

Gastroesophageal Reflux Disease: GERD

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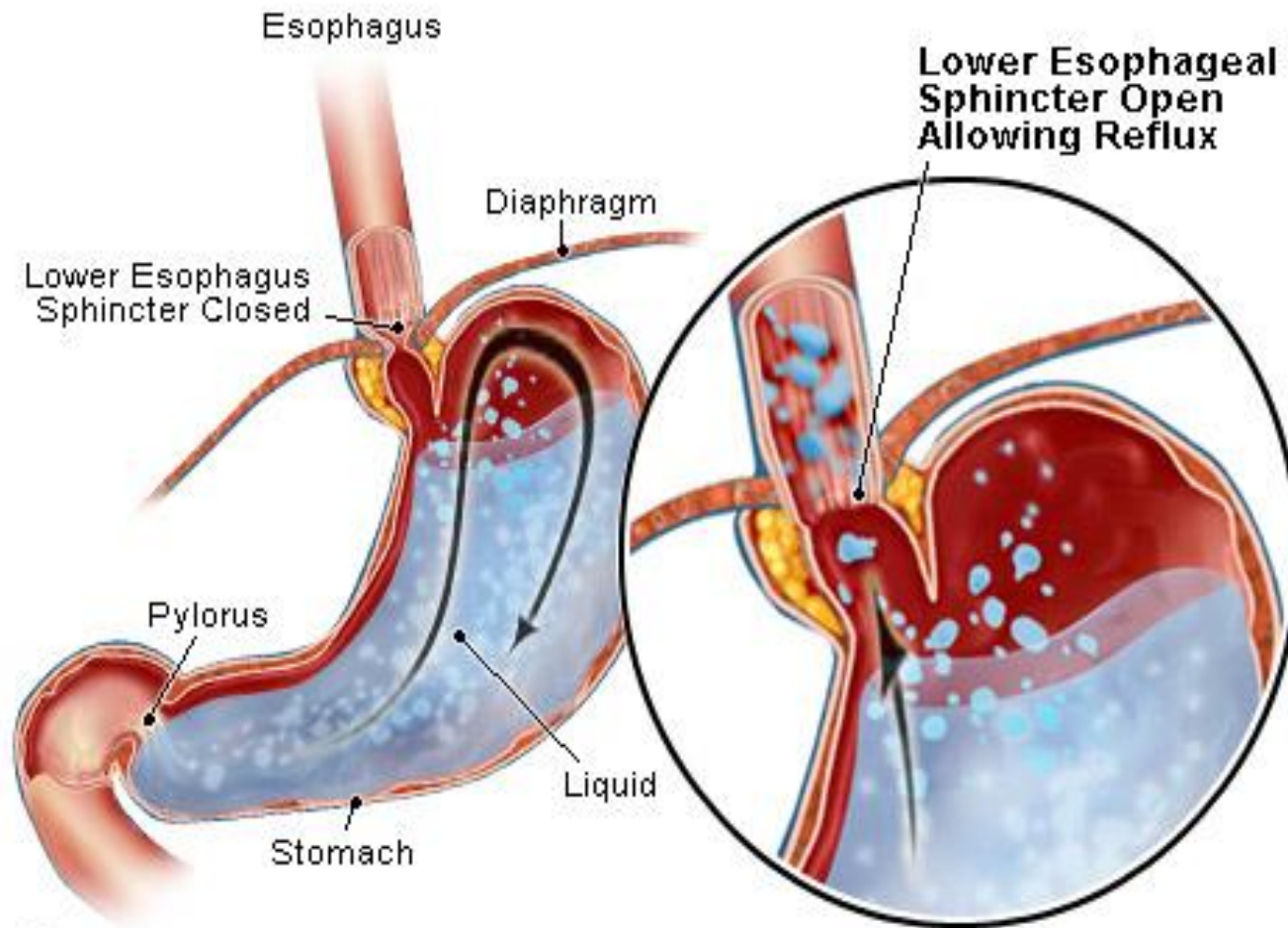
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Upon completion of this lecture, the students will :

- A. Understand the Pathophysiology of reflux esophagitis.**
- B. Know clinical features of reflux esophagitis**
- C. Describe the morphology (gross and microscopic features) of reflux esophagitis**
- D. Know the complications of reflux esophagitis**



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Gastroesophageal Reflux

Symptoms OR mucosal damage, produced by the abnormal reflux of gastric contents into the esophagus. Often chronic and relapsing.

Reflux??

- 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 that are with or without associated esophageal mucosal injury.

Physiologic vs Pathologic (how to differentiate?)

- Physiologic GERD
 - Postprandial:
after eating a meal
 - Short lived
 - Asymptomatic
 - No nocturnal symptoms
not during sleep
- Pathologic GERD
 - Symptoms
 - Mucosal injury
 - Nocturnal symptoms
during sleep

Pathophysiology of GERD

GERD is caused by either:

- Abnormal Lower Esophageal Sphincter (LES).

OR

- Increased abdominal pressure.

Pathophysiology

Abnormal **L**ower **E**sophageal **S**phincter (**LES**) :

1. **Functional** (Frequent transient LES relaxation)
2. **Mechanical** (Hypotensive LES)
3. **Foods** (e.g. Coffee, alcohol)
4. **Medications** (e.g. Calcium channel blockers)
5. **Location** (Hiatal Hernia)

The most common cause of GERD .

