

Diseases of the esophagus-1

Manar Hajeer, MD, FRCPath

University of Jordan, School of medicine

Anatomy and histology:

Muscular tube
extending from
the epiglottis
to the GEJ.

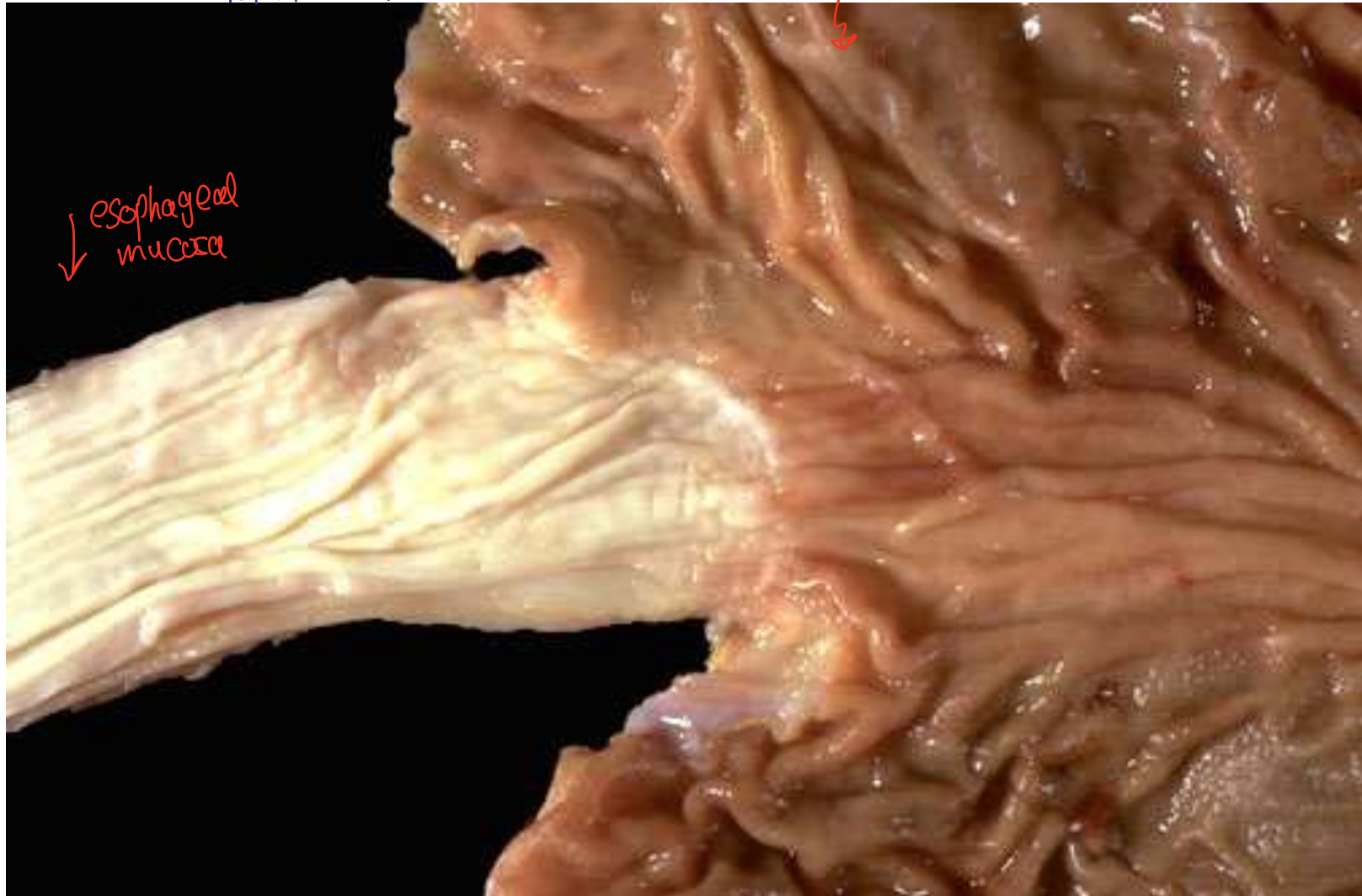
↓
Gastro Esophageal
sphincter
inferiorly

Lined by
stratified
squamous
epithelium.

normally

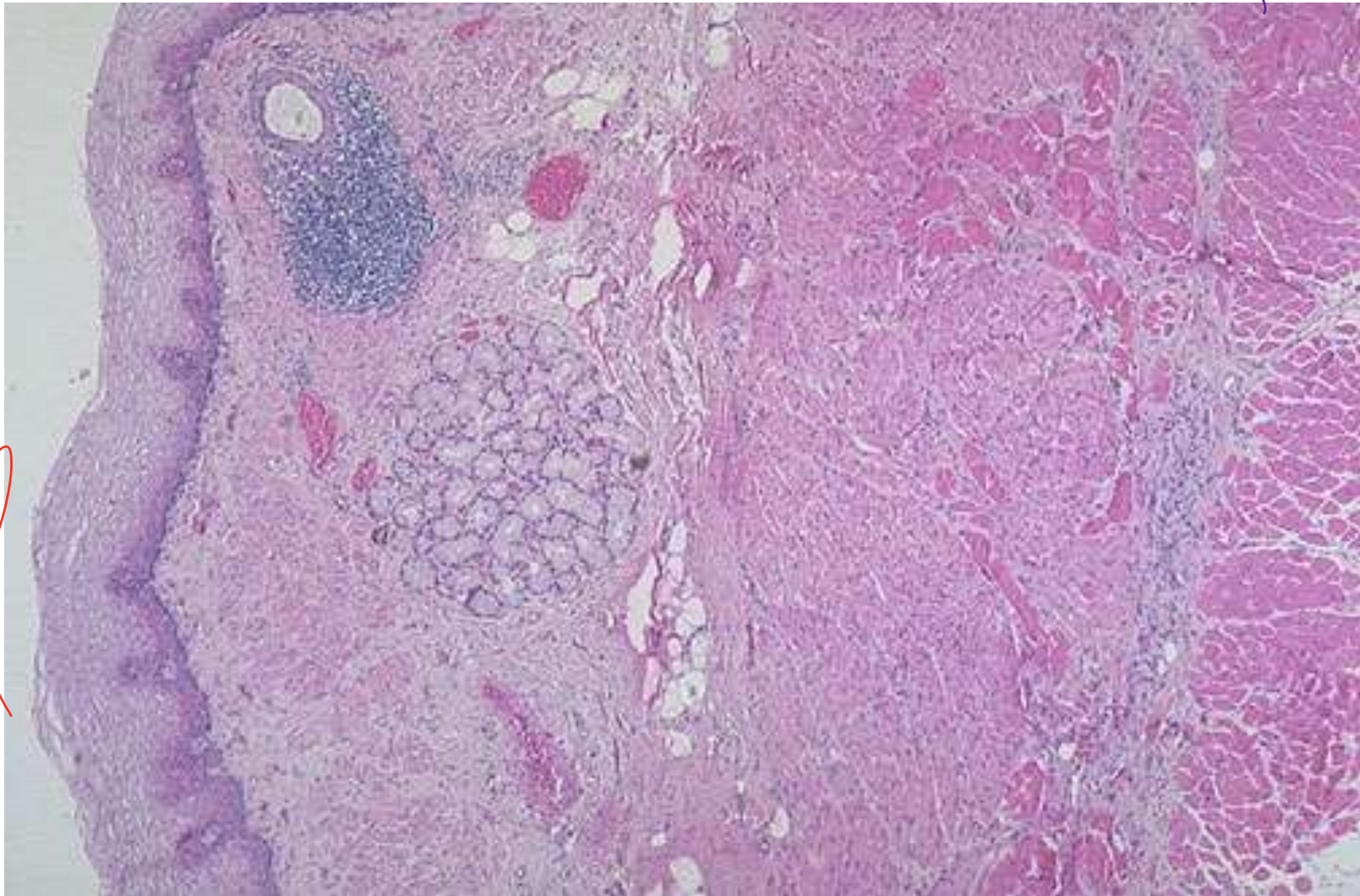
normal esophageal mucosa
which have tan-colored
to pale-pink

normal gastric mucosa
which has light-brown
color



Submucosal

Muscularis Propria



Stratified sq epithelium (lining)
↳ part of mucosa

Diseases that affect the esophagus

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

1-Mechanical Obstruction

- ▶ Congenital or acquired.

