بسم الله الرحمن الرحيم

PATHOLOGY

MID | Lecture 1

Esophageal diseases 1

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﴿ وَإِن تَتَوَلَّوْا يَسْتَبْدِلْ قَوْمًا غَيْرَكُمْ ثُمَّ لَا يَكُونُوا أَمْثَالَكُم ﴾

اللهم استعملنا ولا تستبدلنا

Diseases of the esophagus-1

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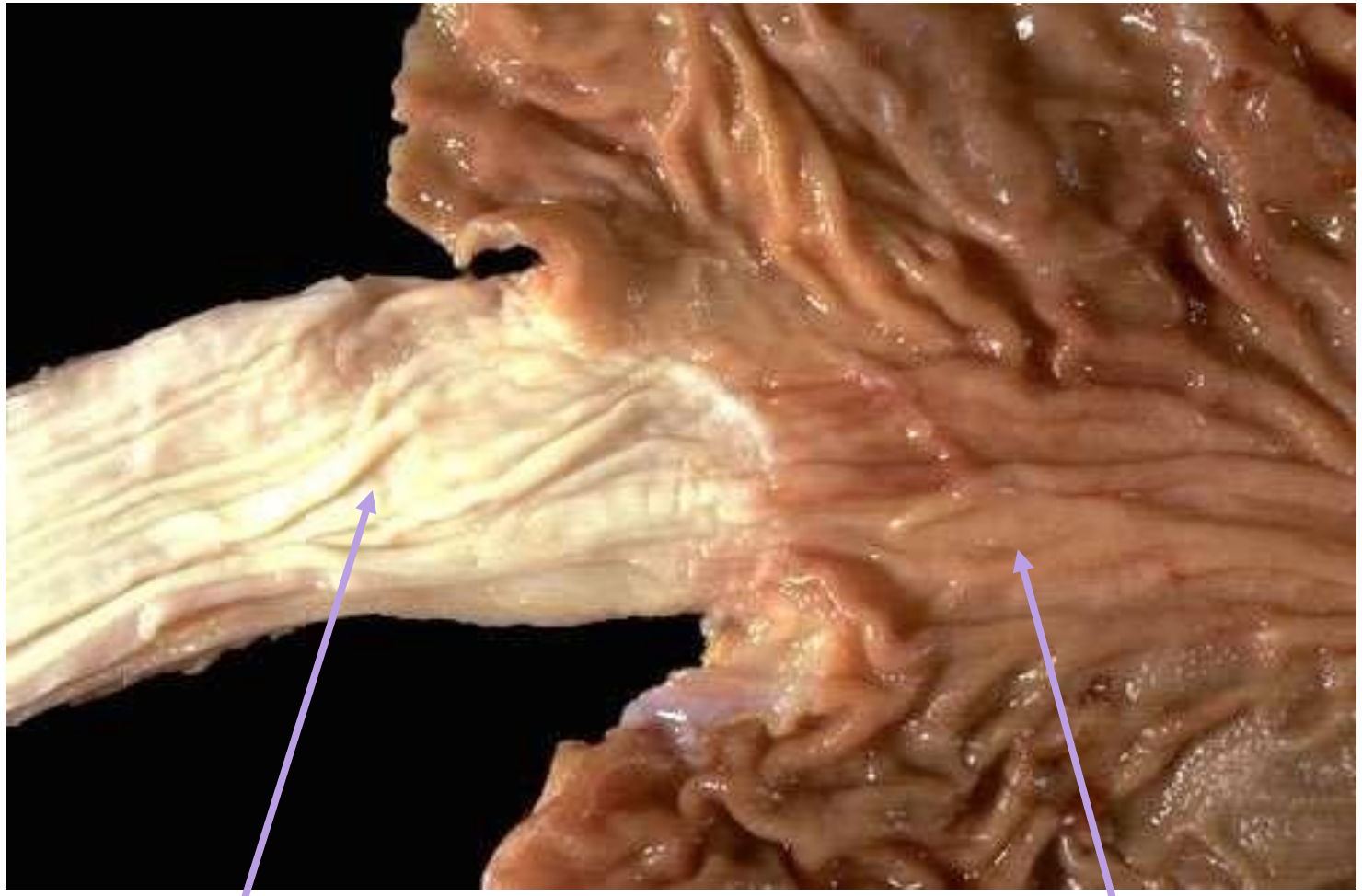
Anatomy and histology:

Muscular tube extending from the epiglottis to the GEJ.

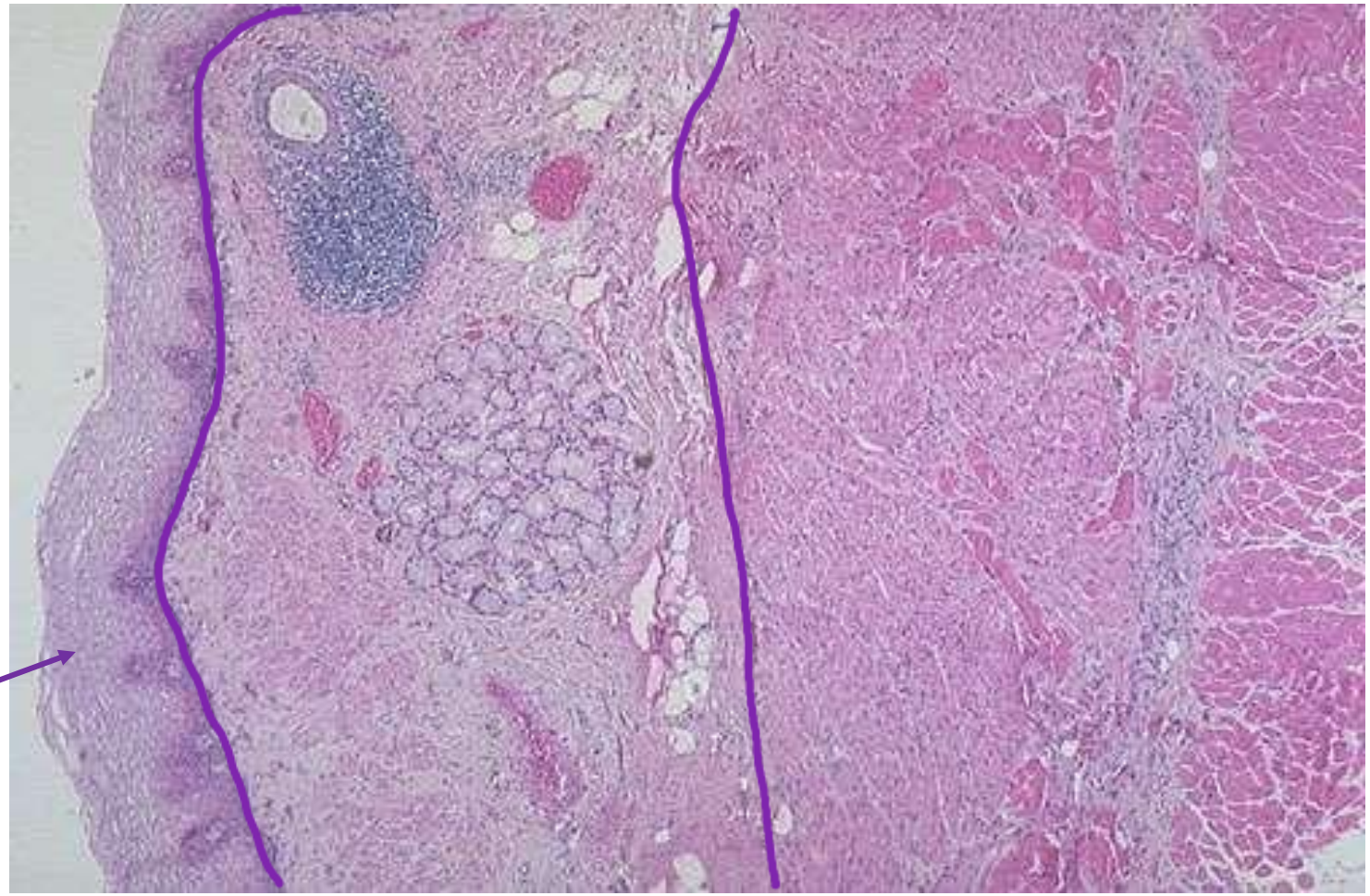
Lined by stratified squamous epithelium.

