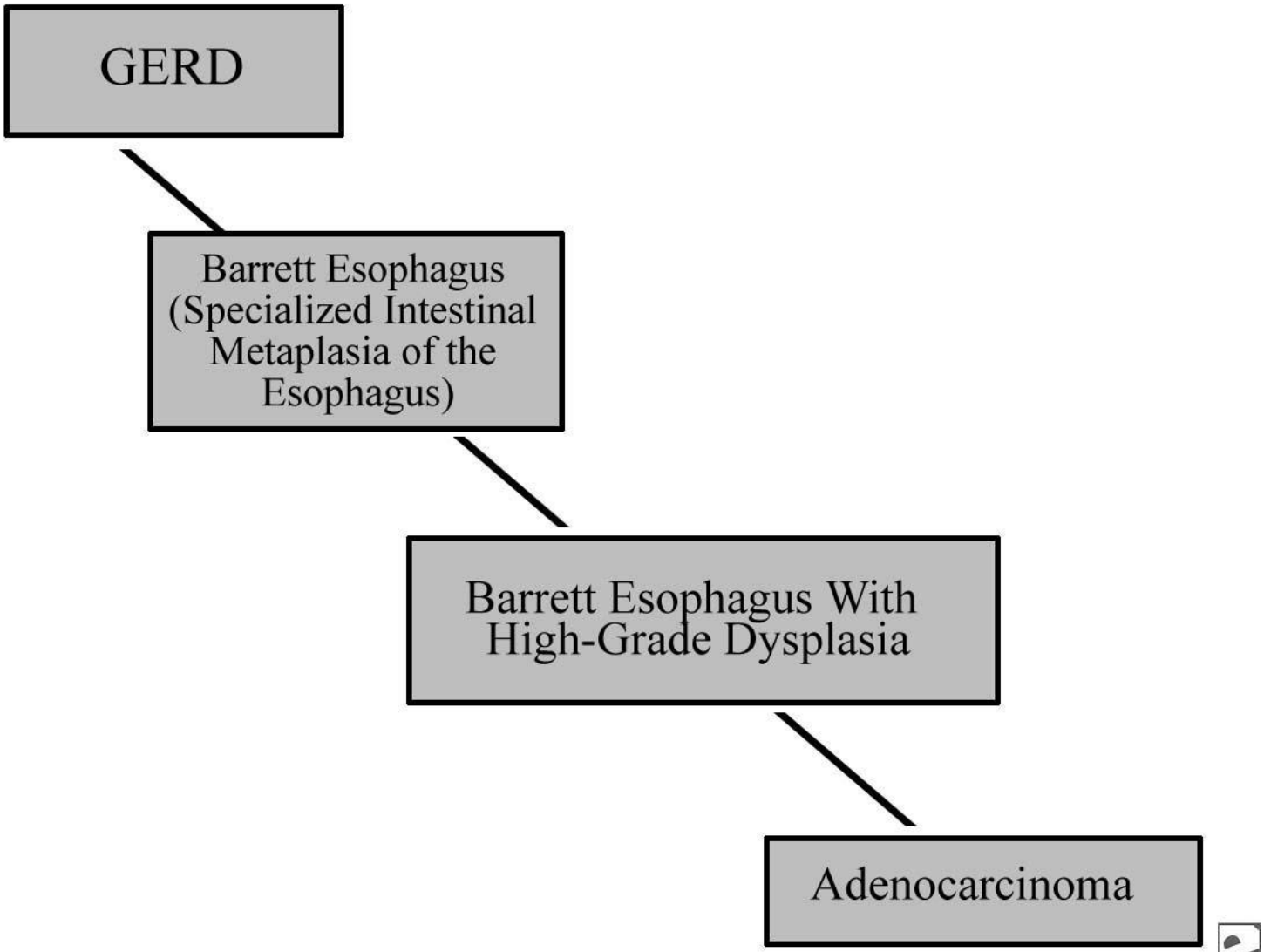
Treatment

- H 2 receptor Blockers
- Proton pump inhibitors

Antireflux surgery



Summary


GERD
Barrett Esophagus (Specialized Intestinal Metaplasia of the Esophagus)
Barrett Esophagus With High-Grade Dysplasia
Adenocarcinoma

Objectives

Upon completion of this lecture the students will :