- ▶ Examples:

- ▶ Atresia
- ▶ Fistulas
- ▶ Duplications
- ▶ Agenesis (v rare)
- ▶ Stenosis.

appear
shortly after birth
non-compatible with
eating or drinking

esophagus
is not developed
at all

Atresia

Aspiration: when food or liquid enter lung

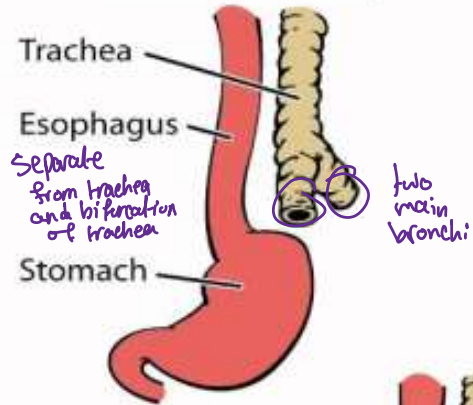
Aspiration pneumonia: infection following aspiration

Part of esophageos is and replaced by thin cord

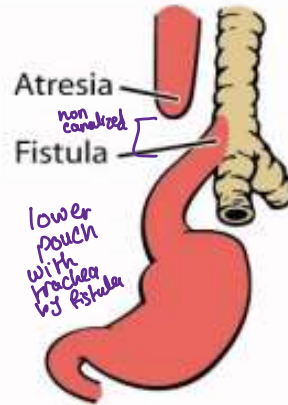
- ▶ Thin, **non-canalized** cord replaces a segment of esophagus.
- ▶ Most common location: at or near the tracheal bifurcation *connect*
- ▶ +/- fistula (upper or lower esophageal pouches to a bronchus or trachea). Risk of aspiration + pneumonia
aspiration

interfere with swallowing
Mechanical obstruction

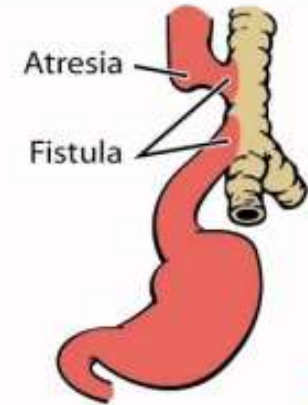
Normal Anatomy



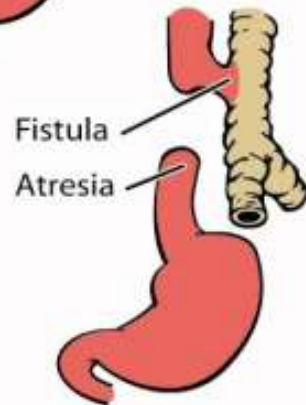
Atresia with distal Fistula



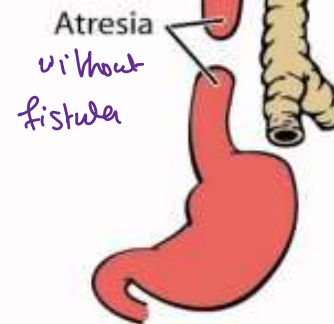
Atresia with double Fistula



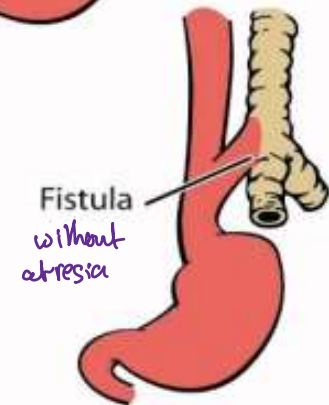
Proximal part to trachea



Atresia with proximal Fistula



Atresia



Fistula



Clinical presentation:

- ▶ Shortly after birth: regurgitation during feeding *non-compatible with life*
- ▶ Needs prompt surgical correction (rejoin).
to be able to eat and swallow
- ▶ **Complications if w/ fistula:**
- ▶ Aspiration
- ▶ Suffocation
- ▶ Pneumonia
- ▶ Severe fluid and electrolyte imbalances.
↳ Bcz inability to eat and nutritional problem

Esophageal stenosis

- ▶ Acquired >>> Congenital.

- ▶ Fibrous thickening of the submucosa & atrophy of the muscularis propria.

impede flow of food through esophagus

- ▶ Due to inflammation and scarring of previous injury

- ▶ Causes: Gastroesophageal Reflux Disease

- ▶ Chronic GERD. ulceration → repair by fibrosis → stenosis → narrowing of esophagus

- ▶ Systemic sclerosis. fibrosis of submucosa

- ▶ Irradiation

- ▶ Ingestion of caustic agents

acid or alkaline
chemical esophagitis → complicated by fibrosis and stenosis

Clinical presentation

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

2-Functional Obstruction

↳ You don't see something that interfere with the passage of food but there's problem in innervation

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

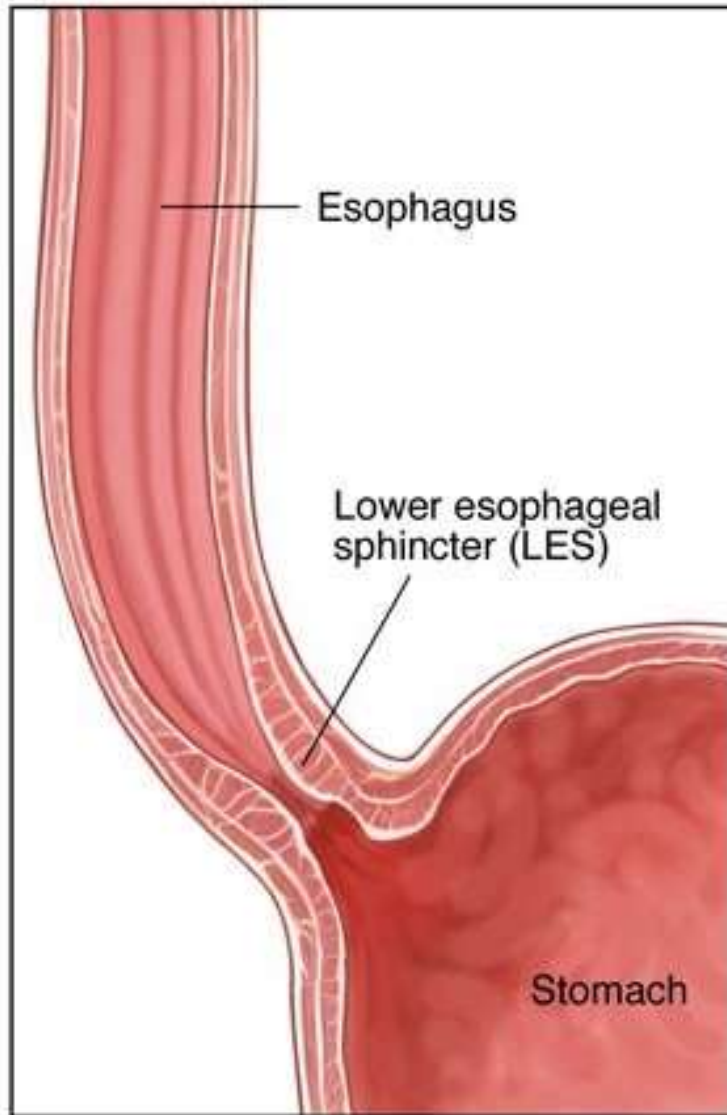
Esophageal dysmotility: dis-coordinated peristalsis or spasm of the muscularis.

