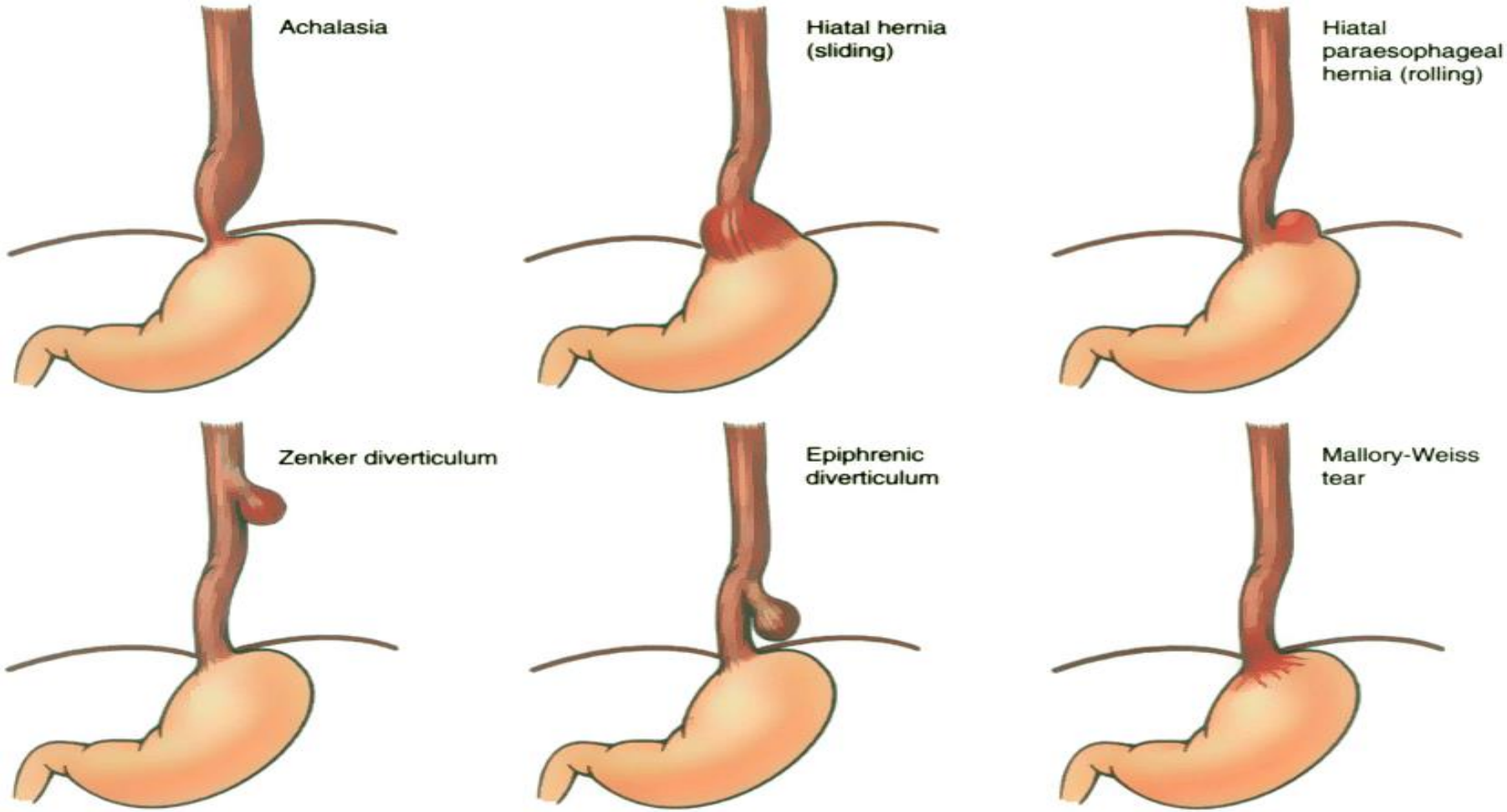
GIT pathology

Dr Methaq Mueen

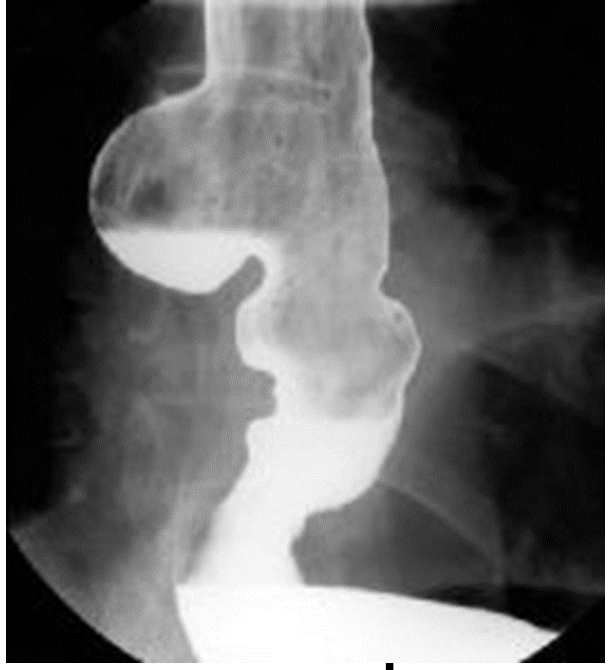
LEC 2

Motor dysfunction



Motor dysfunction

2-*Diverticulae:*



Definition: is a "focal out pouching of the alimentary tract wall that contains all or some of its constituents";

In general they are divided into:

1. False diverticulum is an out pouching of the mucosa and submucosa (No muscular layer) through weak points in the muscular wall.

2. True diverticulum consists of all the layers of the wall and is thought to be due to motor dysfunction of the esophagus.