GEJ: gastroesophageal junction
Junction = Sphincter



Normal esophageal mucosa which has Tan to Pale Pink color.

In contrast with the **normal** gastric mucosa which has a light brown color.


Stratified squamous epithelium lining
Submucosal layer
Muscularis propria

Diseases that affect the esophagus

1. **Obstruction: mechanical or functional.**
2. **Vascular diseases: varices.**
3. **Inflammation: esophagitis.**
4. **Tumors.**

-1Mechanical Obstruction

- Congenital or acquired.
- Examples:
  - Atresia
  - Fistulas
  - Duplications
  - Agenesis (very rare)
  - Stenosis.

usually congenital

Atresia, Fistula, Duplication usually they present shortly after birth & they are non-compatible with eating and drinking or swallowing food.

Most of the cases are acquired

Agenesis means that the esophagus is not developed at all.

Atresia

- Thin, non-canalized cord replaces a segment of esophagus.

  This will interfere with the swallowing process and cause mechanical obstruction.

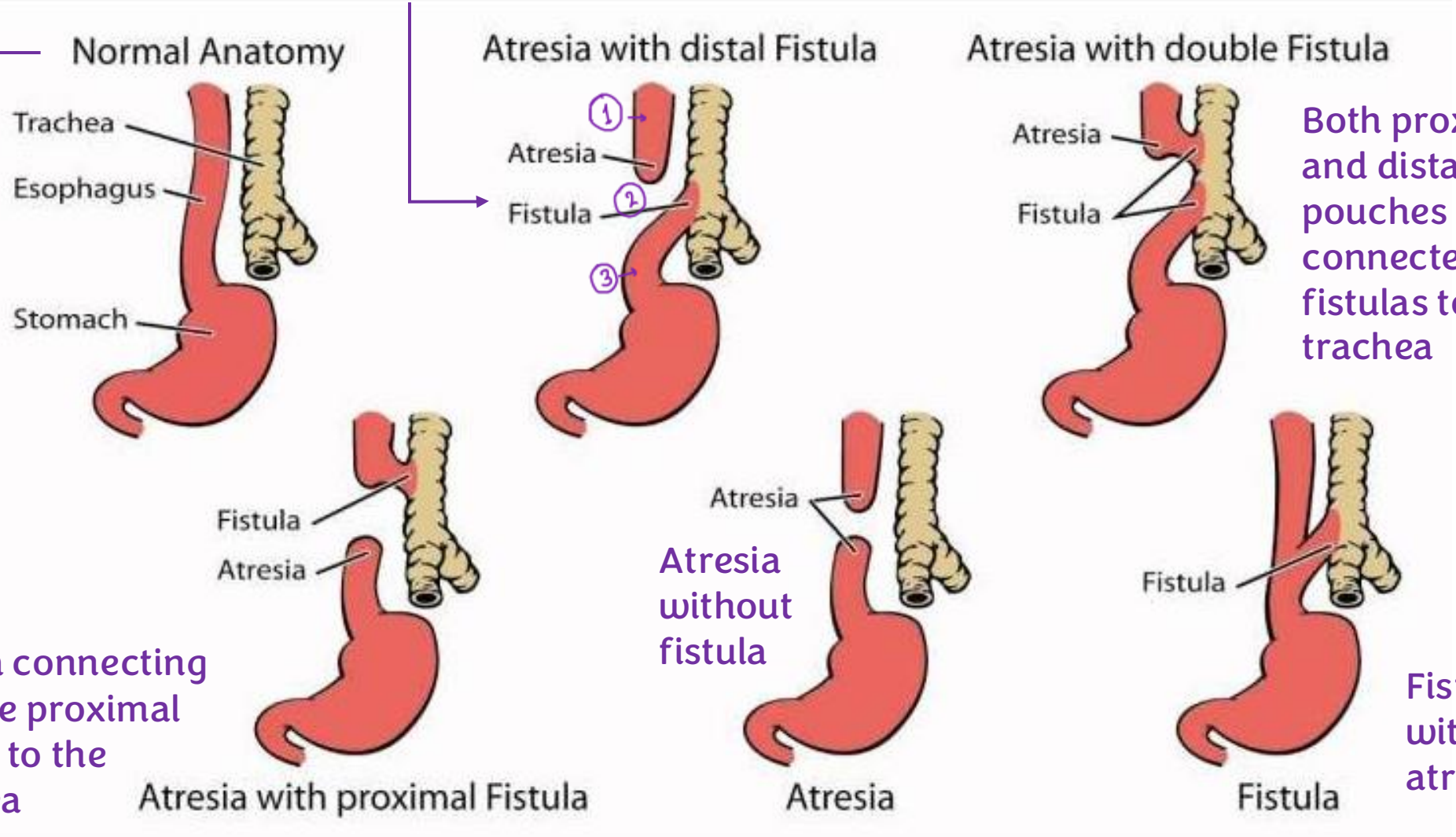
- Most common location: at or near the tracheal bifurcation.

- +- fistula (upper or lower esophageal pouches to a bronchus or trachea). This association connects the upper part or the lower part of the esophagus with bronchus or trachea, with consequent risk of aspiration or aspiration pneumonia.

Aspiration is when something you swallow enters your airway instead of the esophagus.

1-Proximal pouch.
2-the white color here means non-canalized.
3-Distal pouch (connected by fistula to the trachea).

Full esophagus separated from the trachea and the bifurcation of the trachea into two main-bronchi



Both proximal and distal pouches are connected by fistulas to the trachea

Fistula connecting just the proximal pouch to the trachea

Atresia without fistula

Fistula without atresia

Clinical presentation:

- Shortly after birth: regurgitation during feeding

  This is non-compatible with life.

- Needs prompt surgical correction (rejoin).

  in order for the baby to be able to eat and swallow.

  with

- **Complications if w/ fistula:**
- Aspiration
- Suffocation
- Pneumonia
- Severe fluid and electrolyte imbalances.

  Because of the inability to eat, and nutritional problems.

Esophageal stenosis

Most of the cases

- Acquired>>>Congenital.
- Characterized by: Fibrous thickening of the submucosa & atrophy of the muscularis propria.

This will cause impedance of food flow through the esophagus.