propria of esophagus this will lead to inefficient delivery of food to stomach

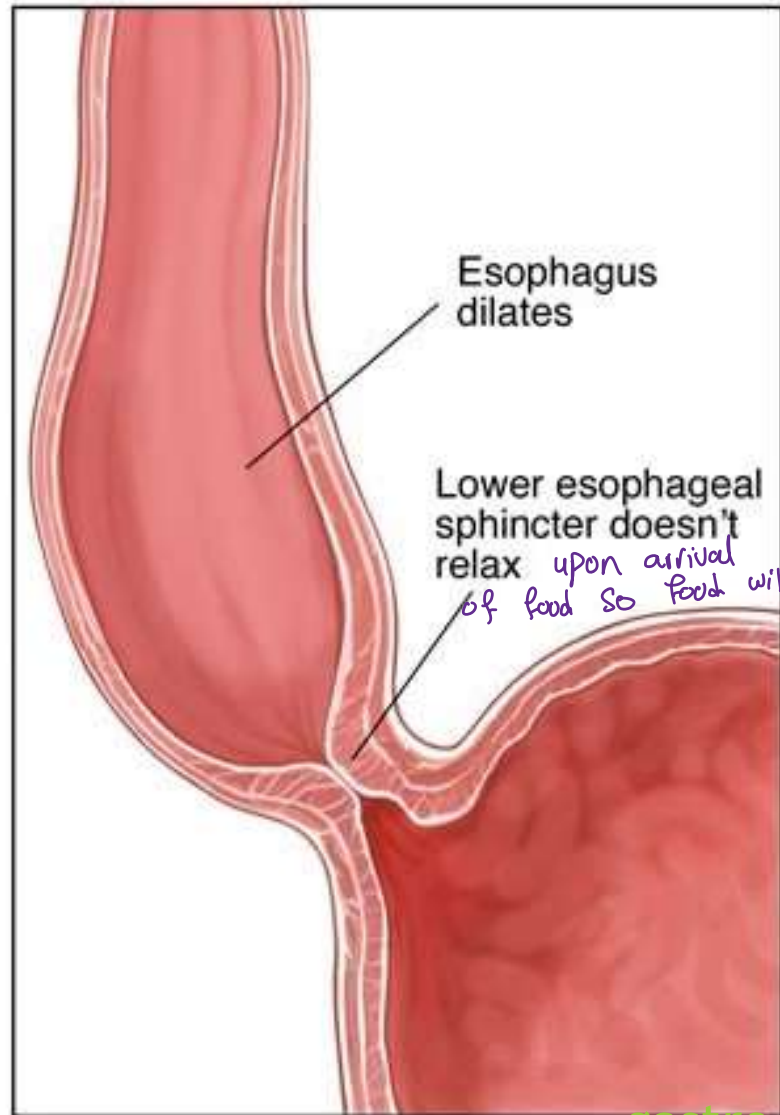
Achalasia: the most important cause.

Achalasia

- ▶ Triad: *Lower esophageal sphincter muscle tone ↑ [semi closed]*
- ▶ Incomplete LES relaxation
- ▶ Increased LES tone
- ▶ Esophageal aperistalsis. *no peristalsis*
- ▶ Primary >>>secondary.



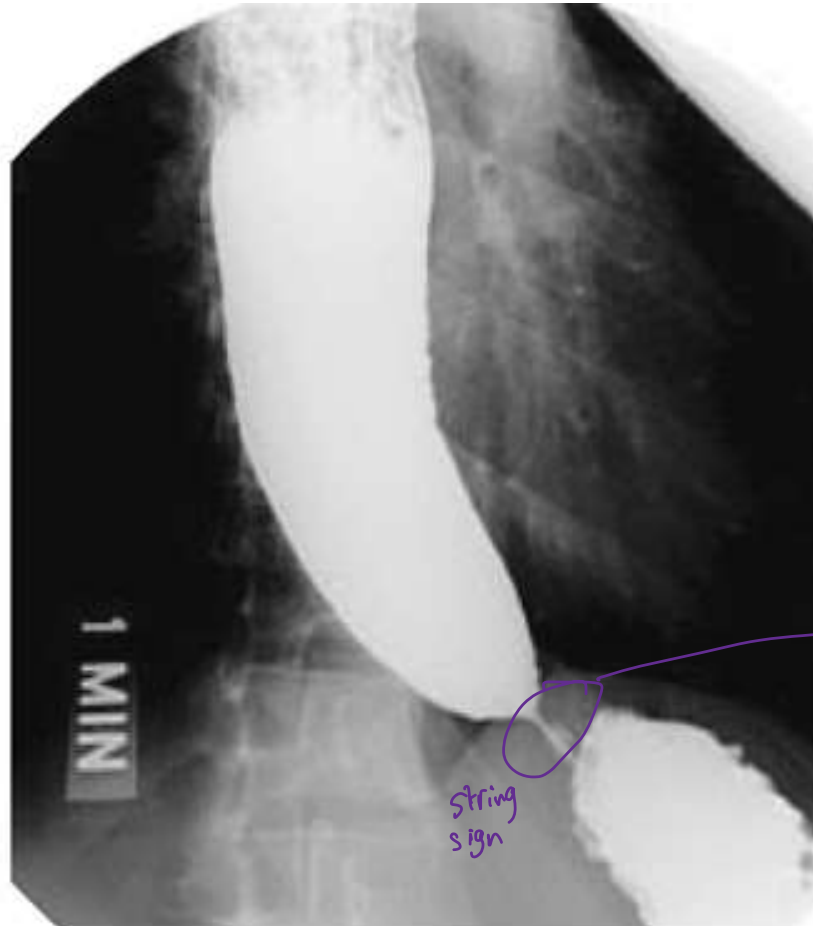
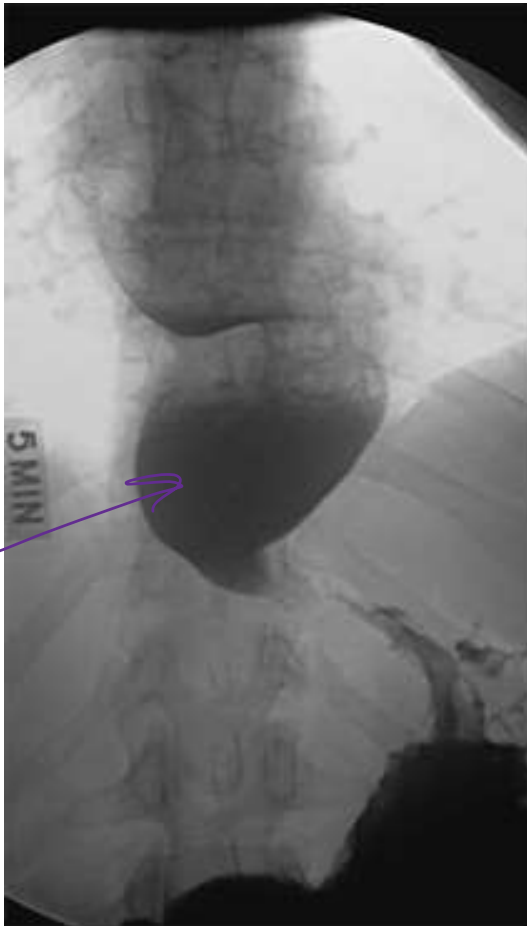
Normal



Achalasia

Barium Swallow

So
Barium
accumulates
in esophagus
and it will
dilate



string
sign

semi
closed
very few food
enter to stomach

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

Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

Primary achalasia

Degeneration of
distal esophageal
inhibitory
neurons.