Decrease the pressure of the LES.

Hypotensive LES: The LES resting pressure below 10 mmHg with normal esophageal body contractions (**low pressure on LES**)

Hiatal Hernia : occurs when part of the stomach protrudes into the thoracic cavity through the *esophageal Hiatus* of the diaphragm

Normal pH of stomach is from 1.3 to 3

Pathophysiology

Increased Abdominal Pressure:

1- Obesity

2- Pregnancy

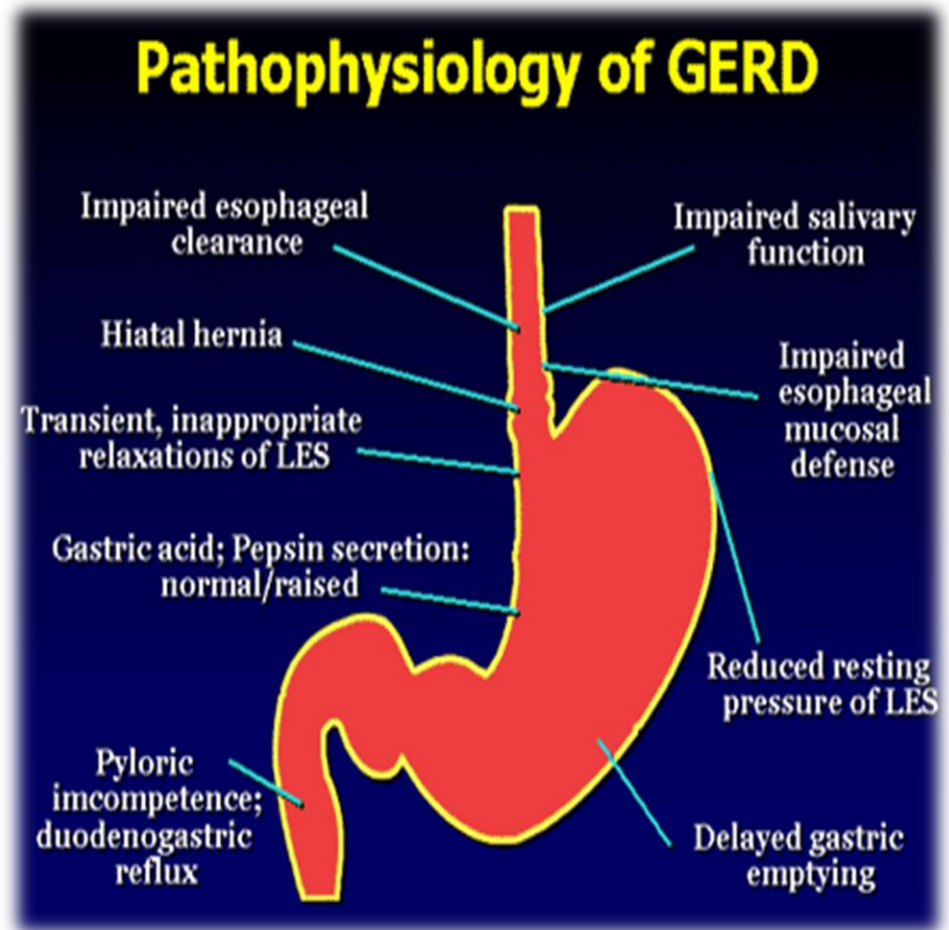
3- Increased Gastric Volume

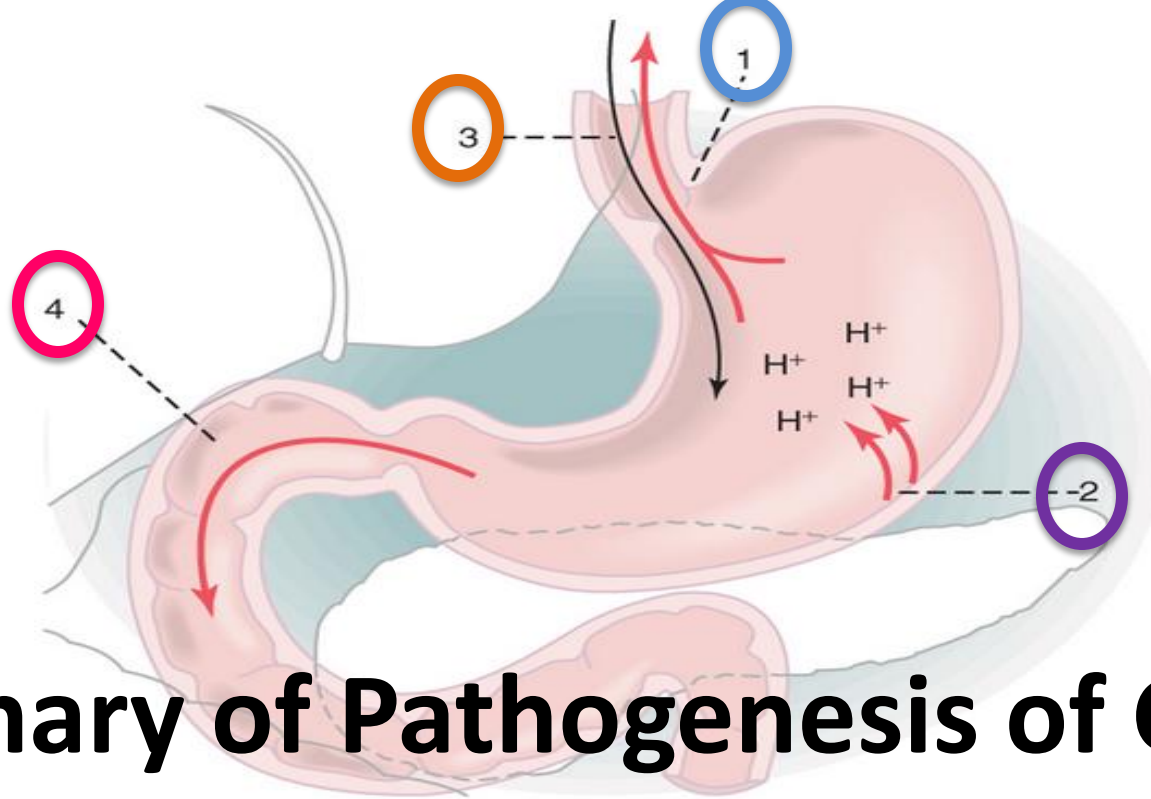
(Delayed Gastric Emptying → ↑ Gastric Volume)