- Due to inflammation and scarring

- **Causes:**
- Chronic GERD.
- Systemic sclerosis.
- Irradiation
- Ingestion of caustic agents

GERD can be associated with ulcerations that are repaired by fibrosis leading to **stenosis** and narrowing of the esophagus.

Systemic sclerosis is due to fibrosis of the submucosa.

المواد الحارقة

Acids & Alkaline: they can cause chemical esophagitis which can be complicated later on by fibrosis and **stenosis**.

GERD: Gastroesophageal Reflux Disease

Clinical presentation

- Progressive dysphagia.
- Difficulty eating solids that progresses to problems with liquids.

2-Functional Obstruction

: when you don't see something that interferes with the passage of food, but there is an abnormality in the innervation.

Efficient delivery of food and fluids to the stomach requires coordinated waves of peristaltic contractions.

Esophageal dysmotility: discoordinated peristalsis or spasm of the muscularis.

Achalasia: the most important cause.

And this will lead to inefficient delivery of food through the esophagus to the stomach

Achalasia

- **Triad:**
- Incomplete LES relaxation
- Increased LES tone

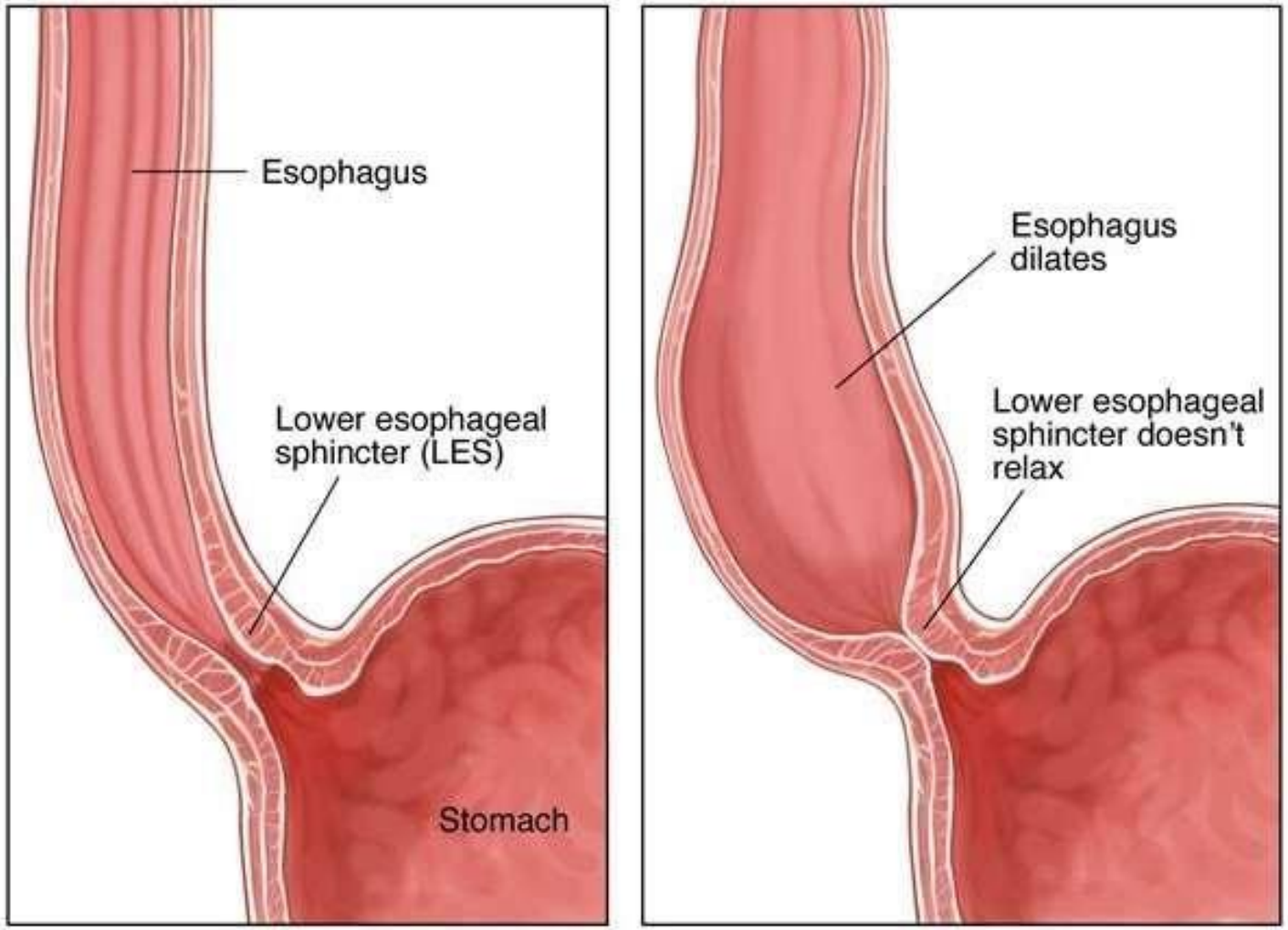
—> no complete relaxation —> the sphincter will be semi-closed.

- Esophageal aperistalsis. = No peristaltic movement.
- Primary >>>secondary.