Responsible for inhibition
of contraction lead to
relaxation of muscle


Idiopathic

Most common

overall this will lead to
increase muscle tone of LES



Secondary achalasia

- ▶ Loss of neural innervation due to damage in:
 - ▶ **Esophagus.**
 - ▶ **Vagus nerve** *that innervates esophagus*
 - ▶ **Dorsal motor nucleus of vagus**
 - ▶ **Chagas disease**, *Trypanosoma cruzi* infection>>destruction of the myenteric plexus>> failure of LES relaxation>> esophageal dilatation.
- 



Clinical presentation

- ▶ Difficulty in swallowing
- ▶ Regurgitation
- ▶ Sometimes chest pain.
due to aspiration

3-Vascular diseases:

Esophageal Varices

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



Dilated submucosal vein
engorged with blood

Medpics - UCSD School of Medicine

engorged

GI system characterized
by the presence of
portal circulation

Pathogenesis:

- ▶ **Portal circulation:** blood from GIT >> portal vein >> liver (detoxification) >> inferior vena cava.
Hepatic vein
- ▶ 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

Collateral vein

Blocked portal
systemic

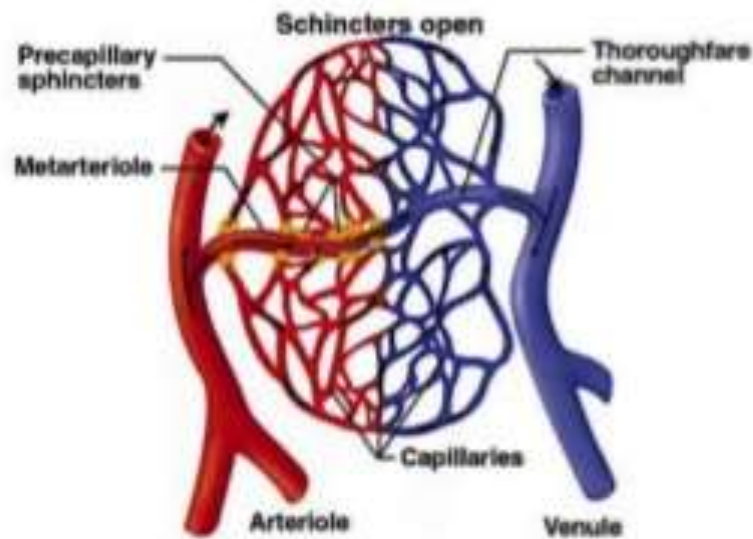


[distal esophagus
proximal stomach]

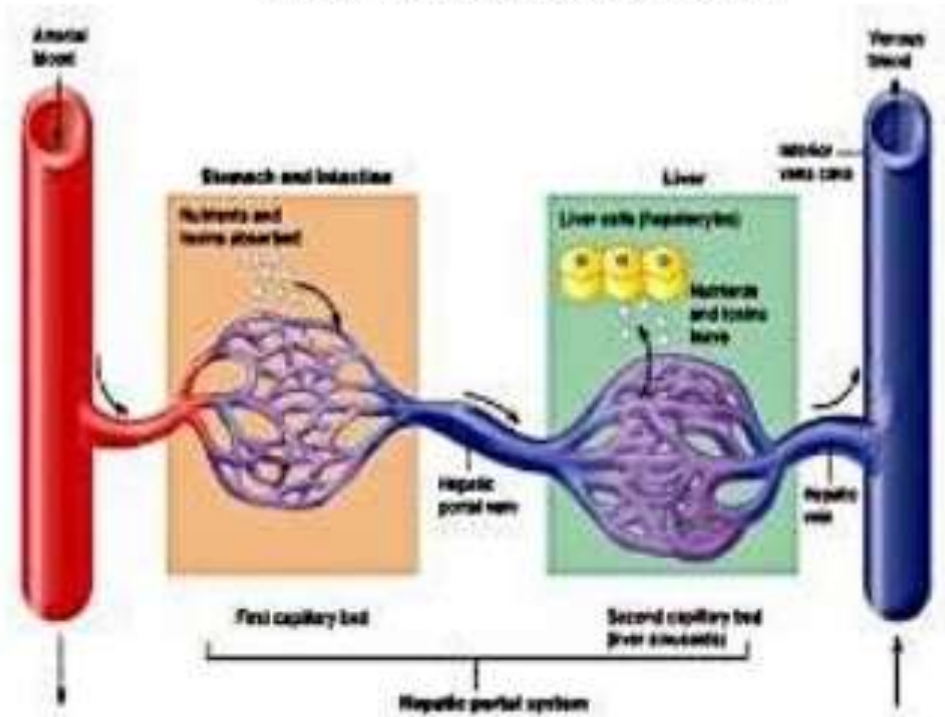
Through areas
which we have
collateral
anastomosis
between the
two circulations