1. Define gastroesophageal reflux disease
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4. Describe the pathological features of

Gastro-esophageal reflux disease

Peptic Ulcer Disease

Objectives

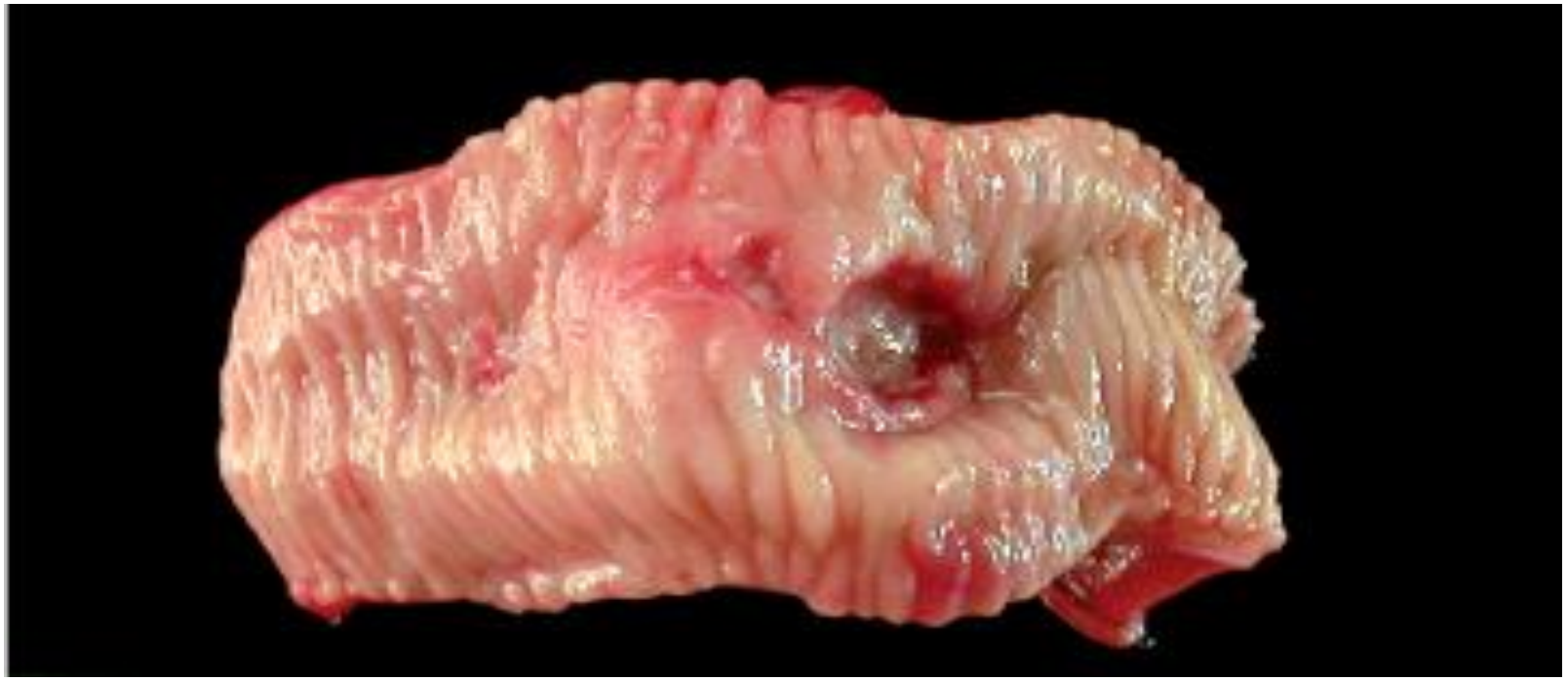
Upon completion of this lecture the students will :