more common

LES: lower esophageal sphincter

Typical features of Achalasia



Normal

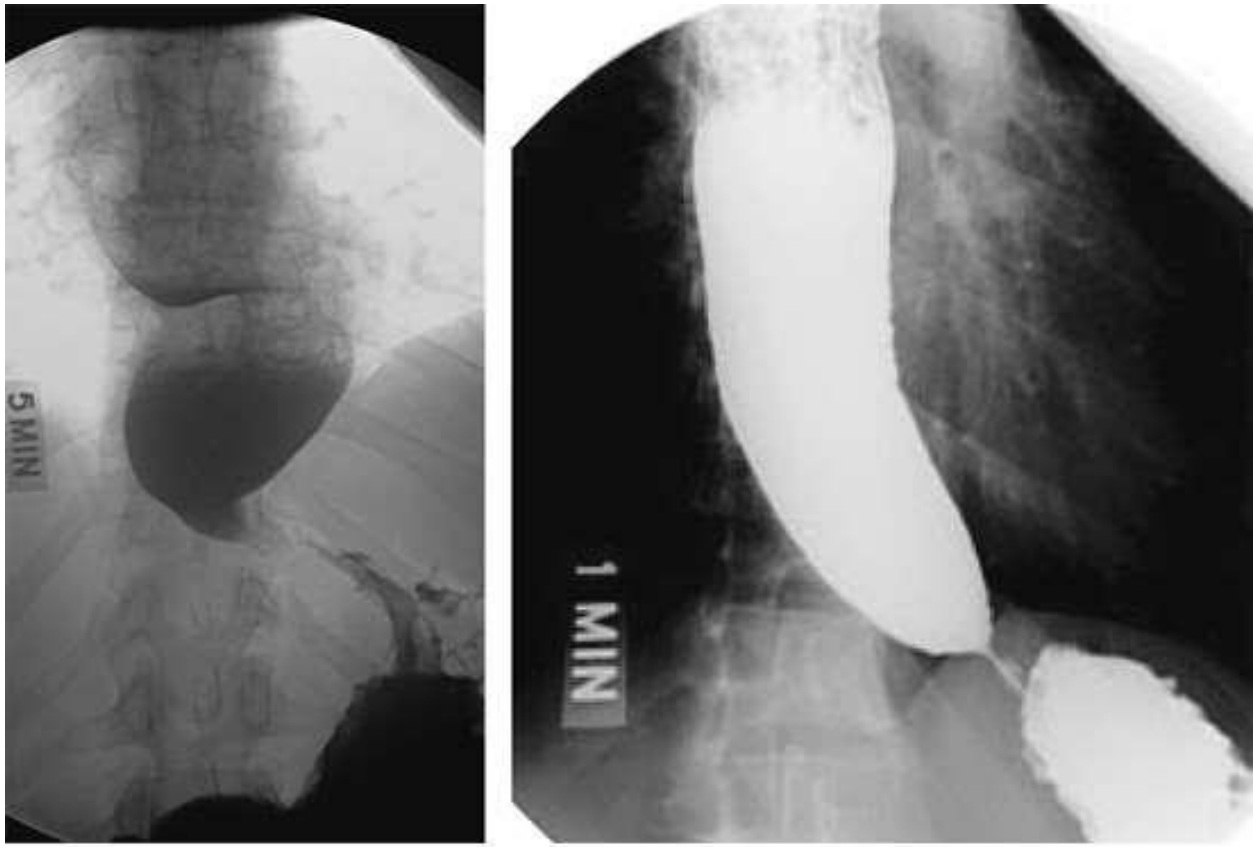
Achalasia

-Increased tone of LES and incomplete relaxation: Sphincter will be semi-closed (won't be open & won't be fully relaxed upon arrival of food) causing accumulation of food in the esophagus & the esophagus will dilate.

-The sphincter is semi-closed, while esophagus is dilated (due to accumulation of food).

gastro.com.cy

Barium swallow test:
We ask the patient to drink barium then we take X-ray images.



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

We can see that:
-the barium here will build up in the esophagus which is dilated.
-LES appears as a string as it is semi-closed, with a passage of very small amount of food to the stomach.

Primary achalasia

-These neurons are responsible for the inhibition of the contraction leading to dilatation or relaxation of muscles.

-The degeneration of these neurons leads to increase tone of LES.

Secondary achalasia

Less common

- Loss of neural innervation due to damage in:

- **Esophagus.**
- **Vagus nerve** (Which innervates the esophagus)
- **Dorsal motor nucleus of vagus**

- **Chagas disease**, Trypanosoma cruzi infection>>destruction of the myenteric plexus>> failure of LES relaxation>> esophageal dilatation.

Myentric plexus is plexus of the gut which is responsible for the peristaltic movement.

Clinical presentation

- Difficulty in swallowing
- Regurgitation
- Sometimes chest pain. Due to aspiration

3-Vascular diseases:

Esophageal Varices

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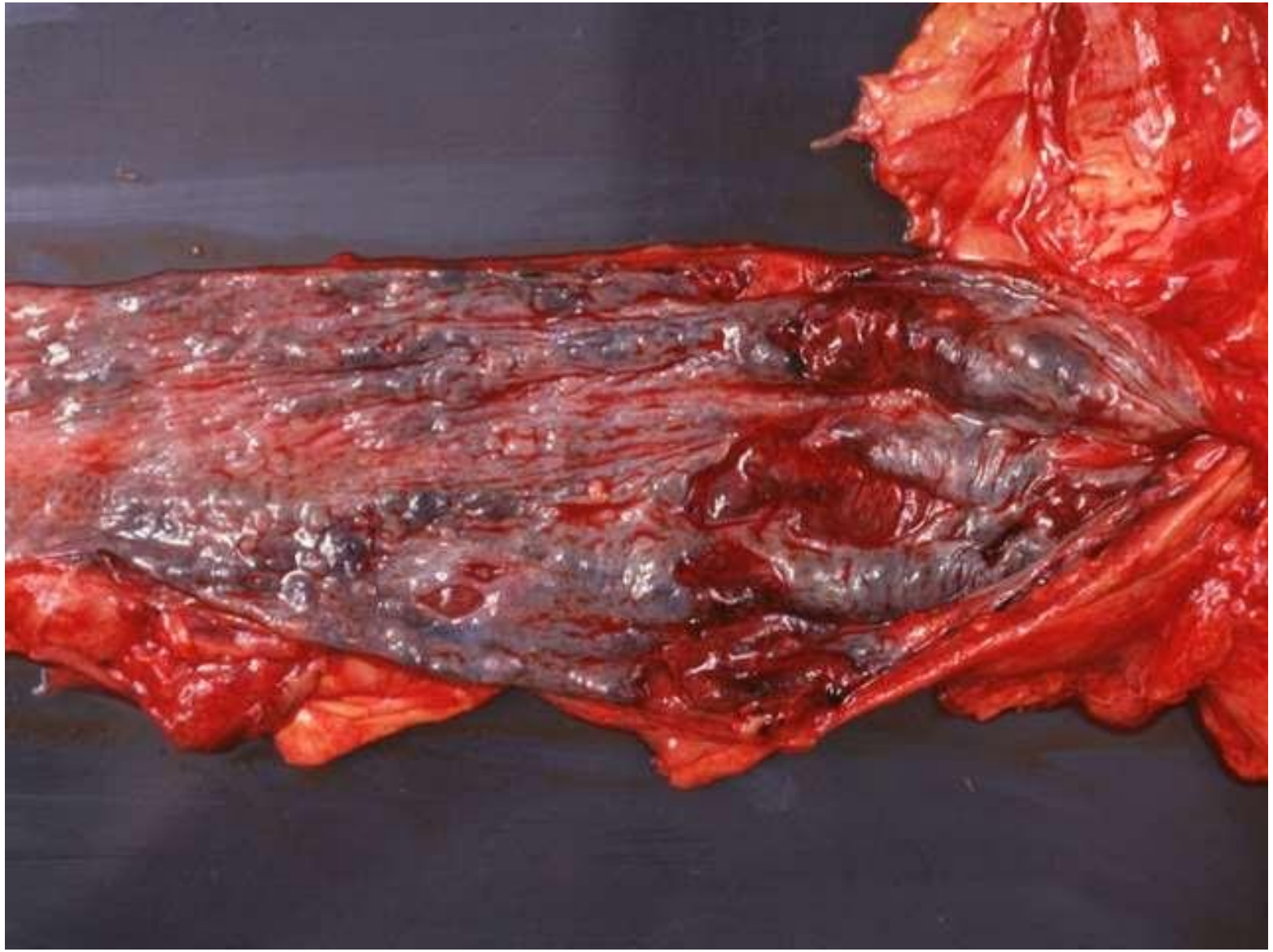
It is the most important vascular disease of the esophagus.

- Tortuous dilated veins within the submucosa of the distal esophagus and proximal stomach.
- Diagnosis by endoscopy or angiography.



These are dilated submucosal veins (the blackish vessels) engorged with blood in the distal esophagus.