Portal system

Usual circulation



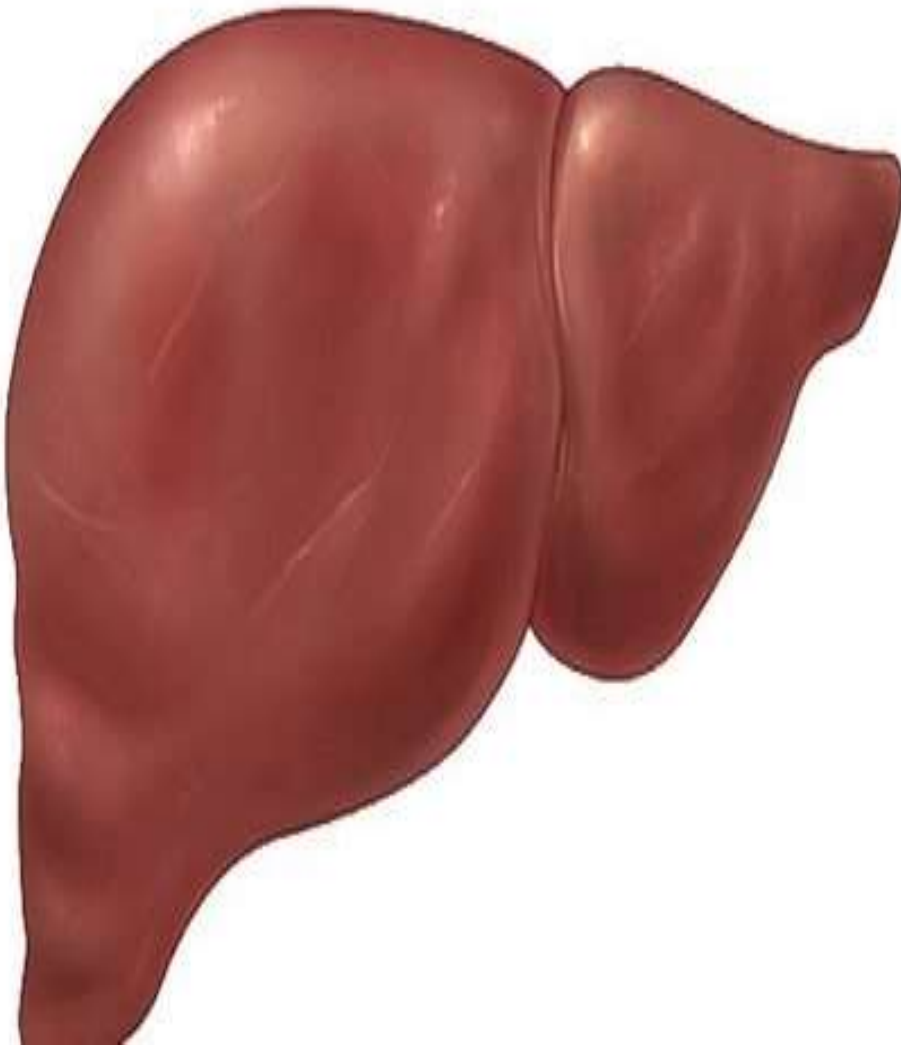
Portal circulation



Causes of portal hypertension

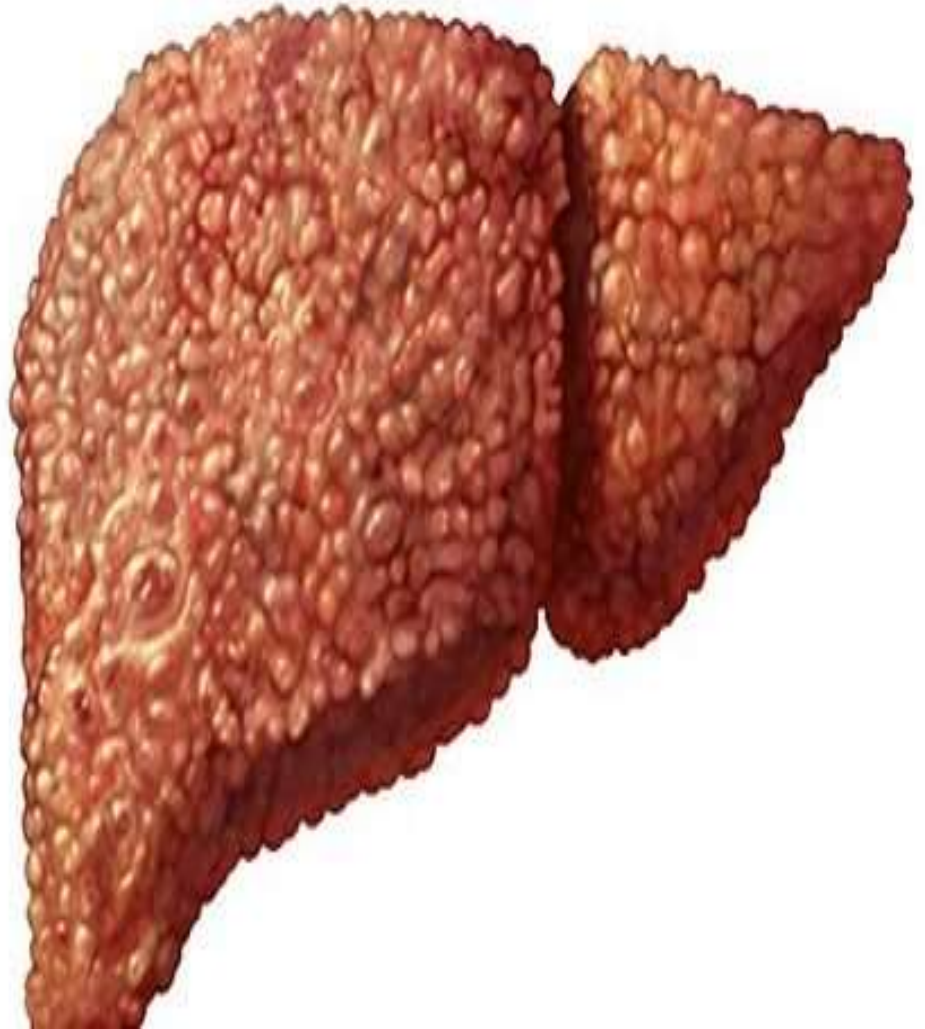
- ▶ Cirrhosis is most common
Alcoholic liver disease.
- ▶ Hepatic schistosomiasis 2nd most
common worldwide.

Normal Liver



Liver with Cirrhosis

nodular liver → lead to
portal hypertension
↳ Chronic liver failure



Clinical Features

Often asymptomatic.

incidental diagnosis
during endoscopy in patient with cirrhosis

BCZ they have periodic surveillance
for development of varices

of varices

Rupture leads to massive hematemesis
and death.

vomiting of blood

20% of patients die from the first
bleed despite interventions.

Death due to hemorrhage, hepatic
coma, and hypovolemic shock

Rebleeding in 60%.

who withstand
first bleeding

4-ESOPHAGITIS

inflammation

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

Esophageal Lacerations

retching: زفني عجز
vomiting
قيء
قيء (عز)

Lower esophagus

Mallory Weiss tears are most common

severe vomiting

Due to severe retching or forceful prolonged vomiting

There's no time for esophagus to Relax

Gastric contents in vomitus

>>>stretching>>>tear

Distention

Present with hematemesis.

Fresh colored blood

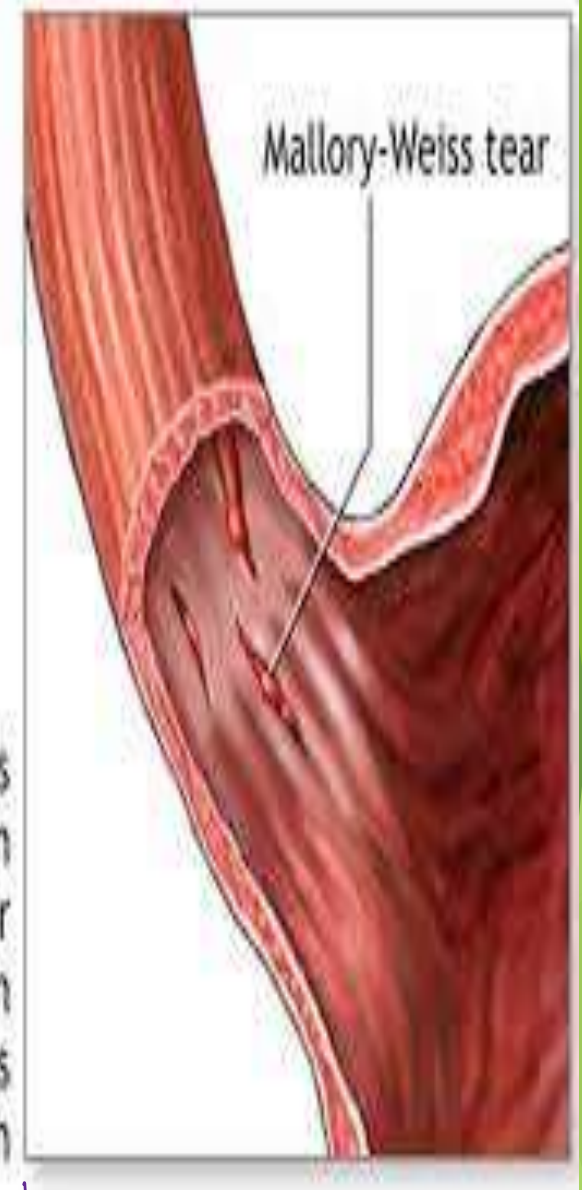
Linear
lacerations

longitudinally
oriented

Cross the GEJ.