Types of esophageal diverticulae:

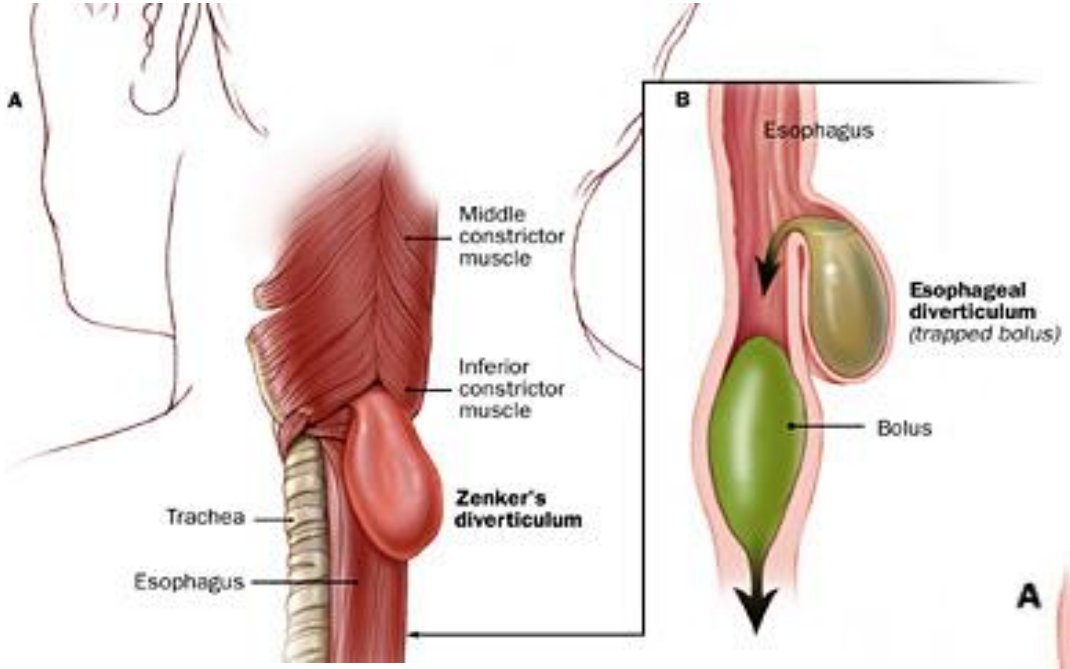
They may develop in **three regions** of the esophagus

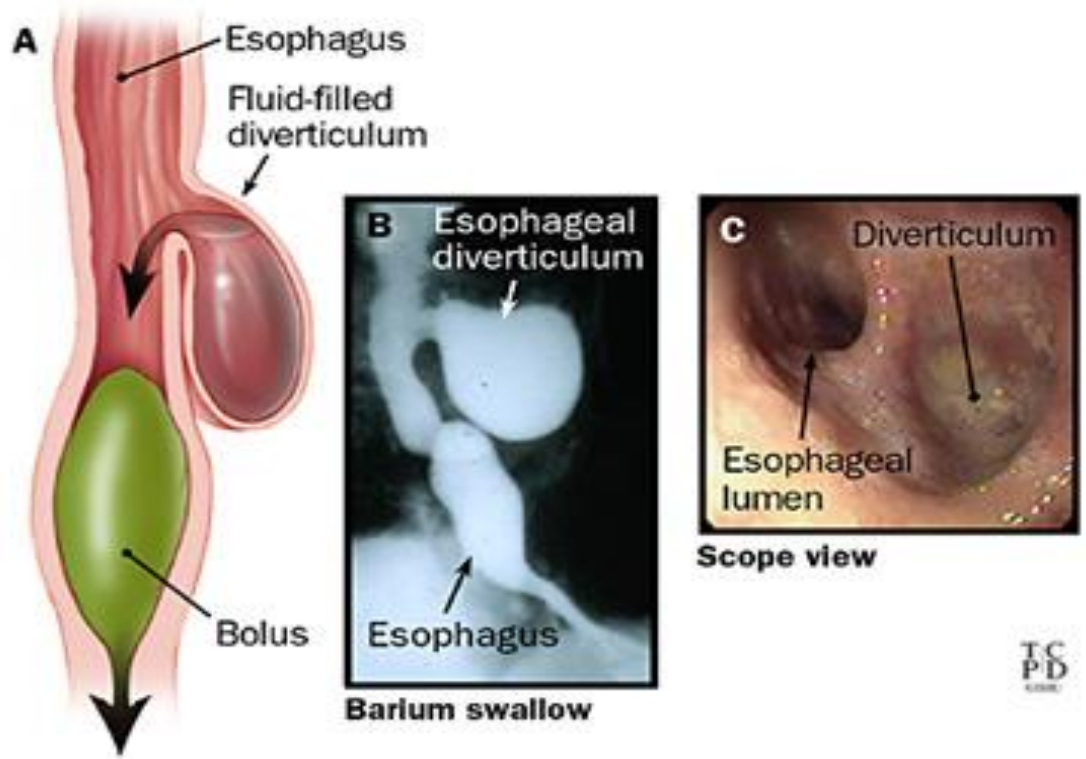
1. upper pulsion (Zenker diverticulum), located immediately above the upper esophageal sphenictor(UES)(in the hypopharyngeal area)

Etiological cause: *occurs due to increased pressure in the oropharynx during swallowing against a closed upper esophageal sphincter*.

1-motor dysfunction of the cricopharyngeal muscle(incomplete relaxation)

2-Elevated resting tone of the entire upper esophageal sphincter (UES).


A
Middle constrictor muscle
Inferior constrictor muscle
Zenker's diverticulum
Trachea
Esophagus
B
Esophagus
Esophageal diverticulum
(trapped bolus)
Bolus


A
Esophagus
Fluid-filled diverticulum
Bolus
B
Esophageal diverticulum
Esophagus
Barium swallow
C
Diverticulum
Esophageal lumen
Scope view

Treatment :When symptoms are severe, surgical intervention is the rule.

Mic Zenker diverticula are lined with stratified squamous epithelium with a thin lamina propria.

No muscular layer exists.

Fibrosis surrounding the diverticulum is common

2.middle (Traction diverticulum) near the midpoint of the esophagus

Cause: scarring resulting from e.g mediastinal lymphadenitis caused by T.B,

which lead to pulling of the esophageal wall leading to **sac** formation.