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.





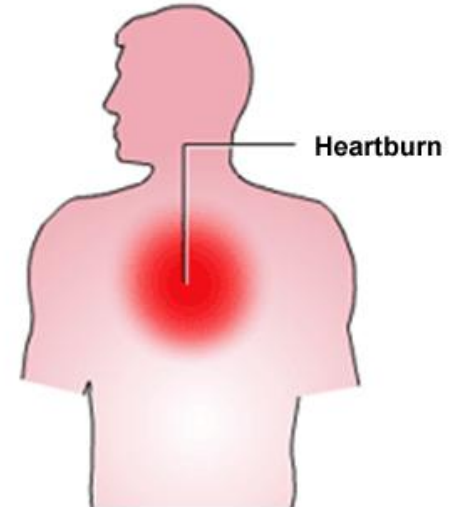
Summary of Pathogenesis of GERD

- 1- Impaired LES, lower pressures or frequent transient lower esophageal sphincter relaxation.
- 2- Hyper-secretion of acid .
- 3- Decreased acid clearance resulting from impaired peristalsis or abnormal saliva production.
- 3- delayed gastric emptying or duodenogastric reflux of bile salts and pancreatic enzymes.

Clinical Manifestations of GERD

1) Most common symptom is **heartburn**:

- a. Substernal pain
- b. Radiates to shoulder, throat, and back
- c. often occurs at night
- d. severe; after meals.



*because of its location it could be confused with angina.

*may take minutes to hours to manifest
(do not feel it immediately)

*aggravated by stress

*Relieved by antacids

2) Regurgitation

effortless return of gastric contents into the pharynx without nausea, retching, or abdominal contractions (particularly on bending or lying down)

3) Respiratory symptoms; wheezing , chronic cough, hoarseness and chest pain. ((due to Reflux of gastric contents))

**is sometimes associated with asthma*

Less Common Symptoms:

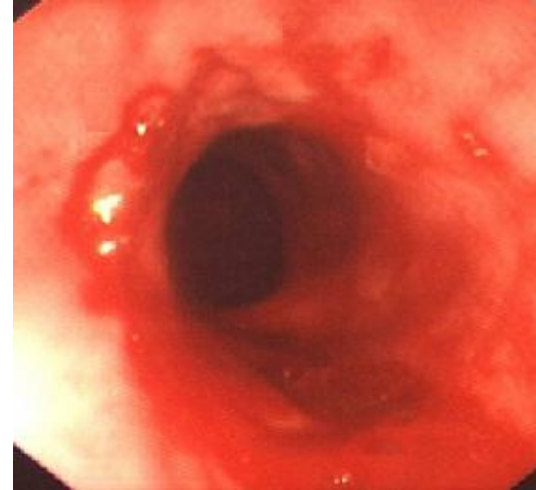
- **Water brash (heartburn):** regurgitation of fluid that may be sour or almost tasteless
- Hyper-salivation
- **Globus:** Lump in throat irrespective of swallowing
- **Odynophagia:** painful swallowing
- Esophageal ulcer

Peptic Ulcer is the Chronic Pattern of GERD

Morphology of GERD

Macroscopically:

- Mild: hyperemia, with virtually no histologic abnormality [nonerosive reflux disease (NERD)]