Superficial *on mucosa*

Heal quickly , no
surgical
intervention



Mallory-Weiss
tear is a tear in
the mucosal layer
at the junction
of the esophagus
and stomach

*Typical scenario : Patient vomiting fresh blood
after forceful vomiting*

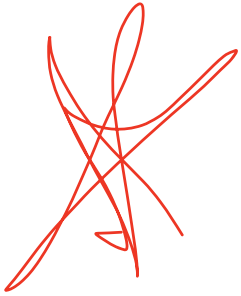
Reassure patient that this will not happen again and tears will heal

Chemical Esophagitis

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

Bcz large size [Drink alot of water
and stay upright]

graft versus host
disease



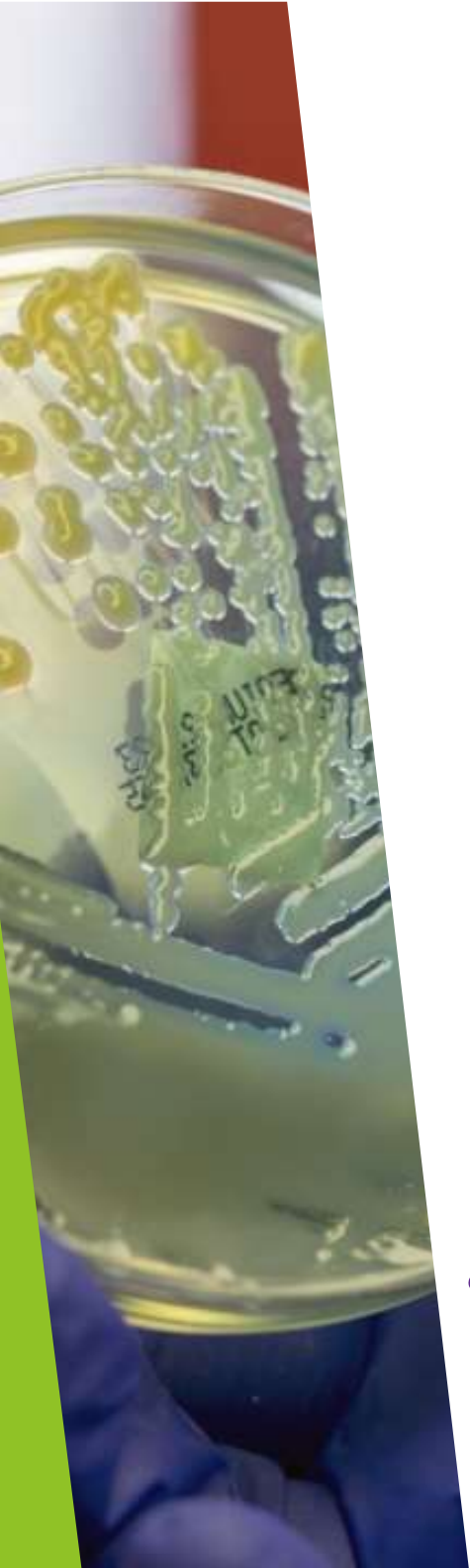
Clinical symptoms & morphology

- ▶ Ulceration and acute inflammation.
- ▶ Only self-limited pain, odynophagia (pain with swallowing).
- Complicated by:
 - esophageal stenosis
- ▶ Hemorrhage, stricture, or perforation in severe cases

Infectious esophagitis

- ▶ Mostly in debilitated or immunosuppressed.
- ▶ Viral (HSV, CMV)
- ▶ Fungal (candida >>> mucormycosis & aspergillosis)
- ▶ Bacterial: 10%.

or Secondary after fungal or viral esophagitis



► Candidiasis :

- Adherent.
- Gray-white pseudo membranes
- Composed of matted fungal hyphae and inflammatory cells