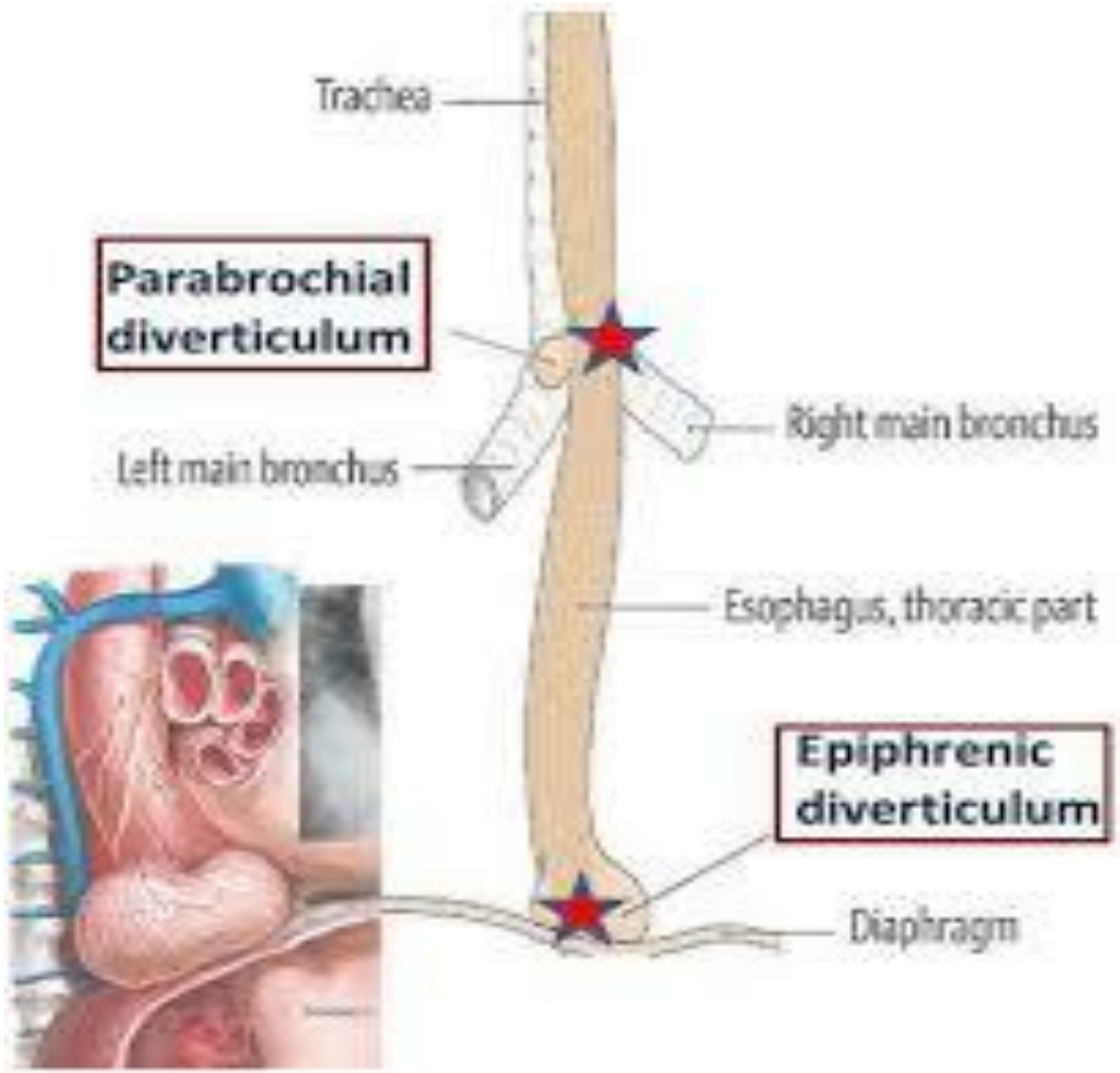
3. Epiphrenic diverticulum true diverticulum immediately above the LES (above the diaphragm).

Causes:

increased pressure during esophageal propulsive contractions against a closed lower esophageal sphincter.

1-Motor disturbances of the esophagus (e.g., achalasia, diffuse esophageal spasm).

2-Reflux esophagitis


Trachea
Parabrochial diverticulum
Right main bronchus
Left main bronchus
Esophagus, thoracic part
Epiphrenic diverticulum
Diaphragm

Signs and symptoms:

- Nocturnal regurgitation of large amounts of food stored in the diverticulum during the day(occasionaly for days) is typical with no dysphagia
- Mass in the neck
- **Complications:**

1- Aspiration pneumonia

2- Perforation

3-hiatus hernia

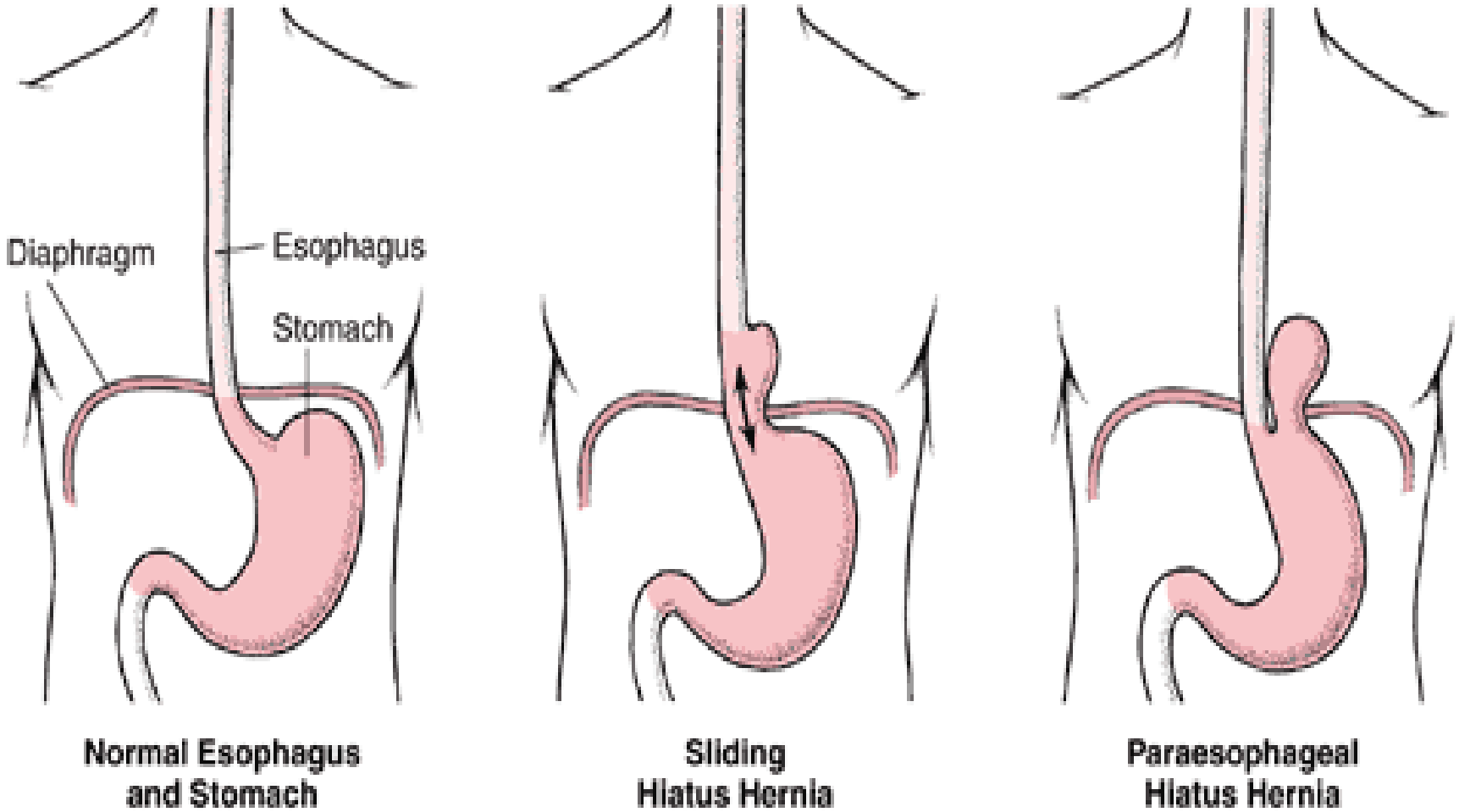
It is a protrusion (herniation) of the sac like gastric segment above the diaphragm into the thorax. due to separation of the diaphragmatic crura and widening of the space between the muscular crura and esophageal wall