Medpics - UCSD School of Medicine

Pathogenesis:

Usually due to portal hypertension

- **Portal circulation**: blood from GIT>>portal vein>>liver (detoxification)>>(via hepatic vein) inferior vena cava.
- Diseases that impede portal blood flow >> portal hypertension >>esophageal varices.
- Distal esophagus : site of Porto-systemic anastomosis.
- **Portal hypertension**>>collateral channels in distal esophagus>>shunt of blood from portal to systemic circulation>>dilated collaterals in distal esophagus>>varices

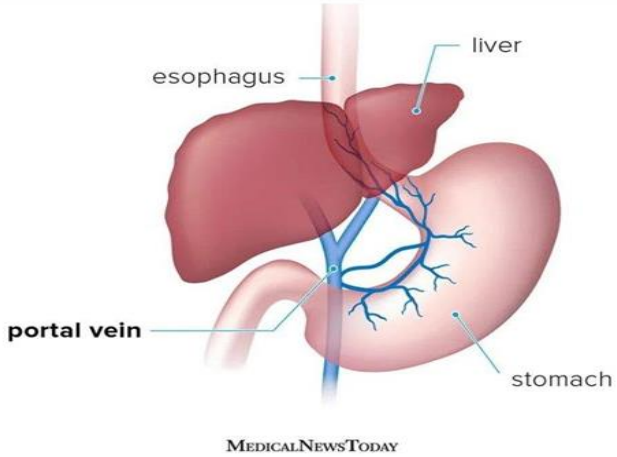
Additional figure

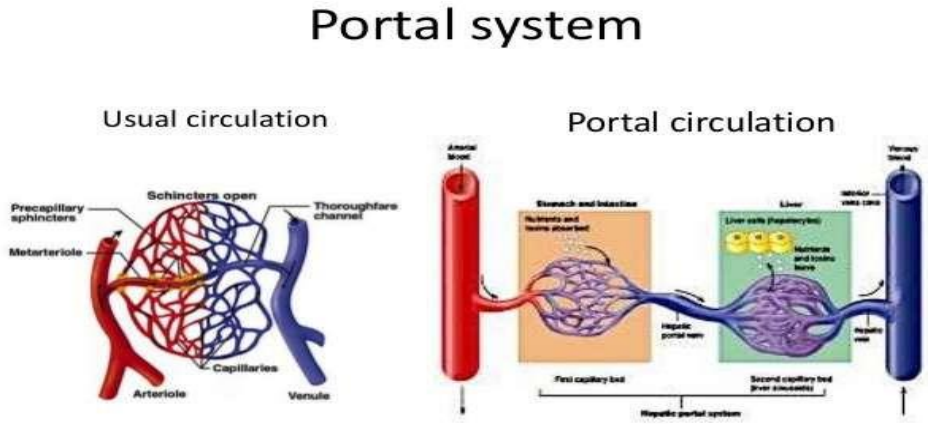
-GI system is characterized by the presence of portal circulation.

-What is a Portal Circulation?

It's a process in which the blood that is collected from GI tract will not go directly to the inferior vena cava, instead it will go through the portal vein to the liver (for detoxification), then through the hepatic vein into inferior vena cava.

-Any disease that impede this portal blood flow will lead to portal hypertension, leading to shunt of blood from the portal circulation to the systemic circulation through the area in which we have a collateral anastomosis between these two circulation, and one of these sites is **distal esophagus proximal stomach** leading to **Esophageal Varices.**



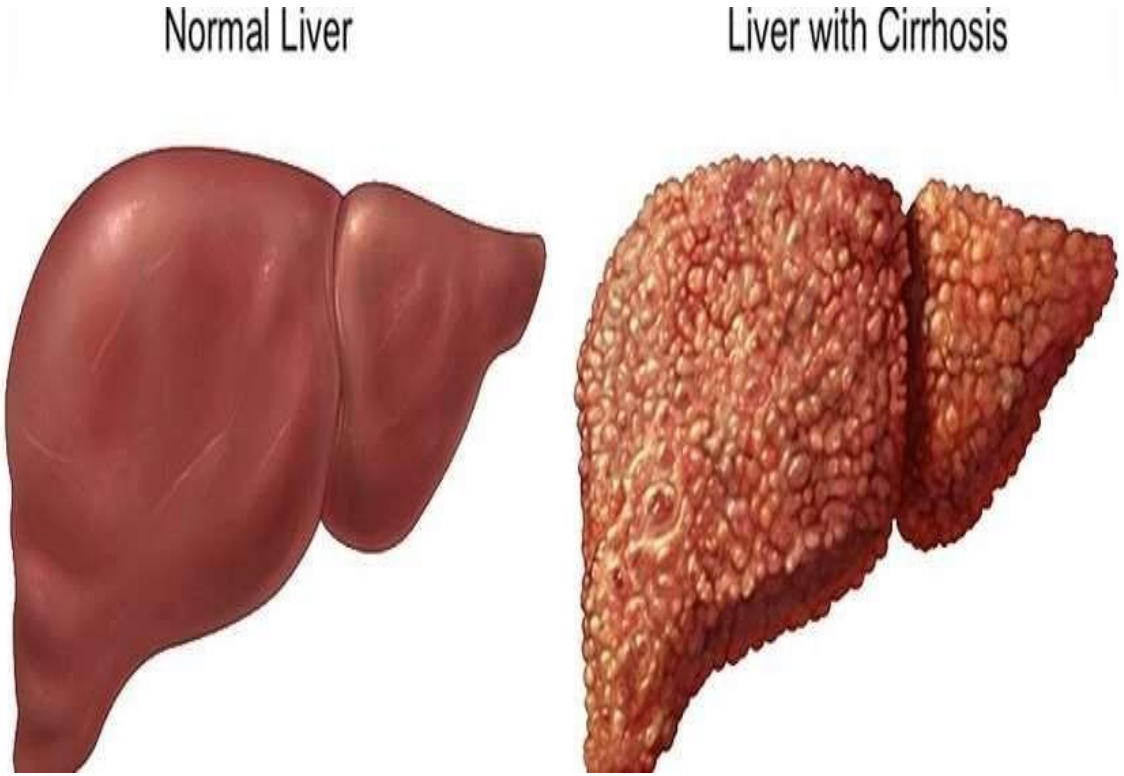


Causes of portal hypertension

- Cirrhosis is most common

  Alcoholic liver disease.

- Hepatic schistosomiasis 2 most common worldwide.



Cirrhosis → liver will transform into nodular liver → portal Hypertension & Chronic liver disease

Clinical Features

- Often asymptomatic.
- Rupture leads to massive hematemesis and death.
- 20% of patients die from the first bleed despite interventions.
- Death due to hemorrhage, hepatic come, and hypovolemic shock
- Rebleeding in 60%.

Discovered incidentally during endoscopy in patients with cirrhosis because they should undergo periodic surveillance for the development of varices .