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

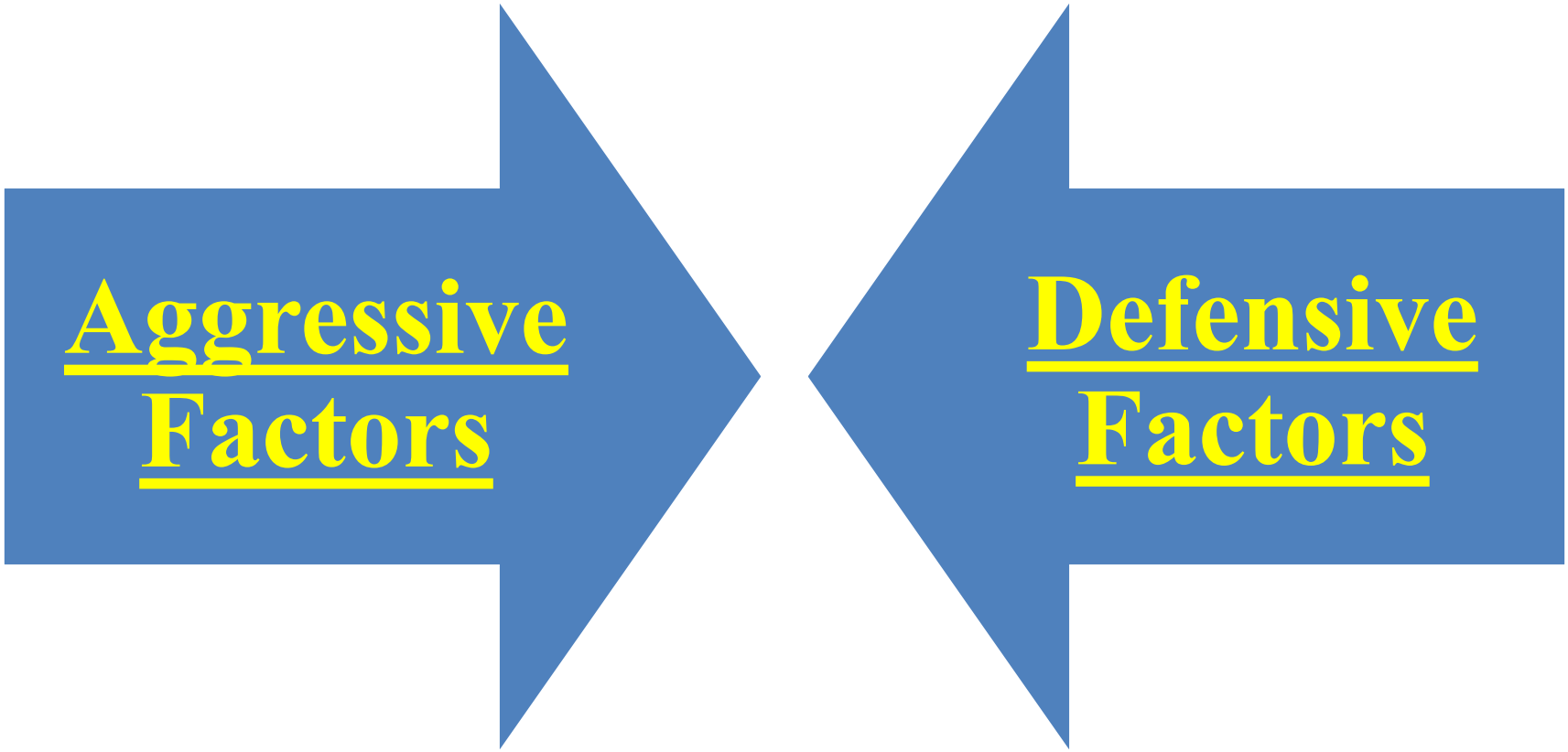
Ulcer

1. *Peptic ulcer*
2. *Stress ulcers (acute gastric ulcers)*

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



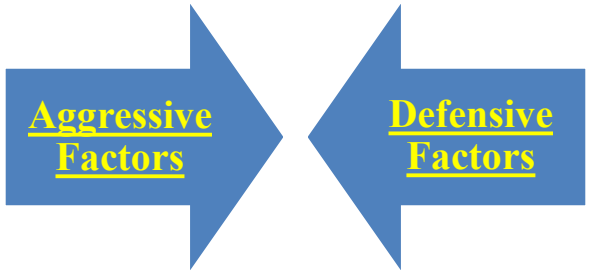
Pathophysiology



Pathophysiology

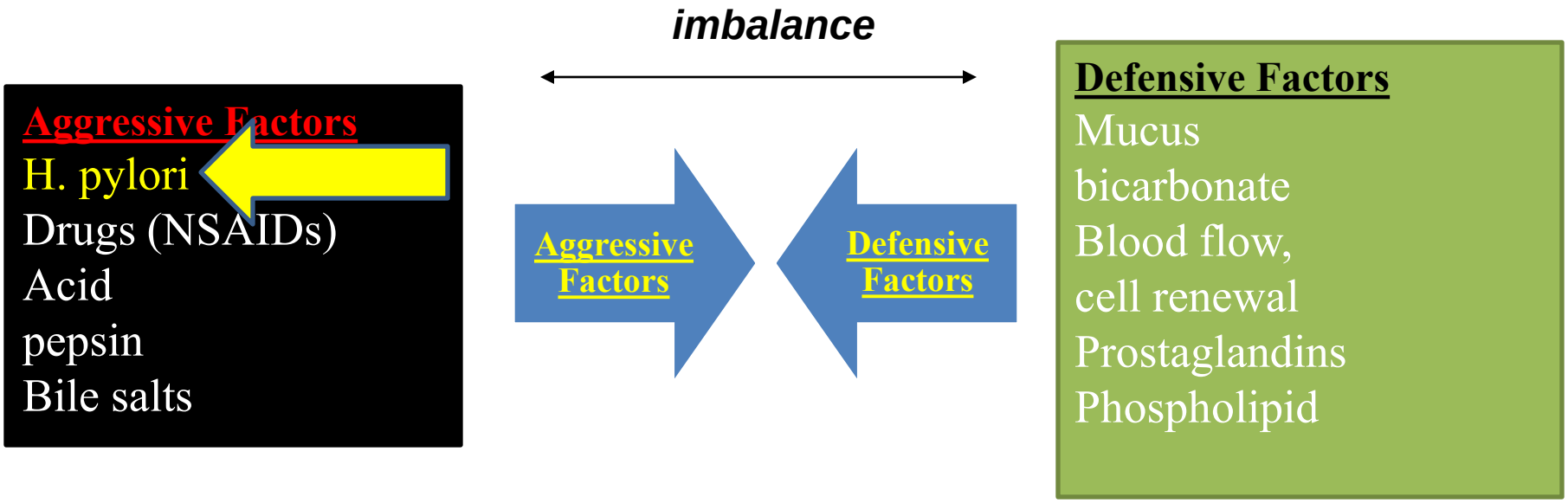
imbalance

Aggressive Factors
H. pylori
Drugs (NSAIDs)