- Severe: Erythema (redness: hyperemia), mucosal edema, erosions, or ulcers



A Severe esophagitis

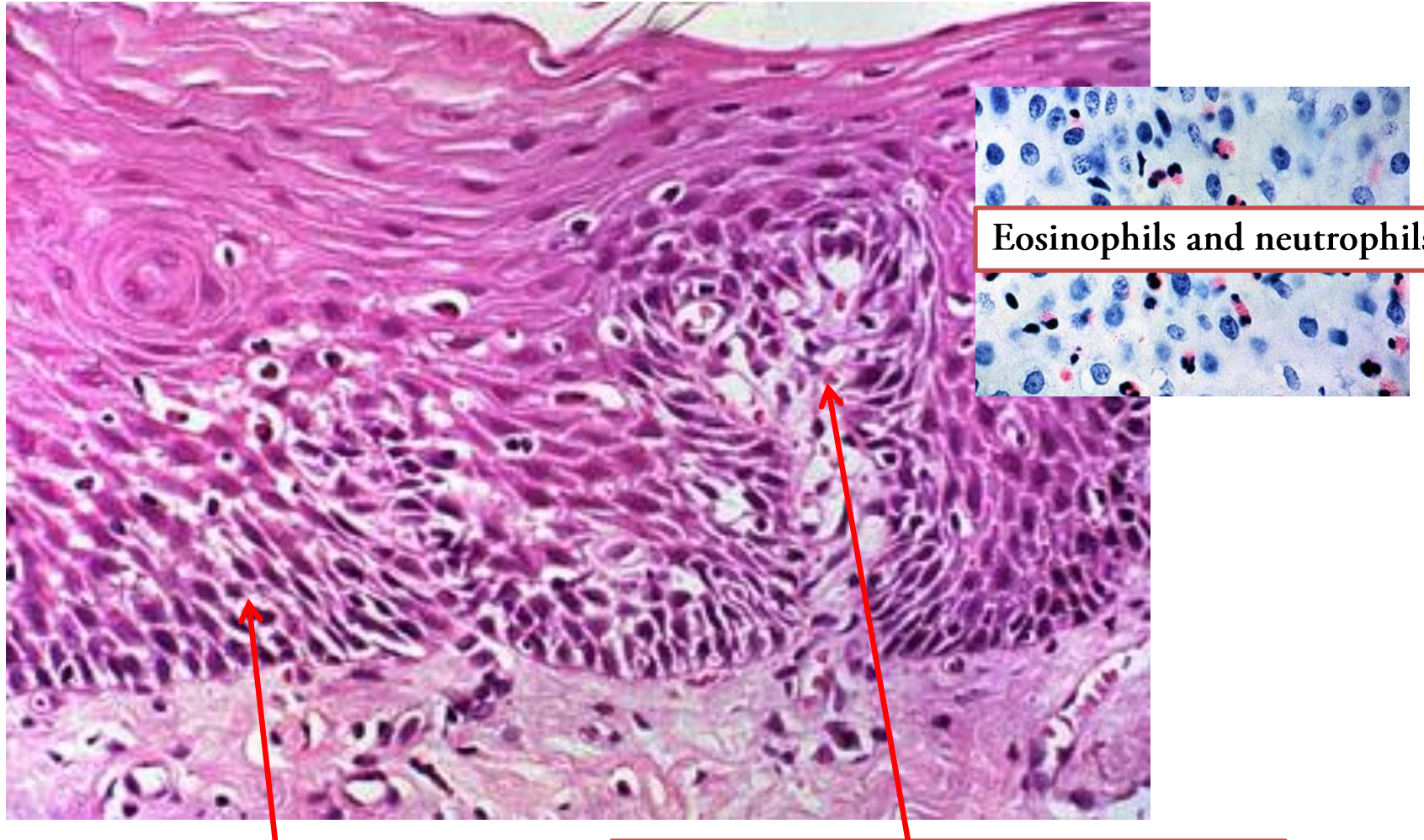
Morphology

Microscopically

- Uncomplicated esophagitis:
 1. *Eosinophils*, with or without neutrophils, in the epithelial layer
 2. *Basal zone hyperplasia*: basal layer representing more than 15% of the total thickness of the epithelium.
 3. *Elongation of lamina propria papillae*: papillae extending more than two-third of the distance to the epithelium surface.
- With more severe injury:

Intra-epithelial *neutrophils*

Esophagitis



Eosinophils and neutrophils

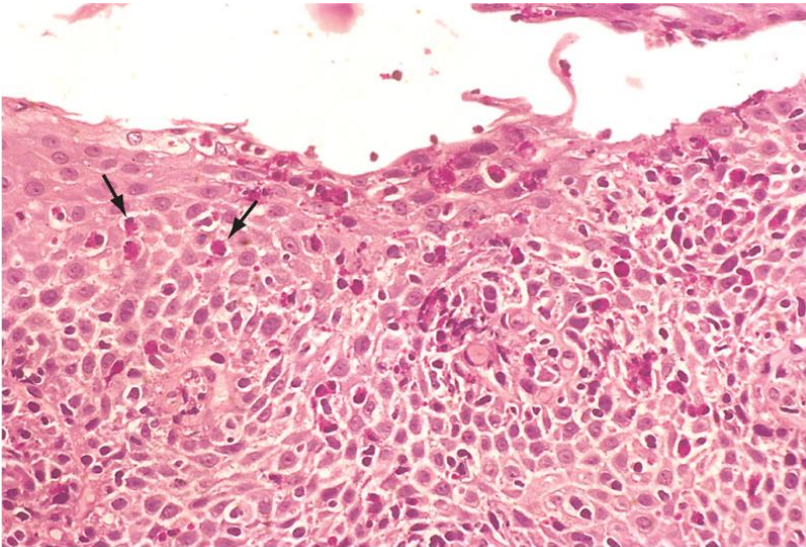
basal zone hyperplasia,

Elongation of lamina propria papillae

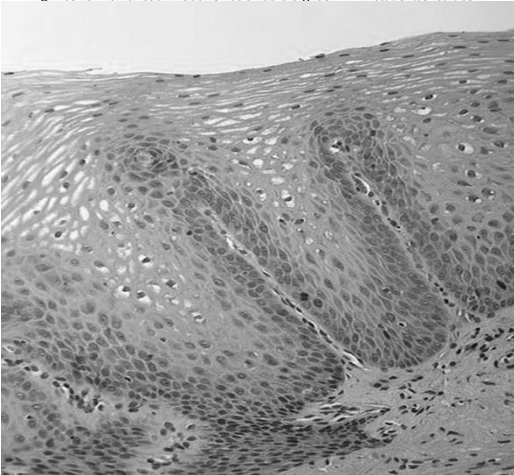
Microscopically

- ↙ Reflux esophagitis showing the superficial portion of the mucosa.

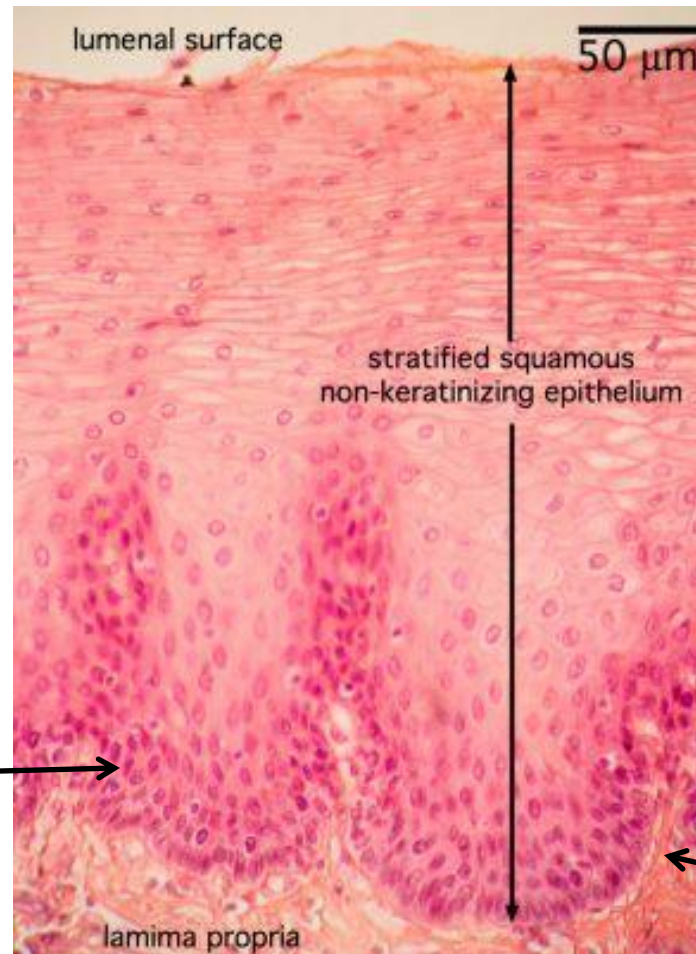
Numerous eosinophils (arrows) are present within the mucosa, and the stratified squamous epithelium has not undergone complete maturation because of ongoing inflammatory damage



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While Normal Histology:



Basal layer:
small cells with
round nuclei –
occupy only 15
% of epithelial
layer

Papillae do not
extend deep
into epithelium

Diagnostic Evaluation

If classic symptoms of heartburn and regurgitation
exist in the absence of “alarm symptoms”
(such as **Bleeding**)
the diagnosis of GERD can be made
CLINICALLY and treatment can be initiated.

BUT

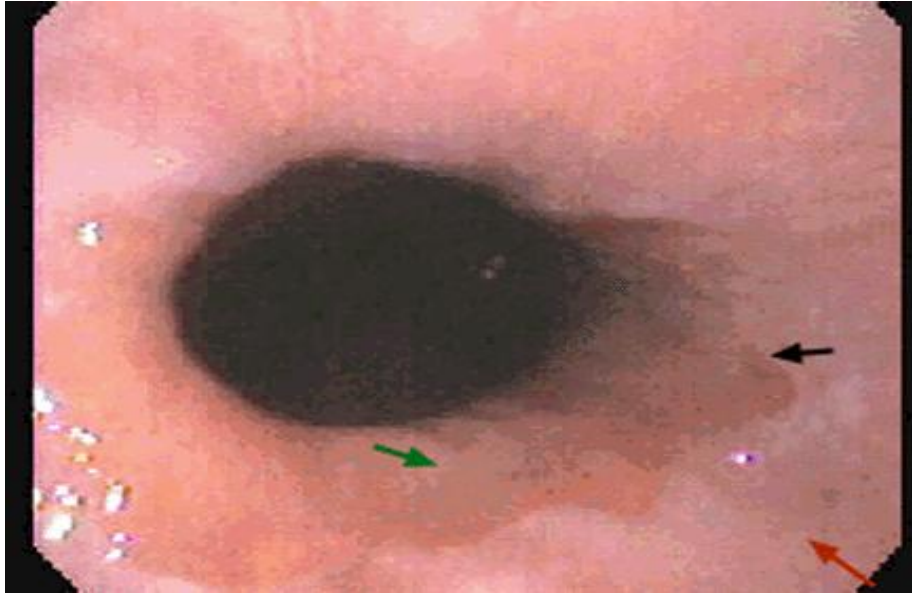
If there is an “alarm symptom”
→ we must do endoscopy