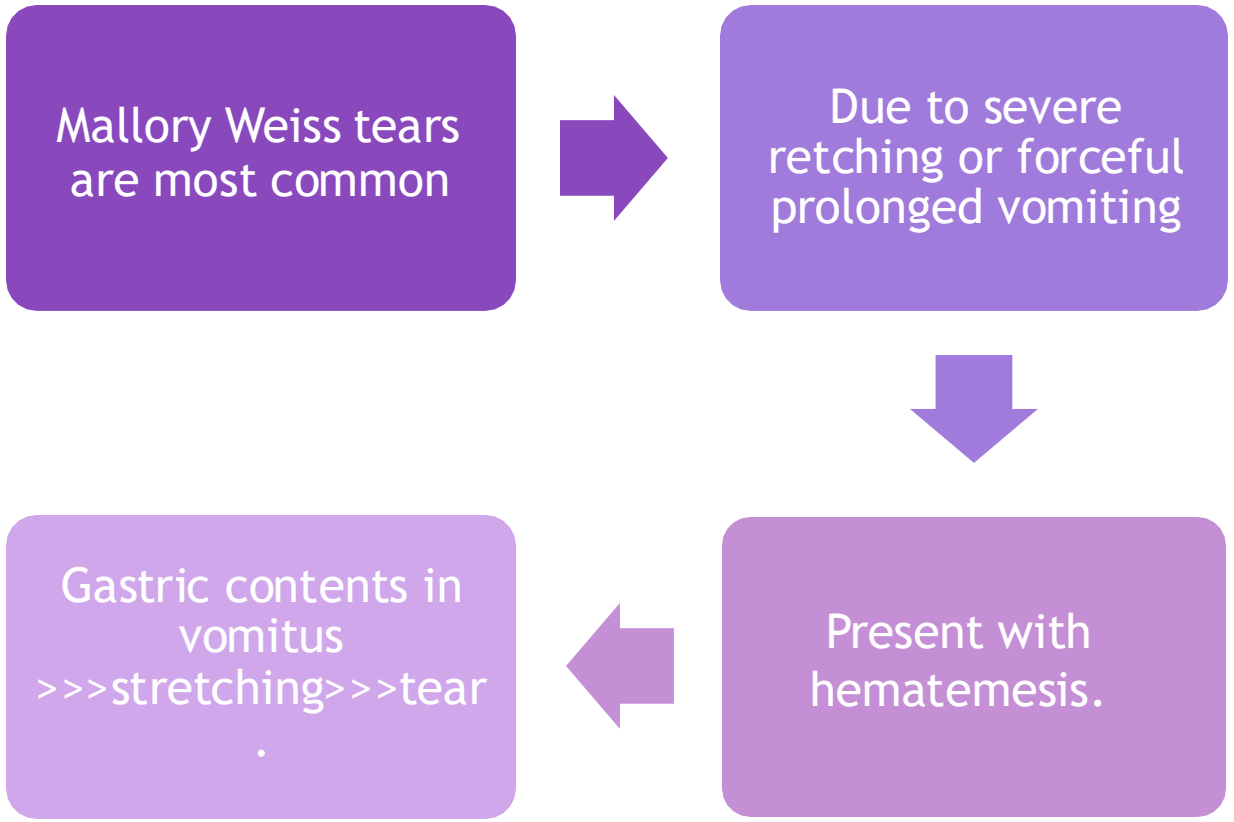
Hematemesis= Vomiting of blood

4-ESOPHAGITIS

Inflammation of esophageal Caused by:

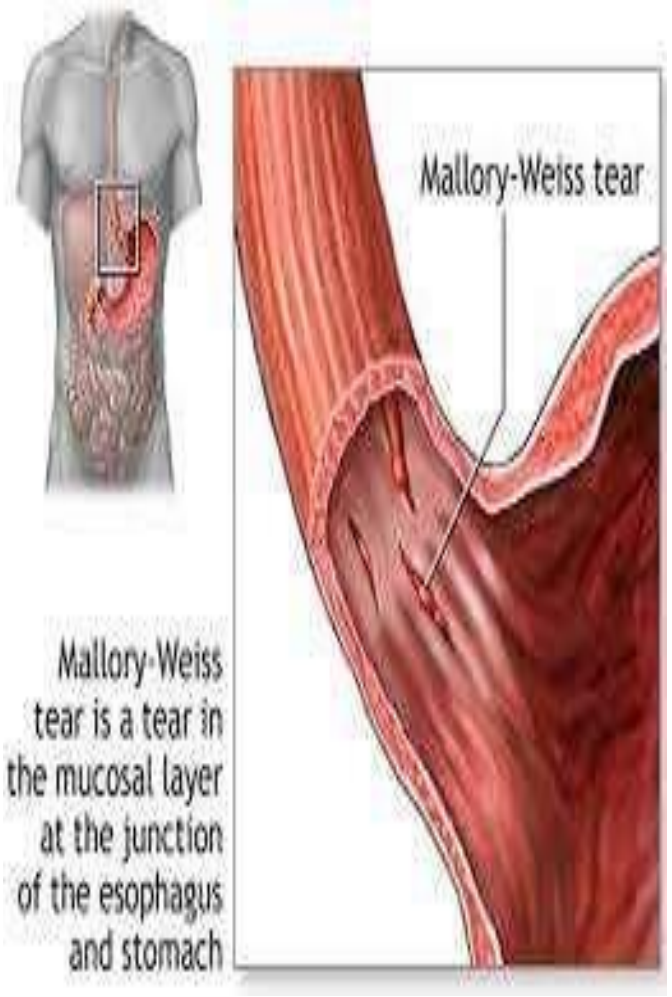
- Esophageal Lacerations.
- Mucosal Injury
- Infections
- Reflux Esophagitis
- Eosinophilic Esophagitis

Esophageal Lacerations



- -During vomiting which is severe & prolonged in this case, there will be no time for esophagus to relax. Consequently, a large amount of gastric content will pass through esophagus causing distension& stretching of esophagus. This lead to a tear presented with hematemesis (Fresh red colored blood)

- Linear lacerations
- longitudinally oriented
- Cross the GEJ.
- Superficial Only on mucosa
- Heal quickly , no surgical intervention



The patient is presented with fresh red colored blood after forceful vomiting -> esophagitis
The physician should reassure the patient that the tears will heal spontaneously

Chemical Esophagitis

- **Damage to esophageal mucosa by irritants**
- Alcohol,
- Corrosive acids or alkalis
- Excessively hot fluids
- Heavy smoking
- Medicinal pills (doxycycline and bisphosphonates)
- Iatragenic (chemotx, radiotx , GVHD)

GVHD: Graft Versus Host Disease

- Biphosphonates are a major cause of medicine pill esophagitis due to large size of tablets that could be stuck in esophagus-Solution: We ask the patient to drink plenty of water & stay in an upright position for a while

Clinical symptoms & morphology

- Ulceration and acute inflammation.
- Only self-limited pain, odynophagia (pain with swallowing).
- Hemorrhage, stricture, or perforation in severe cases Stricture can lead to stenosis

Infectious esophagitis

- Mostly in debilitated or immunosuppressed.