Acid
pepsin
Bile salts



Defensive Factors
Mucus
bicarbonate
Blood flow,
cell renewal
Prostaglandins
Phospholipid

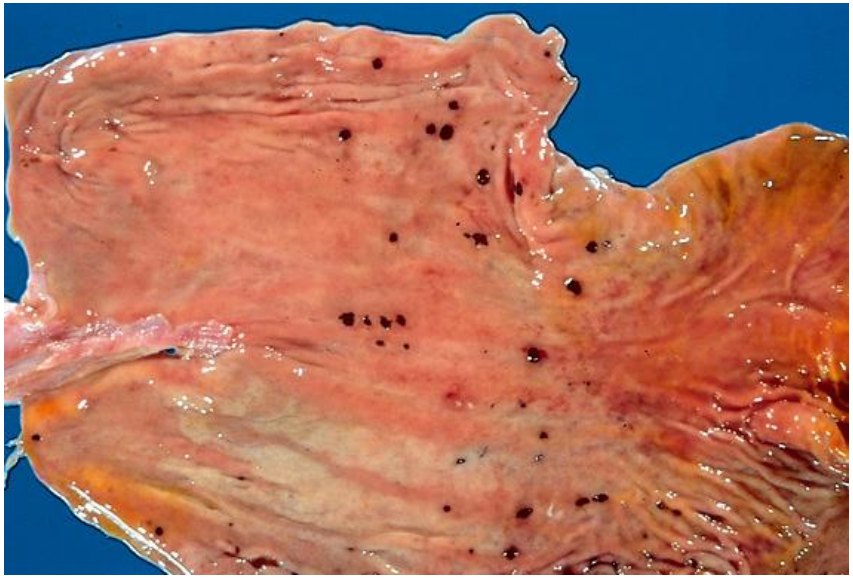
Pathophysiology



Acute peptic ulcers

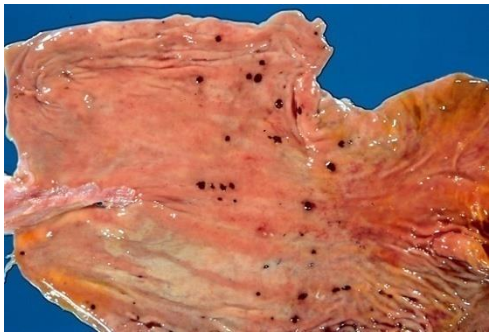
Pathophysiology

- As part of an acute *gastritis*
- As a complication of a severe *stress response*
- As a result of extreme *hyperacidity*.