1- sliding hiatus hernia : 95% of hernia cases.

*Here the gastroeophageal junction is displaced upwards above the diaphragm.

*Usually it is associated with reflux esophagitis.

Hiatus hernia



- **Causes:**

1- Abnormal short esophagus.

2- Esophageal spasm and traction of the stomach.

3- Repeated episodes of increased intra abdominal pressure (coughing, vomiting, --- etc.)

C/F: heartburn and regurgitation of gastric juice due to LES incompetence .

2-paraesophageal(Rolling)hiatus hernia:

* Here separated portion of the stomach (mainly the gastric fundus) enters the thorax through a wide diaphragmatic foramen.

- It is **NOT** associated with reflux esophagitis.
- But may strangulate and infarct.

Complications of hiatal hernias include

1. Ulceration, bleeding and perforation (both types)
2. Reflux esophagitis (frequent with sliding hernias)
3. Strangulation and infarction (paraesophageal hernias)

- ***Esophageal laceration***

  Also called **Mallory Weiss** syndrome .

  - It consists of longitudinal tears in the esophagus at the gastroesophageal junction(GEJ)
  - Seen in alcoholic individuals after sever vomiting

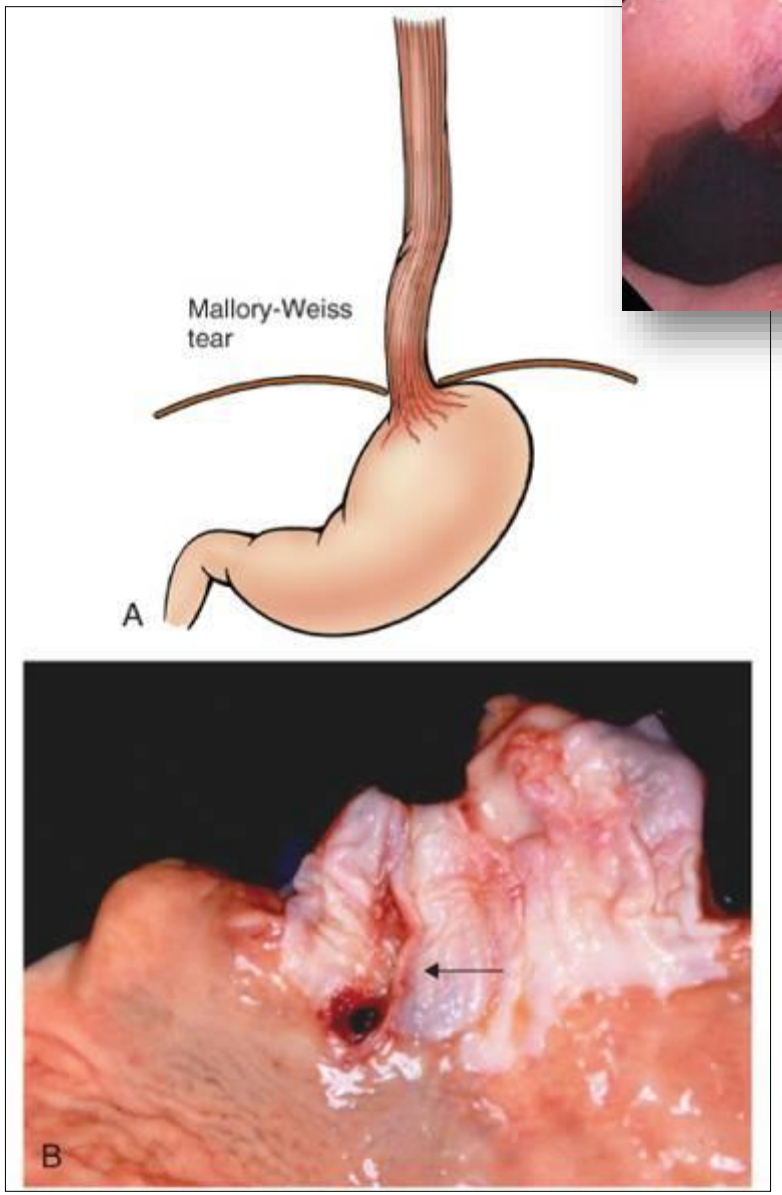
Mallory-Weiss Tear (Syndrome)

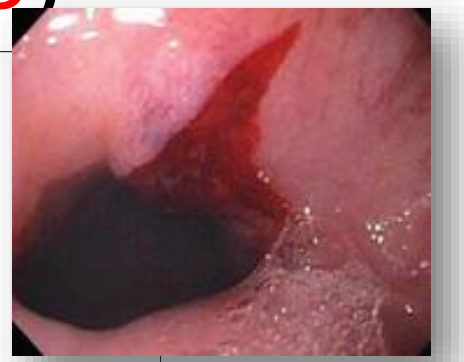
Severe/forced vomiting.