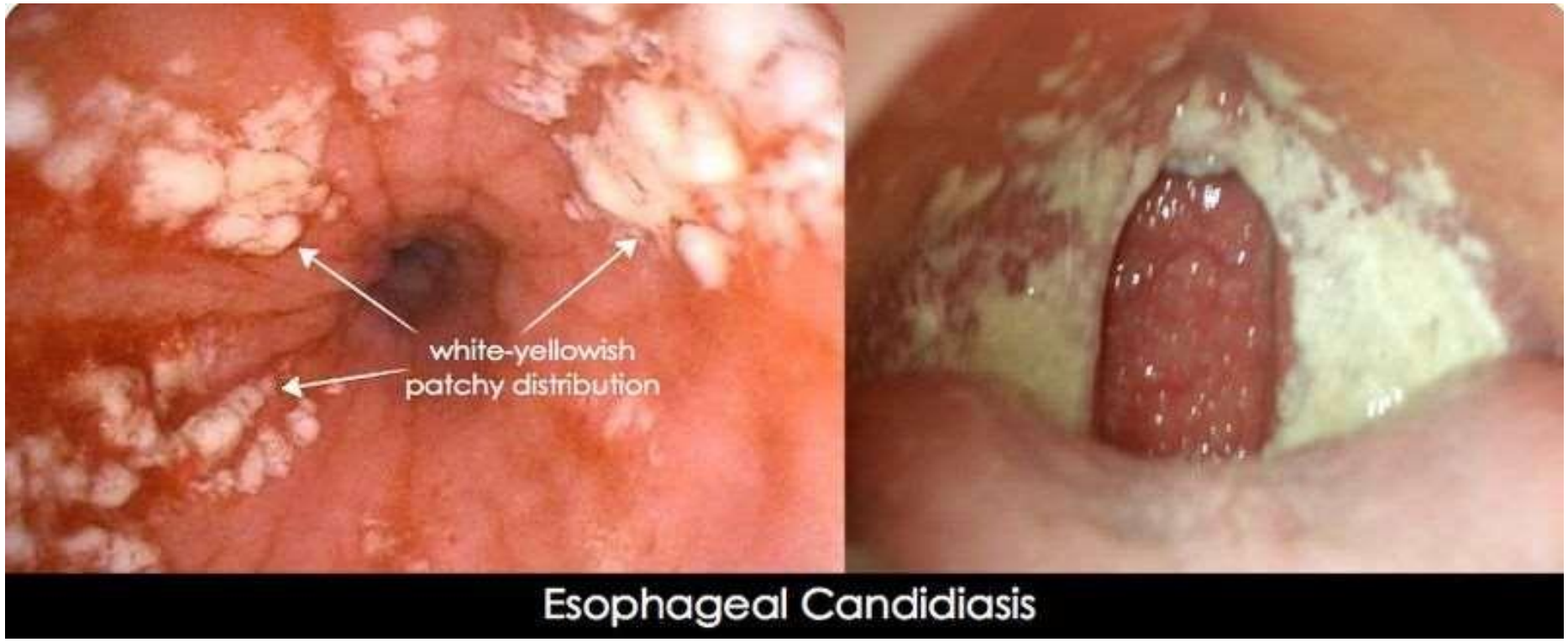
- Viral (HSV, CMV)
- Fungal (candida >>> mucormycosis & aspergillosis)
- Bacterial: 10%. Bacteria is less common, and can be secondary to viral or fungal infection

- Candidiasis :

- Adherent.
- -Adherent to the esophageal mucosa and seen during endoscopy
- Gray-white pseudo membranes
- Composed of matted fungal hyphae and inflammatory cells
- They can be seen microscopically upon biopsy examination-
- This infection can extend to oral mucosa causing oral thrush

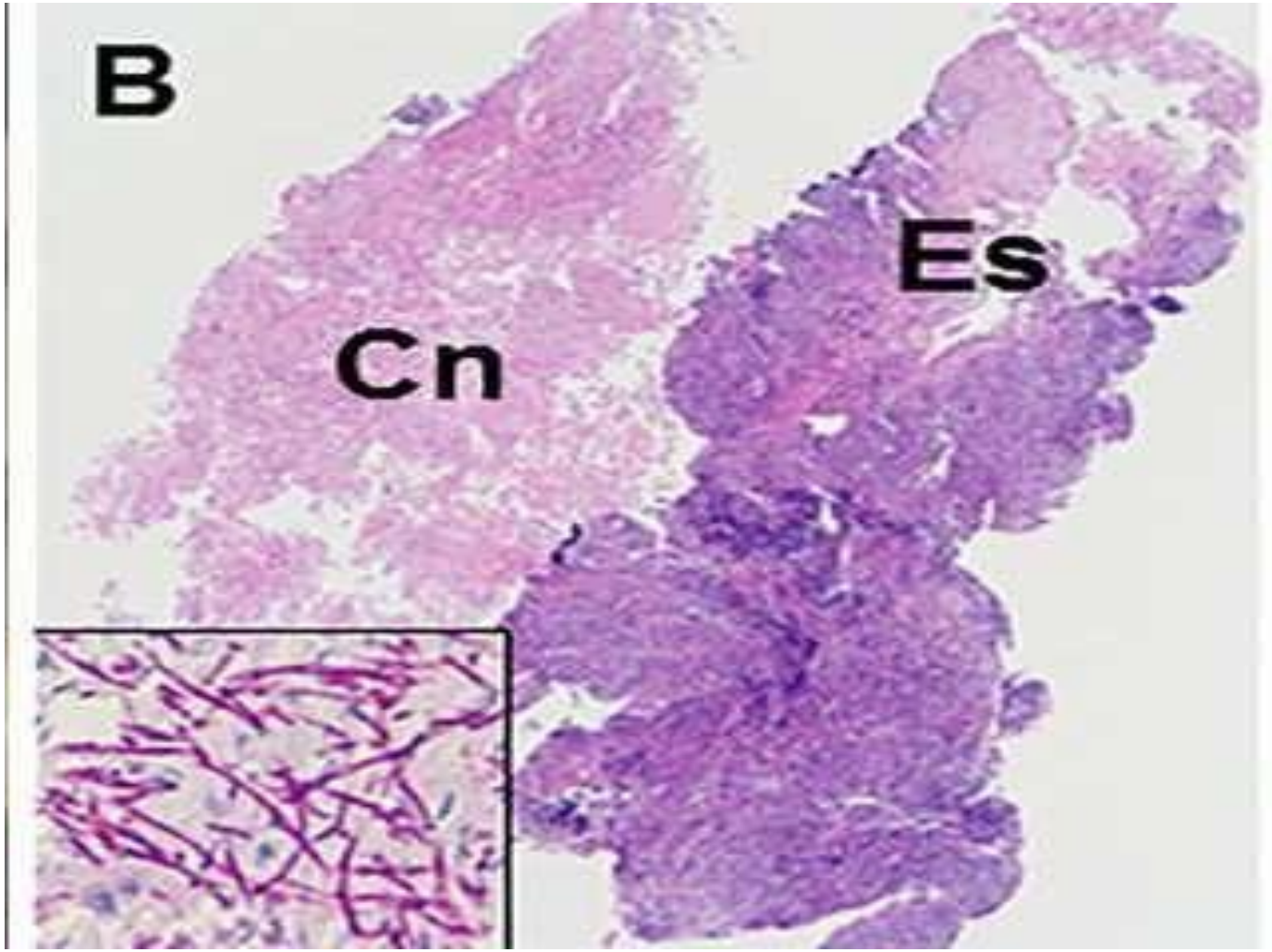
Esophagus

-Oral mucosa with an oral thrush



https://www.pinterest.com/pin/374291419013418659/

Hyphae-



We use periodic acid schiff stain to highlight fungal hyphae

www.researchgate.net/publication/285369734_Esophageal_Candidiasis_as_the_Initial_Manifestation_of_Acute_Myeloid_Leukemia