Complications of GERD

- Erosive esophagitis
 - Stricture
- Barrett's esophagus

Erosive esophagitis

- Any swelling, inflammation or irritation of the esophagus
- Responsible for 40-60% of GERD symptoms
- Severity of symptoms often fail to match severity of erosive esophagitis:
Its symptoms are not always severe as it is



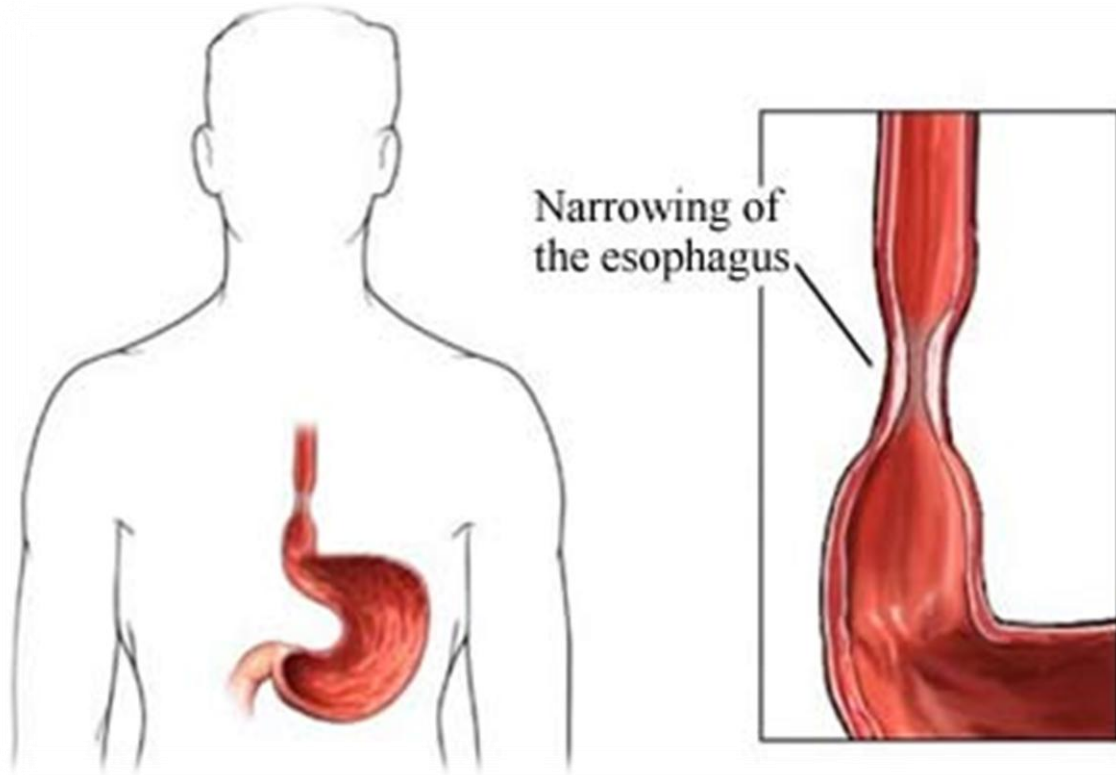
Normal: is light colored

Abnormal: becomes more red or darker in color

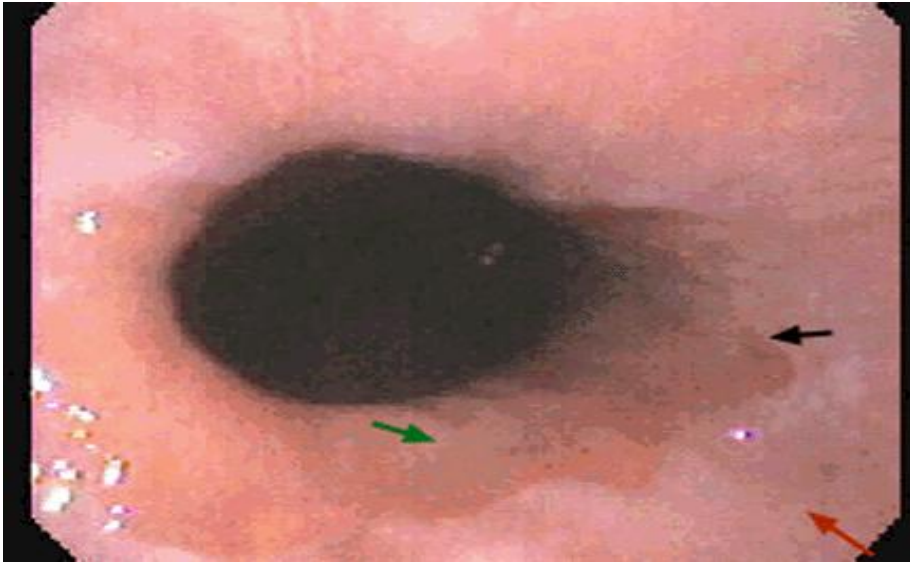
Inflammatory appearance of the esophagus.