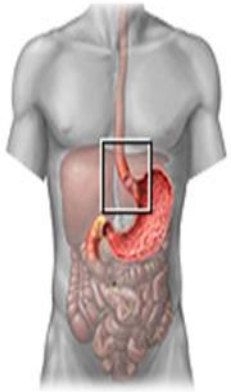
Longitudinal mucosal tear.

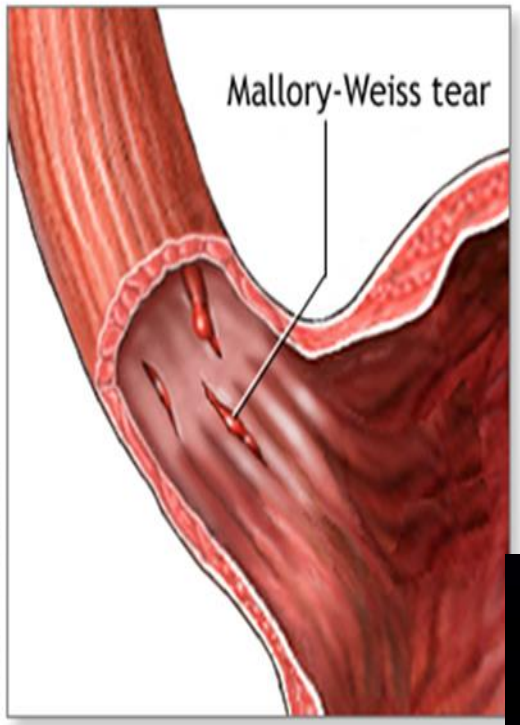
Chronic Alcoholics,

Spontaneous healing.

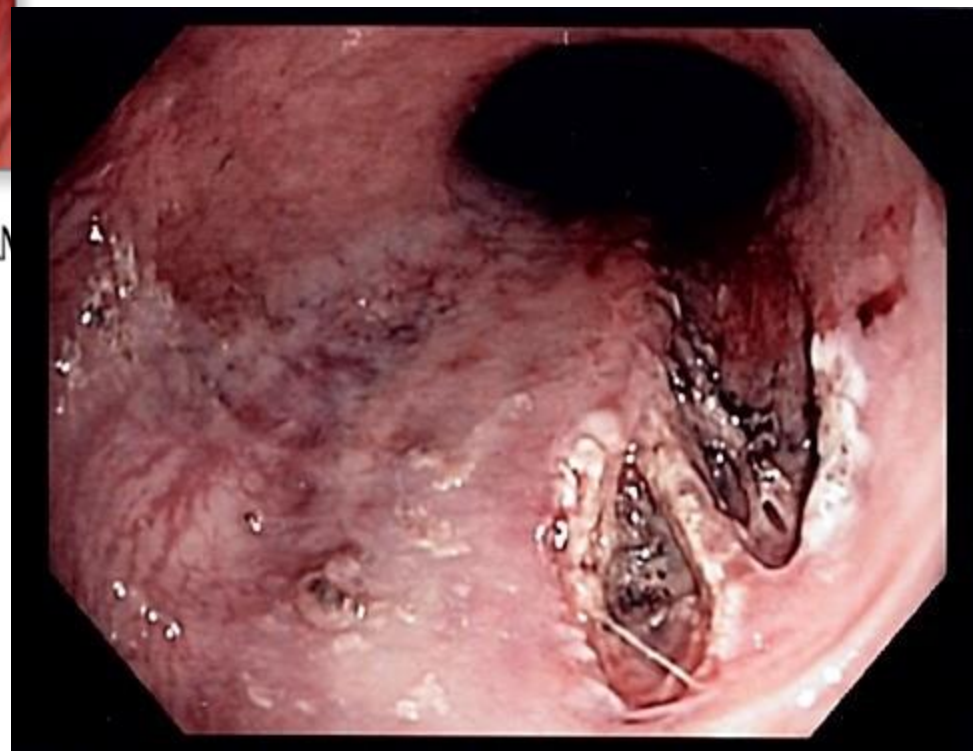








Mallory-Weiss tear is a tear in the mucosal layer at the junction of the esophagus and stomach



pathogenesis

Normally, a reflex relaxation of the gastroesophageal musculature precedes the antiperistaltic contractile wave associated with vomiting. This relaxation is thought to fail during prolonged vomiting, with the result that refluxing gastric contents overwhelm the gastric inlet and cause the esophageal wall to stretch and tear.

.

complications

The linear irregular lacerations extend through the mucosa , or may penetrate **deeply** to perforate the wall leading to upper gastrointestinal bleeding

It forms about 5-10% of UGI bleeding.

Infection of the mucosal defect may lead to inflammatory ulcer or to mediastinitis.

Prognosis: These tears are superficial

Usually the bleeding is not profuse and stops without surgical intervention.

Healing tends to be rapid and complete and it is the usual outcome.

Rarely esophageal rupture occurs.

.

Clinical presentation

Patients often present with hematemesis.

Endoscopically:The roughly linear lacerations of Mallory-Weiss syndrome are longitudinally oriented, range in length from millimeters to several centimeters, and usually cross the gastroesophageal junction.