- Herpes viruses
- Punched-out ulcers Can be seen by endoscopy

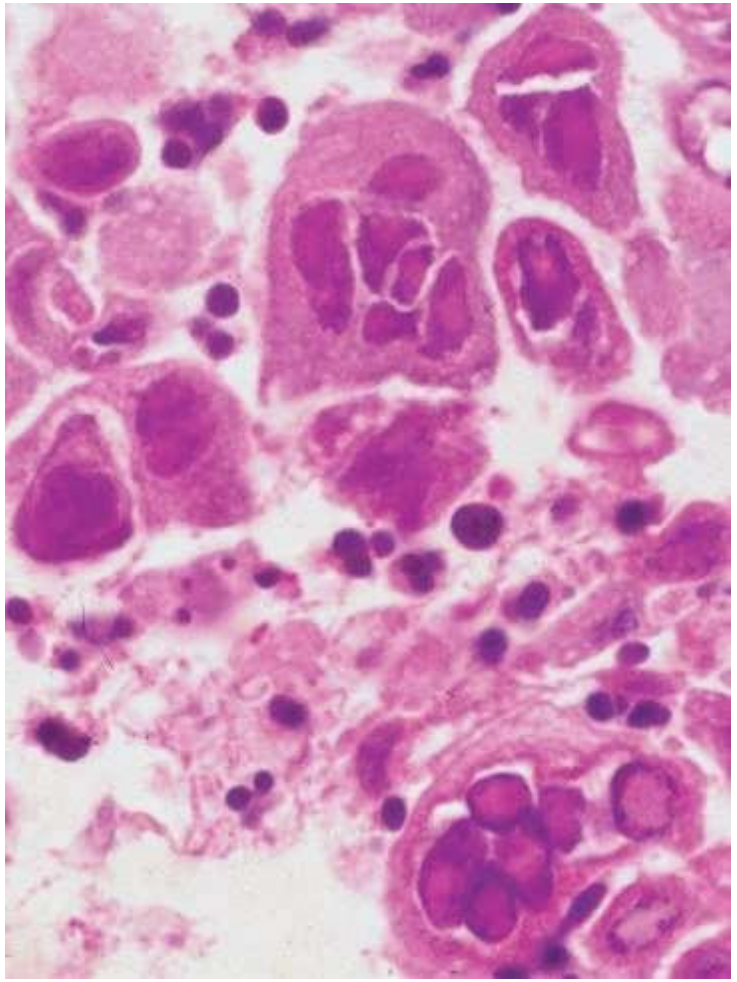
- Histopathologic:
- Nuclear viral inclusions
- Degenerating epithelial cells ulcer edge
- Multinucleated epithelial cells.

Semantic Scholar

-Remember: The esophagusmucosa is normally pale-pink, but here we see the surrounding mucosa erythematous (red)

Here we can see punched-out ulcers

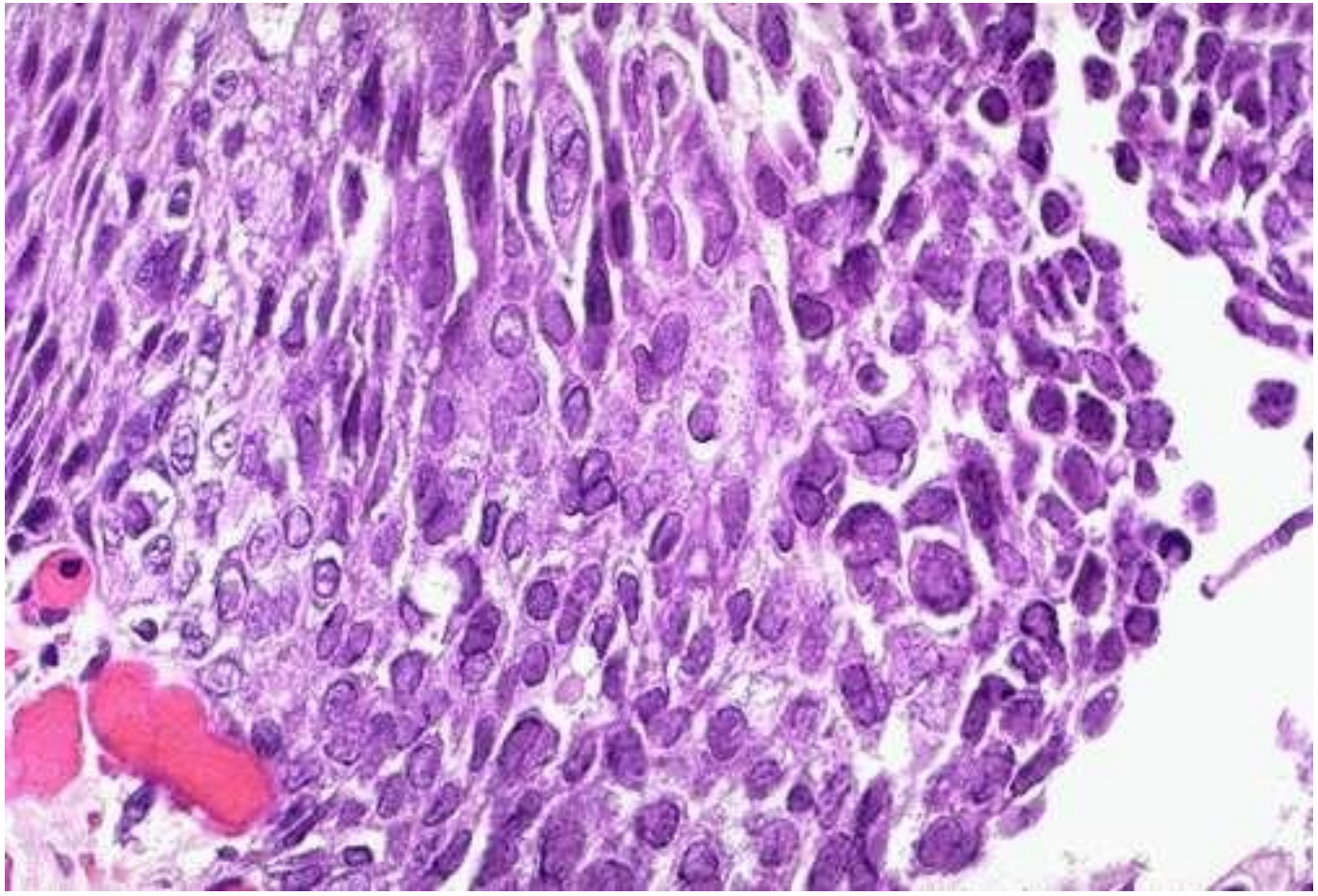
- HSV infection histology: Multinucleated cells, Viral nuclear inclusion, Degeneration of cells



10th edition

Robbins Basic Pathology

- Histology:-Multinucleated cells-The nuclei are characterized by intranuclear inclusion (A typical feature of HSV biopsies)



- CMV :
- Shallower ulcerations.
- Biopsy: nuclear and cytoplasmic inclusions in capillary endothelium and stromal cells.(Mega cells) Cytomegaly=Mega cells=Large cells

CMV infects Endothelial & Stromal Cells in addition to epithelial cells, unlike HSV which only infects epithelial cells

-Large Stromalcells with nuclear inclusion

Large Endothelial cell

Robbins Basic Pathology 10th edition

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			

Additional Resources:

رسالة من الفريق العلمي:

﴿وَاصْبِرْ لِحُكْمِ رَبِّكَ فَإِنَّكَ بِأَعْيُنِنَا﴾