Pathophysiology

Acute peptic ulcers

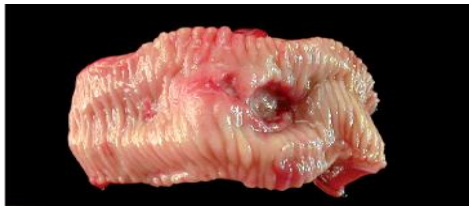


- As part of an acute *gastritis* (acute response to an irritant 'chemical' injury by drugs or alcohol

- As a complication of a severe *stress response* (severe burns (Curling's ulcer), major trauma or cerebrovascular accidents (cushing ulcer)

- As a result of extreme *hyperacidity* (Zollinger-Ellison syndrome).

Chronic peptic ulcer



Peptic Ulcer Disease

Locations

- May occur in any portion of the GI tract exposed to acidic gastric juices
- 98% located in first portion of duodenum or stomach, ratio = 4:1

- Esophagus……. as a result of GERD or acid secretion by ectopic gastric mucosa.
- Gastric mucosa within a Meckel diverticulum can result in peptic ulceration of adjacent mucosa.

Peptic Ulcer Disease

Gastric ulcers

Pathophysiology

- The mucosal defences against acid attack consist of:

1. Mucus-bicarbonate barrier

2. The surface epithelium.

Peptic Ulcer Disease

Pathophysiology

Gastric ulcers

- The mucosal defences against acid attack consist of:

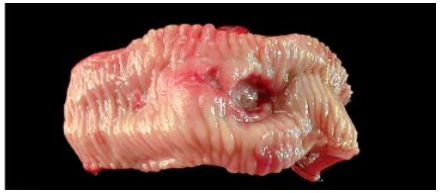
1. Mucus-bicarbonate barrier

Duodeno-gastric reflux (bile)

2. The surface epithelium.

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

Thus peptic ulcers in the stomach, breakdown of mucosal defence is much more important than excessive acid production.



Peptic Ulcer Disease

Duodenal ulcers

Pathophysiology

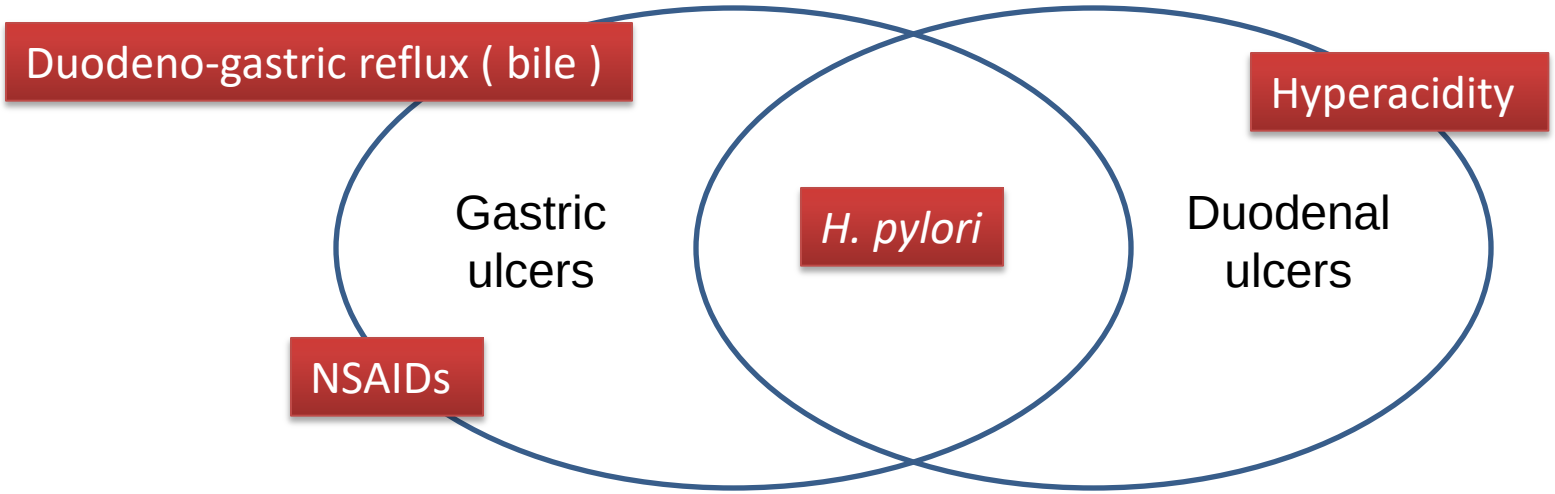
Increased production of acid assumes more importance in the pathogenesis of duodenal ulceration