Esophageal stricture

- A narrowing or tightening of the esophagus
- Is a result of healing of erosive esophagitis
- May need dilation



Barrett's Esophagus



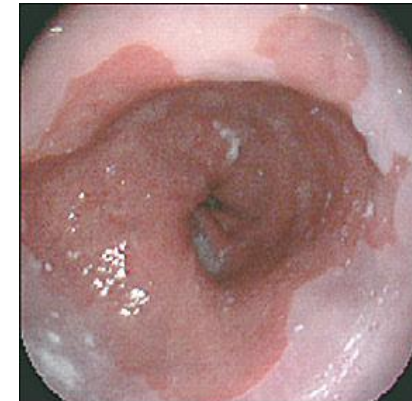
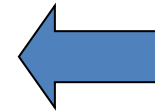
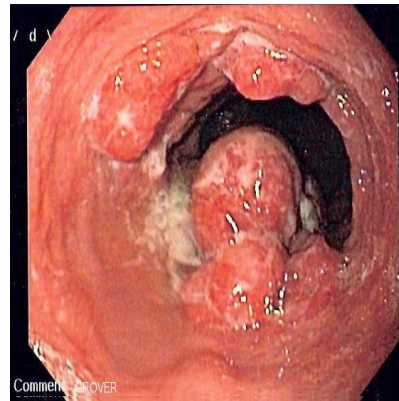
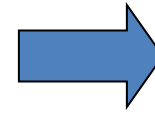
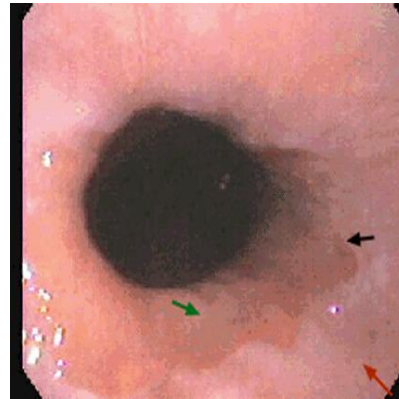
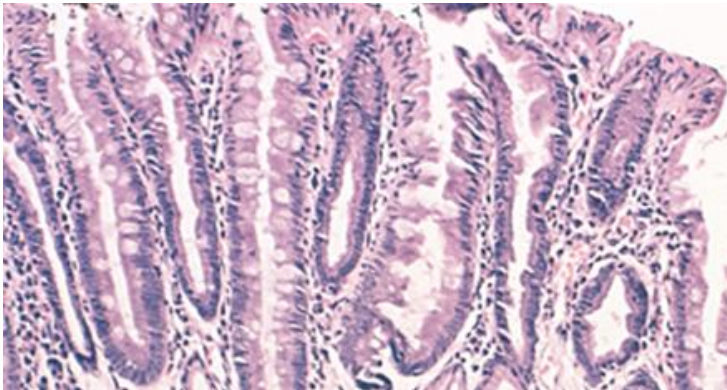
8-15%

- Intestinal metaplasia of the esophagus
change of normal epithelium of esophagus into an **intestinal epithelium** (columnar epithelium) as a process of protection from acids.
- Usually, develops into **Adenocarcinoma**



Pathophysiology of Barrett's Esophagus

- The 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

GERD

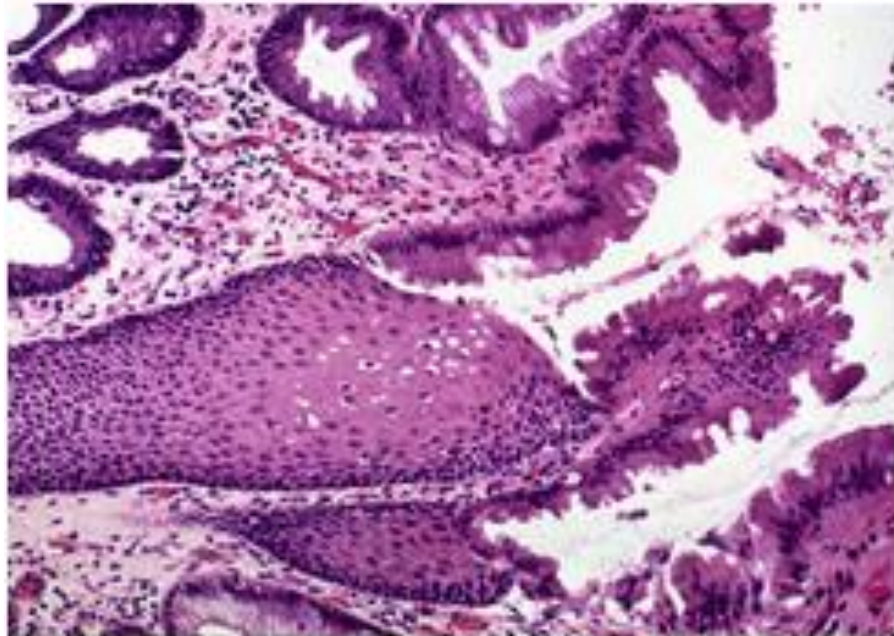
Barrett Esophagus
(Specialized Intestinal
Metaplasia of the
Esophagus)