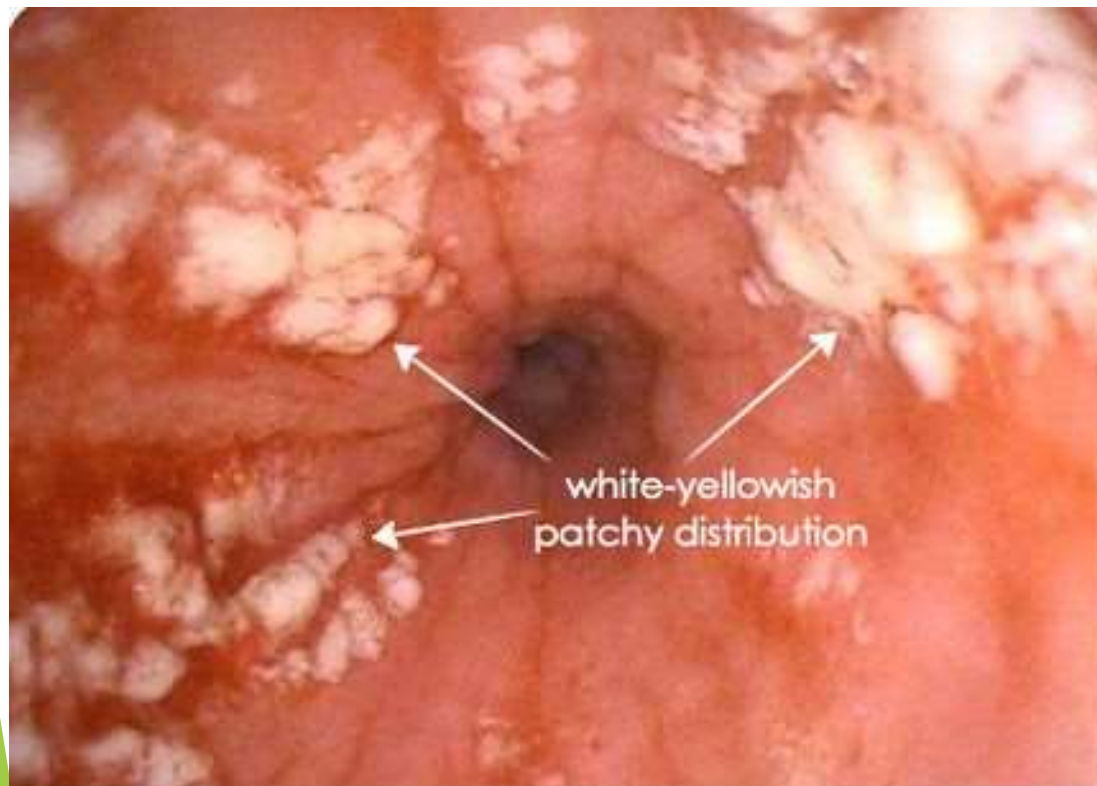
endoscopy



to esophageal mucosa

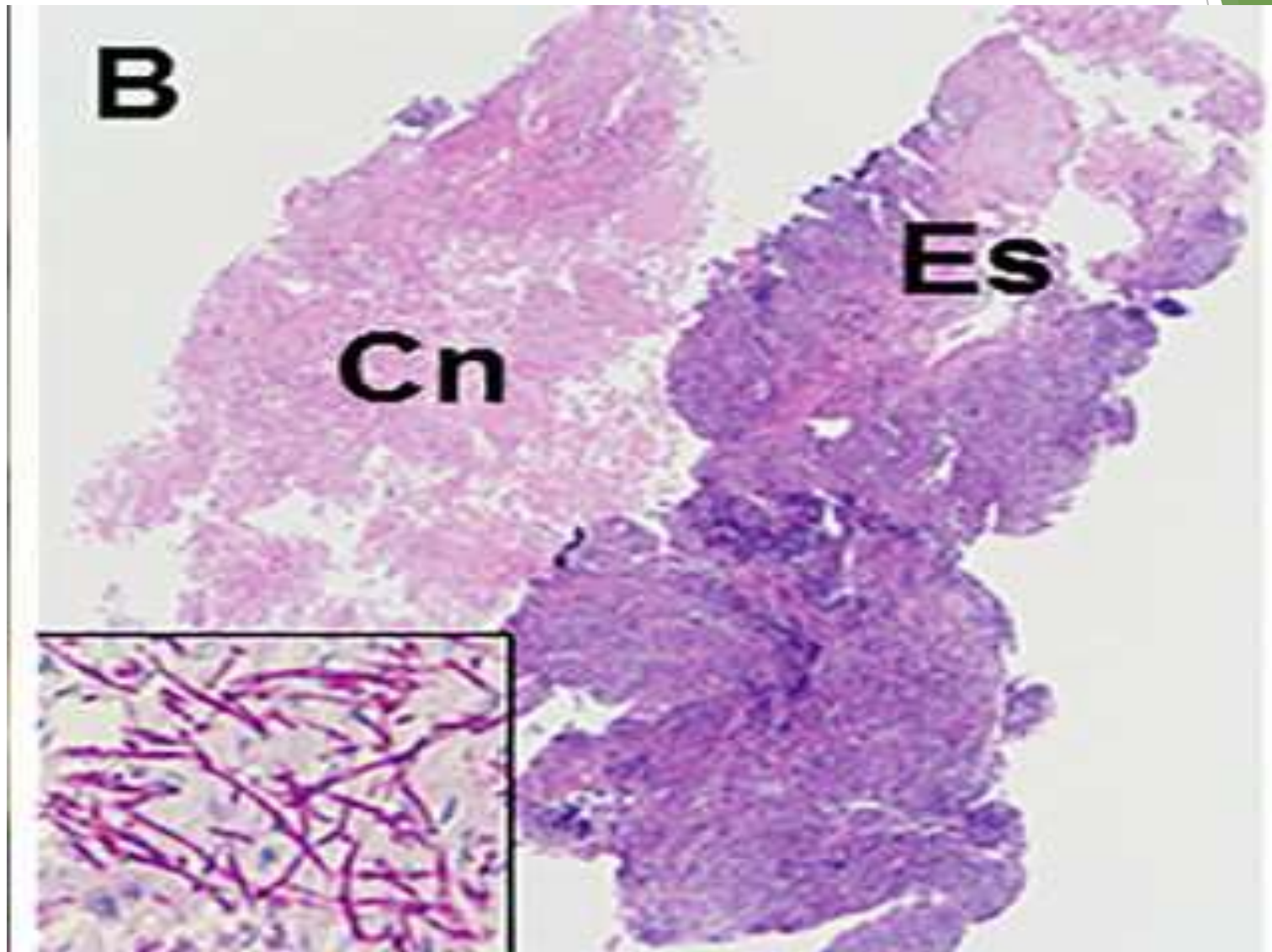
microscopy after biopsy examination

extend to oral
mucosa [oral thrush]



Esophageal Candidiasis

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



Periodic acid schiff stain
PAS - stain → fungal hyphae

www.researchgate.net/publication/285369734_Esophageal_Candidiasis_as_the_Initial_Manifestation_of_Acute_Myeloid_Leukemia

- ▶ **Herpes viruses**
- ▶ Punched-out ulcers
endoscopy
- ▶ Histopathologic:
nuclear changes
- ▶ Nuclear viral inclusions
- ▶ Degenerating ^{*sq*}epithelial cells ulcer edge
- ▶ Multinucleated epithelial cells.
fusion of cells