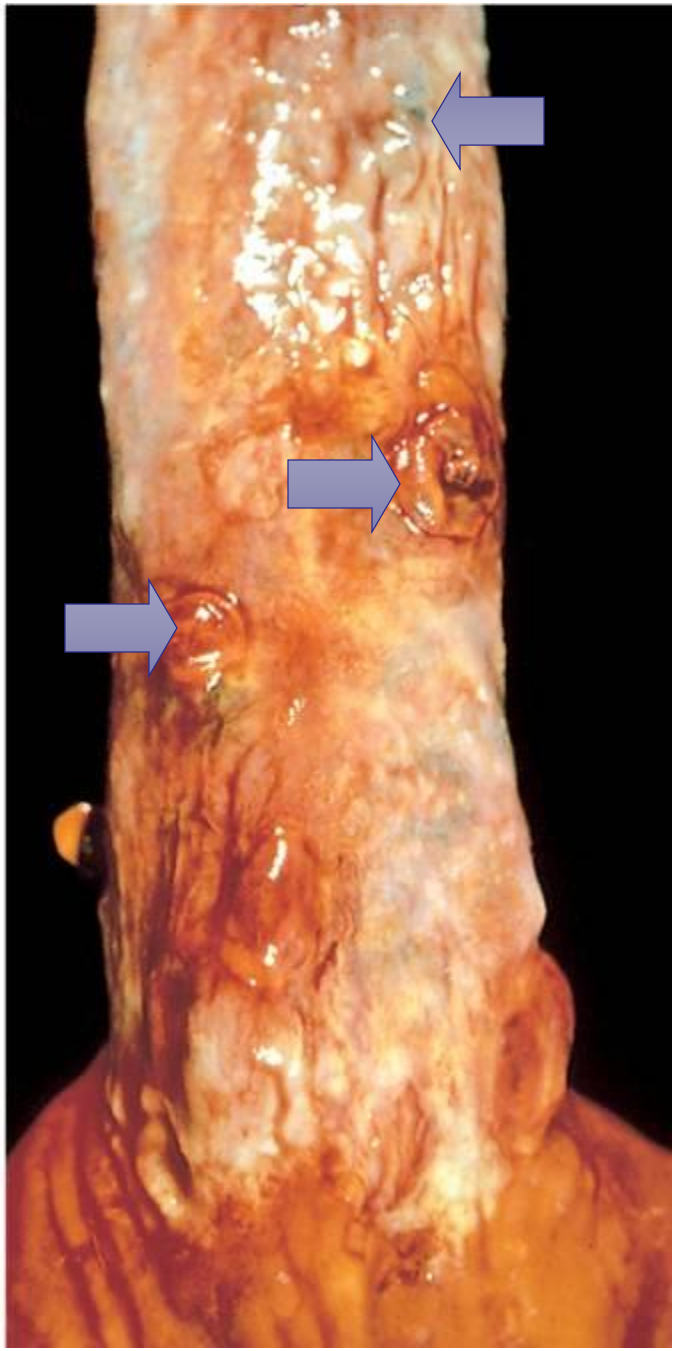
Esophageal Varices:

Dilated veins – lower part.

Pathogenesis: Portal hypertension (Cirrhosis) → Porta-Systemic Shunts open → varices of - lower esophageal veins, peri-umbellical, Rectal V

Rupture → massive bleeding.

- Varices are tortuous dilated veins lying primarily within the submucosa
- of the distal esophagus and proximal stomach



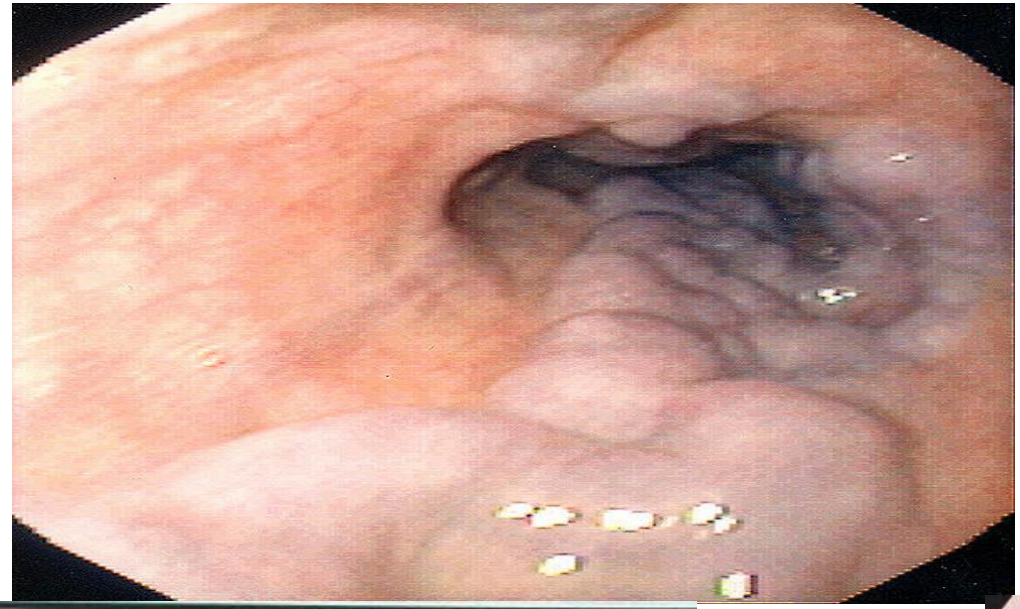
Pathogenesis. Portal hypertension results in the development of collateral channels at sites where the portal and caval systems communicate.

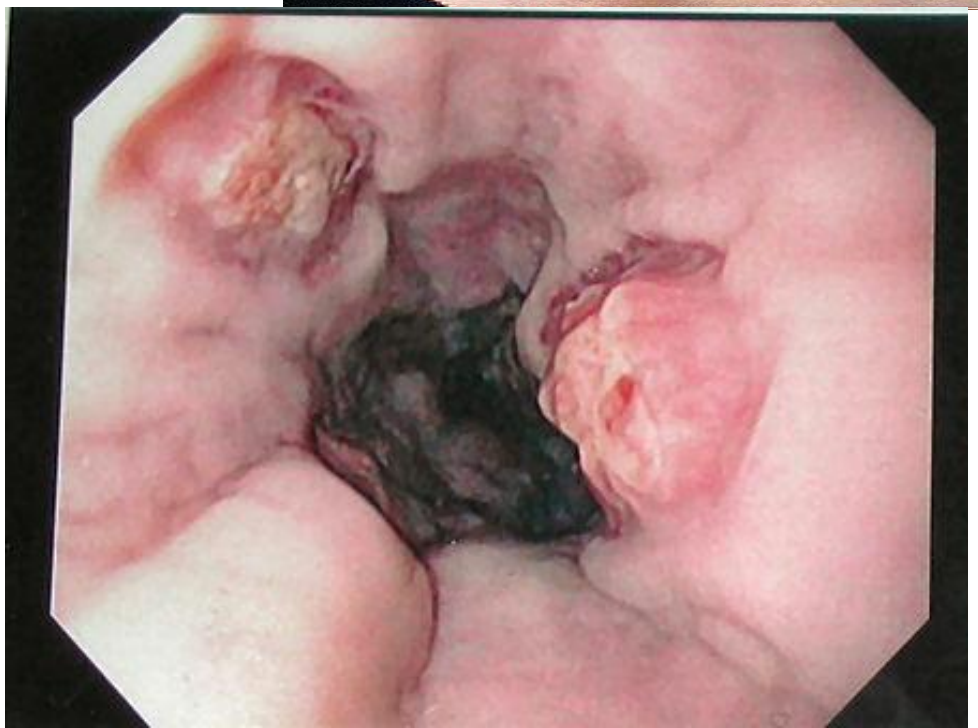
These collateral veins allow some drainage to occur, but at the same time they lead to development of congested subepithelial and submucosal venous plexi within the distal esophagus and proximal stomach.