Barrett Esophagus With
High-Grade Dysplasia

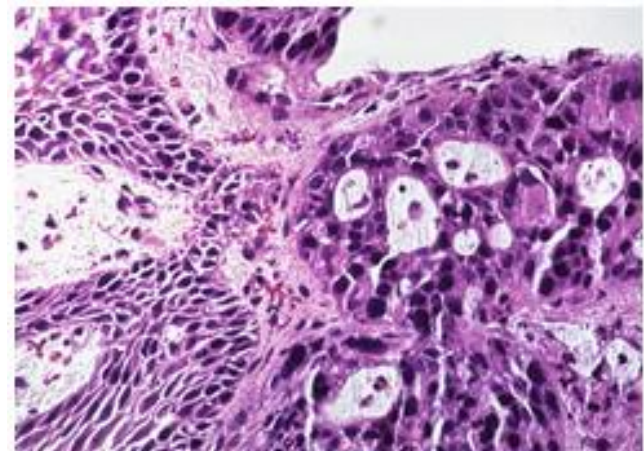
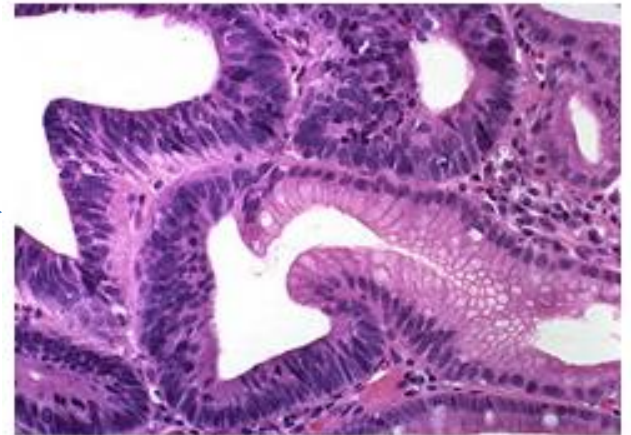
Adenocarcinoma



Barrett's esophagus



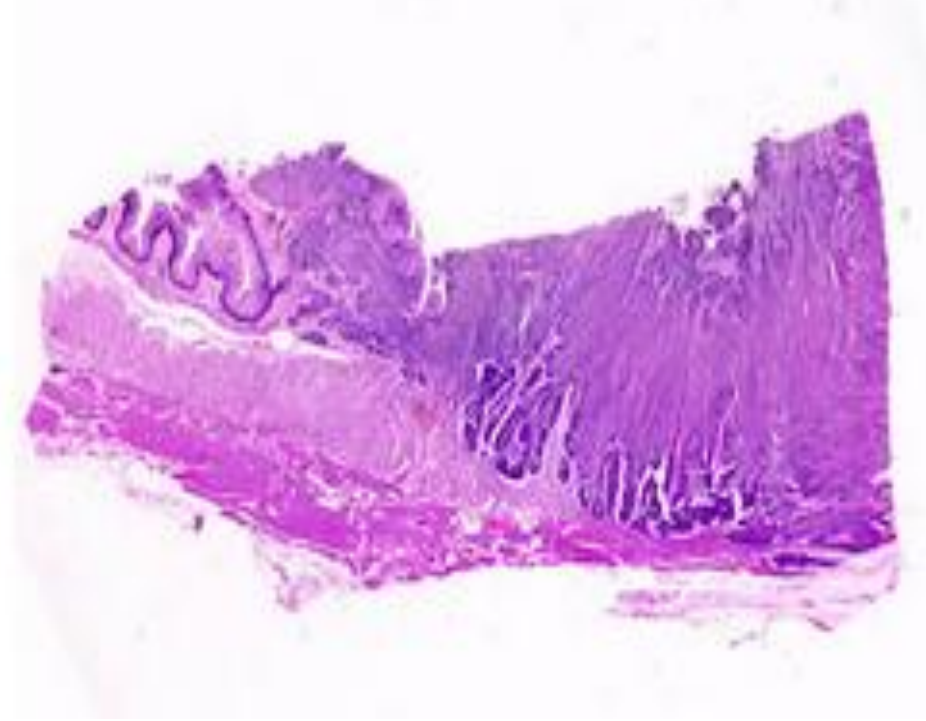
dysplasia



adenocarcinoma

- The risk of cancer in Barrett's esophagus is estimated to be 40 to 100 times
- Endoscopic surveillance (monitoring behaviour) is recommended for all patients with Barrett's esophagus. Endoscopy is performed every 2 years, and biopsies are taken from the area of abnormal mucosa.
- If the biopsies reveal low-grade dysplasia, then the frequency of endoscopies is increased.
- If high-grade dysplastic changes are seen and confirmed by a second pathologist, then the risk of subsequent adenocarcinoma is greater than 25%, and surgical resection of esophagus should be considered.

Adenocarcinoma



Treatment

- H₂ receptor Blockers
- Proton pump inhibitors



Antireflux surgery