normally esophagus mucosa → pale pink

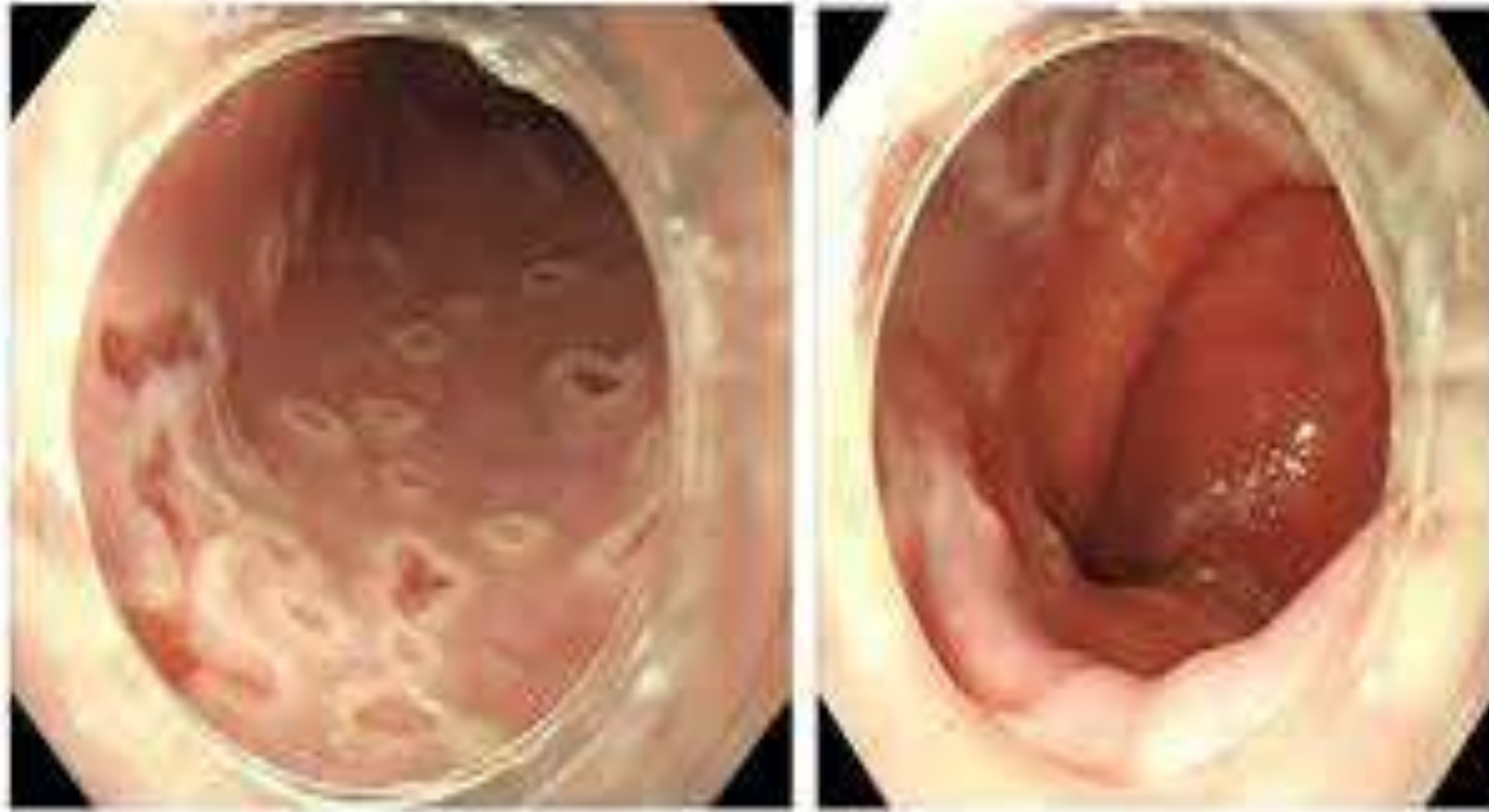
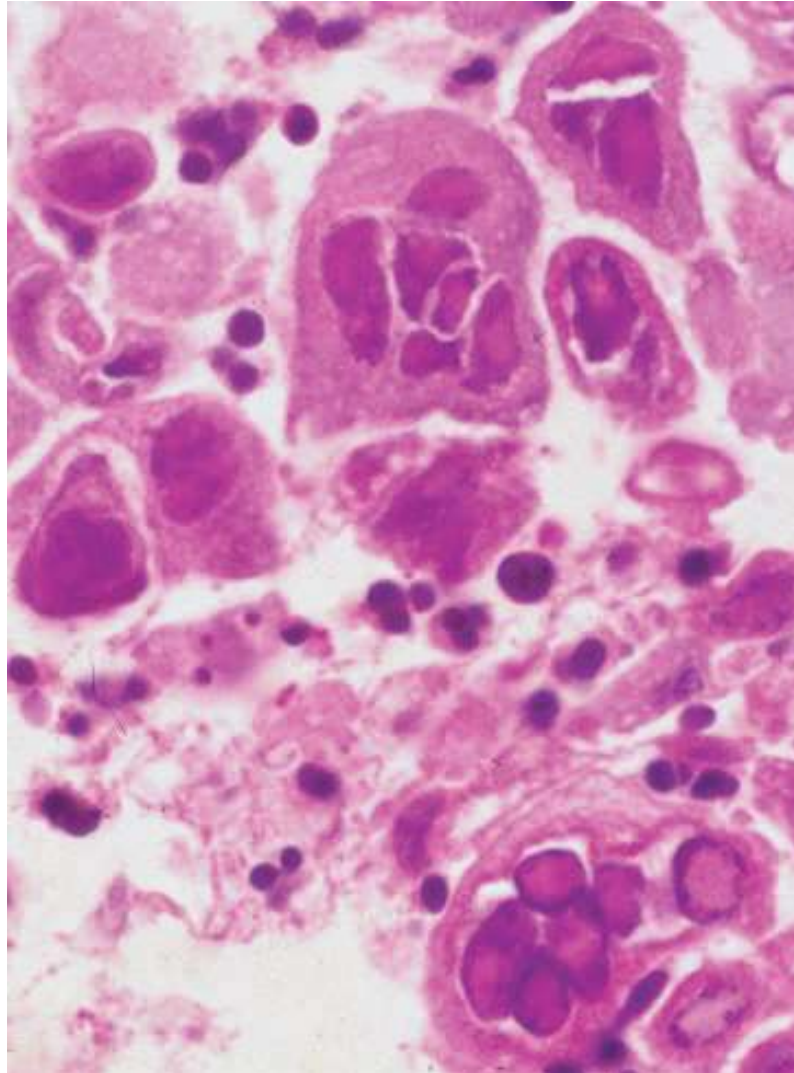
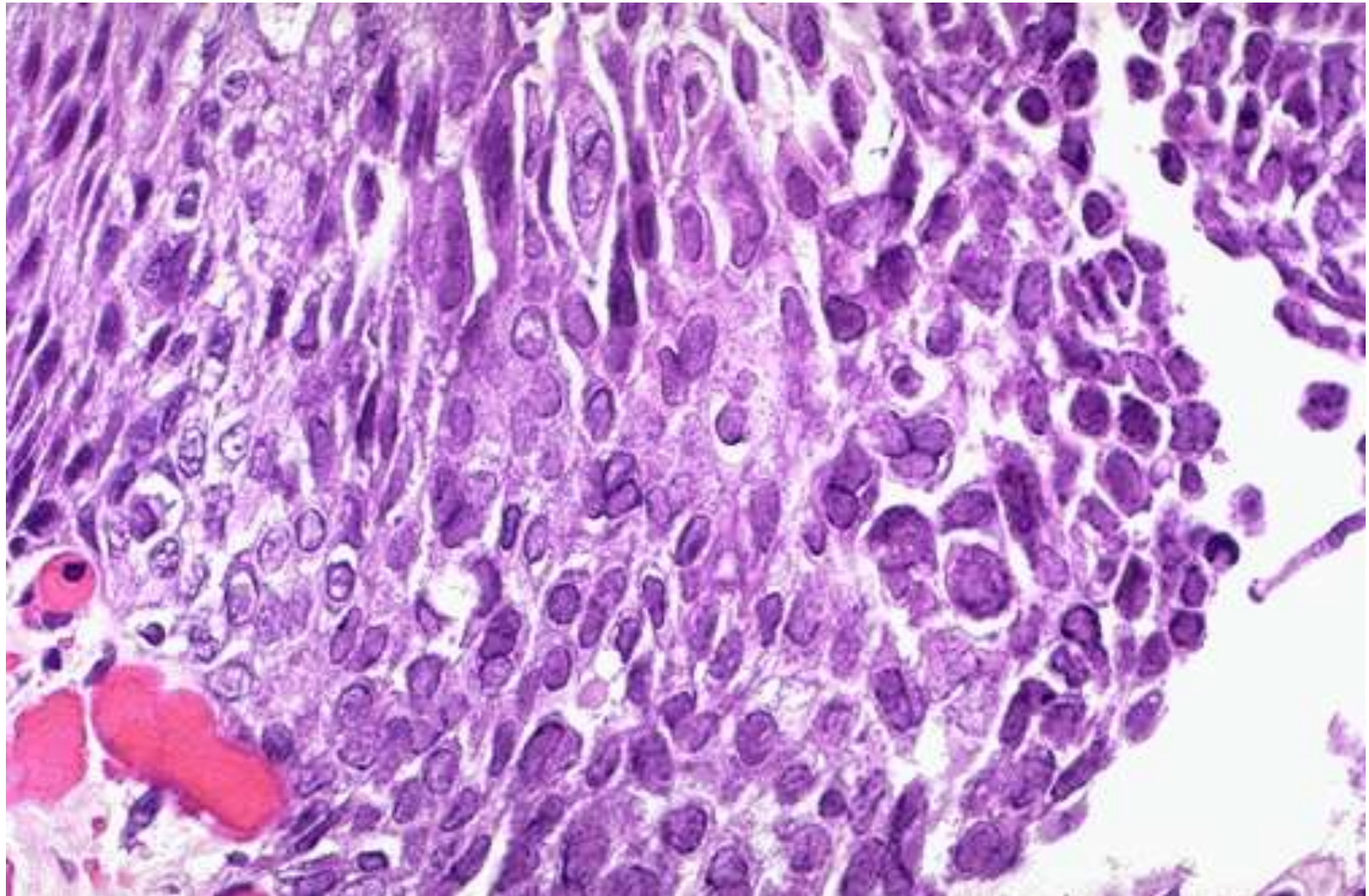


Figure 4. Gastroesophageal Reflux caused the appearance of multiple

Semantic Scholar
Surrounding erythematous (red)
mucosa in esophagus





- ▶ **CMV :**
- ▶ Shallower ulcerations.
- ▶ Biopsy: nuclear and cytoplasmic inclusions in capillary endothelium and stromal cells. (Mega cells)
very large

*not only epithelial cells
as in herpes simplex*