H. pylori-infected individuals secrete 2-6 times as much acid as non-infected controls

Helicobacter Pylori does not colonise normal duodenal epithelium
Helicobacter is involved in duodenal ulceration because there is gastric metaplasia in response to excess acid. Gastric metaplasia paves the way for colonisation by *Helicobacter*

Increased production of acid + *Helicobacter P* = Duodenal ulcers

Peptic Ulcer Disease Pathophysiology



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.

Although more than 70% of individuals with PUD are infected by *H. pylori*, fewer than 20% of *H. pylori*–infected individuals develop peptic ulcer.

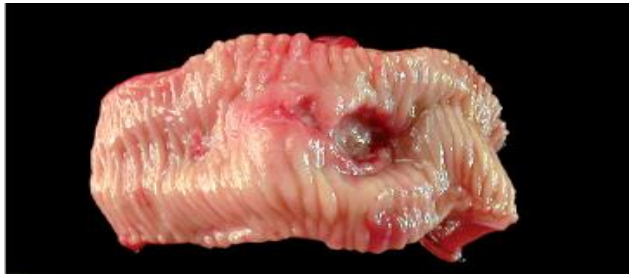
Morphology

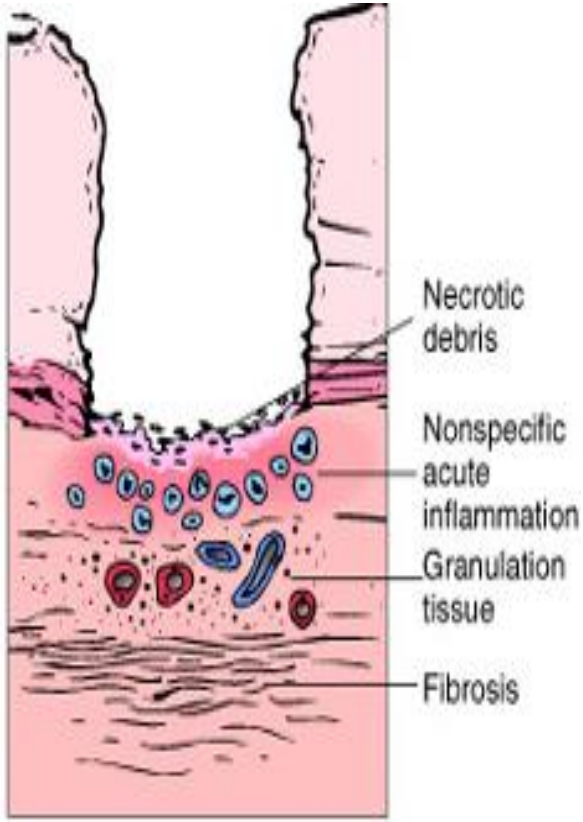
- Gross
- usually less than 20 mm in diameter but they may > 100 mm in diameter.

- **The classic peptic ulcer is a round to oval, sharply punched-out defect**

- **In contrast, heaped-up margins are more characteristic of cancers**

- Microscopy
- the base consists of necrotic tissue and polymorph exudate overlying inflamed granulation tissue which merges with mature fibrous (scar) tissue.





Clinical features

- Epigastric pain (the most common symptom)
  - Gnawing or burning sensation
  - Occurs 2-3 hours after meals
  - Relieved by food or antacids
  - Patient awakens with pain at night.

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

Therapy

Current therapies for PUD are aimed at

I. *H. pylori* eradication
II. Acid suppression

a) Proton pump inhibitors
b) H2 blockers

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