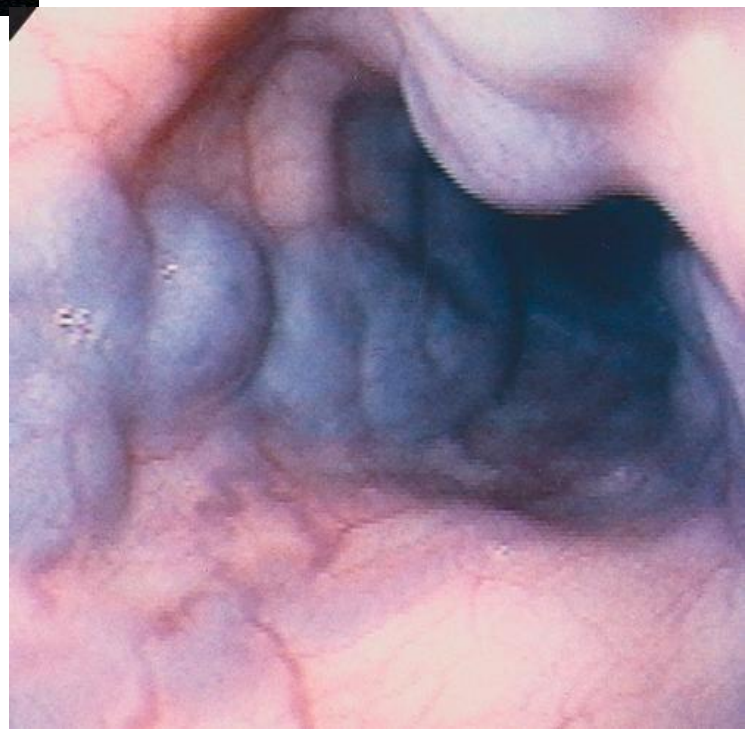
These vessels, termed varices, develop in the vast majority of cirrhotic patients, most commonly in association with alcoholic liver disease.

Worldwide, hepatic schistosomiasis is the second most common cause of varices.

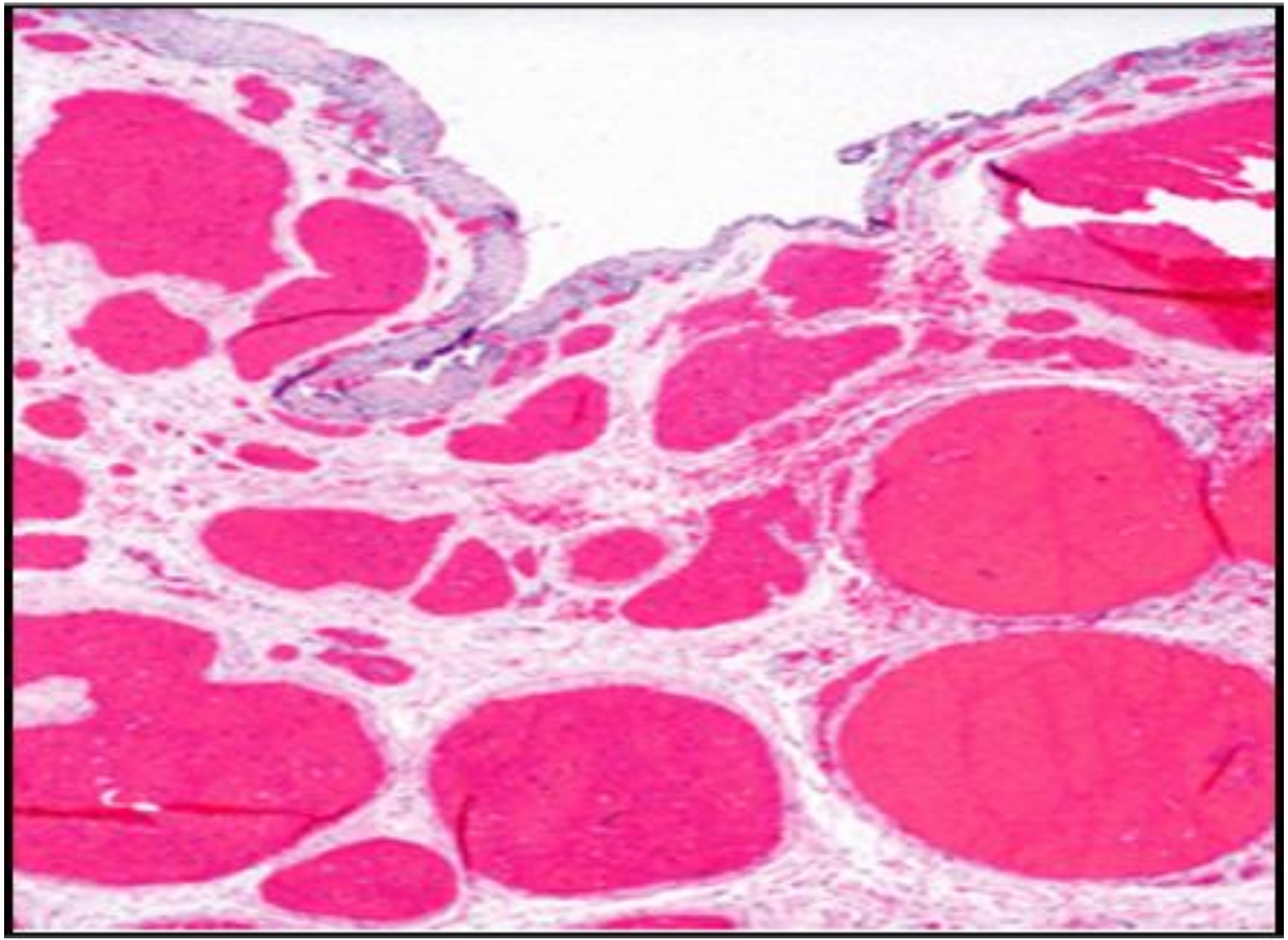
Endoscopic views – dilated tortuous submucosal veins(like bag of worms)



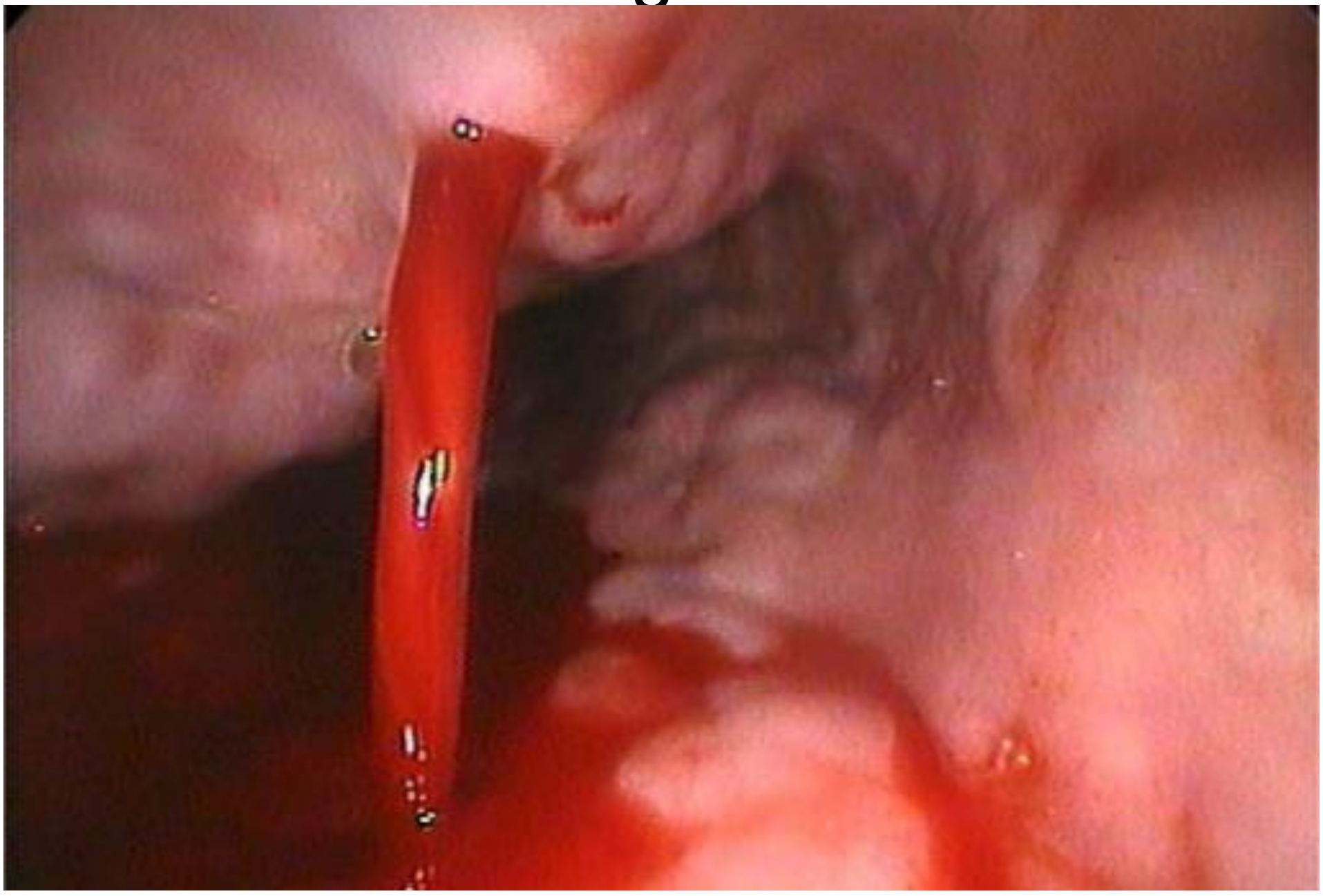




Microscopy



Bleeding varices



Causes for massive Upper GI bleeding

Esophageal varices

Bleeding gastric ulcer

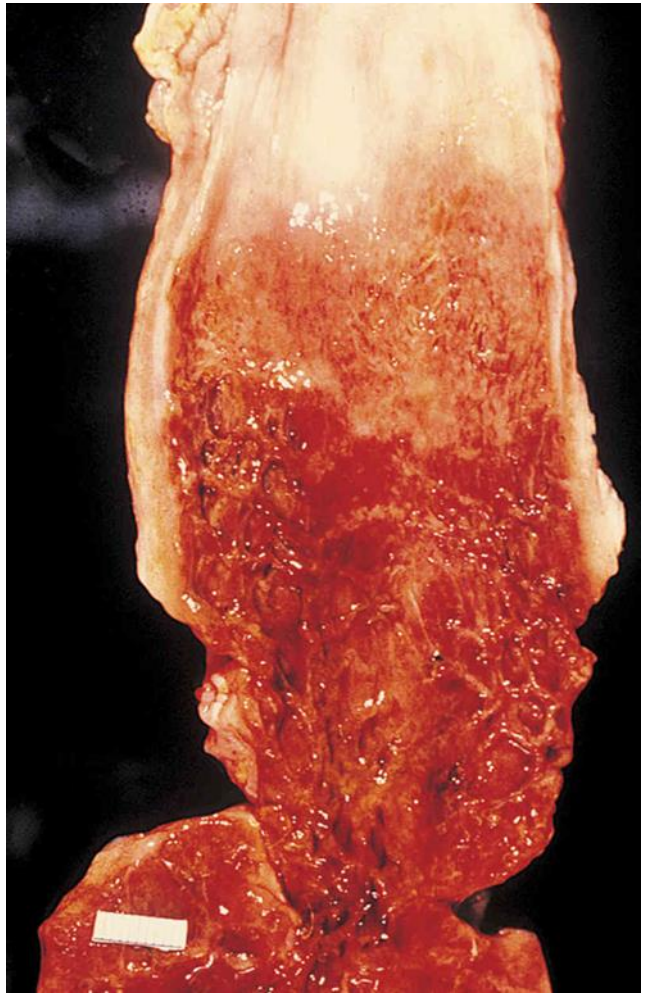
Bleeding duodenal ulcer

- **Esophagitis**

It is inflammation of the esophagus.

The causes are divided into:

- infective
- non infective



- **Infective causes:**
- ***1- Fungal:*** candid, mucormycosis, aspergillosis

Candida albicans:

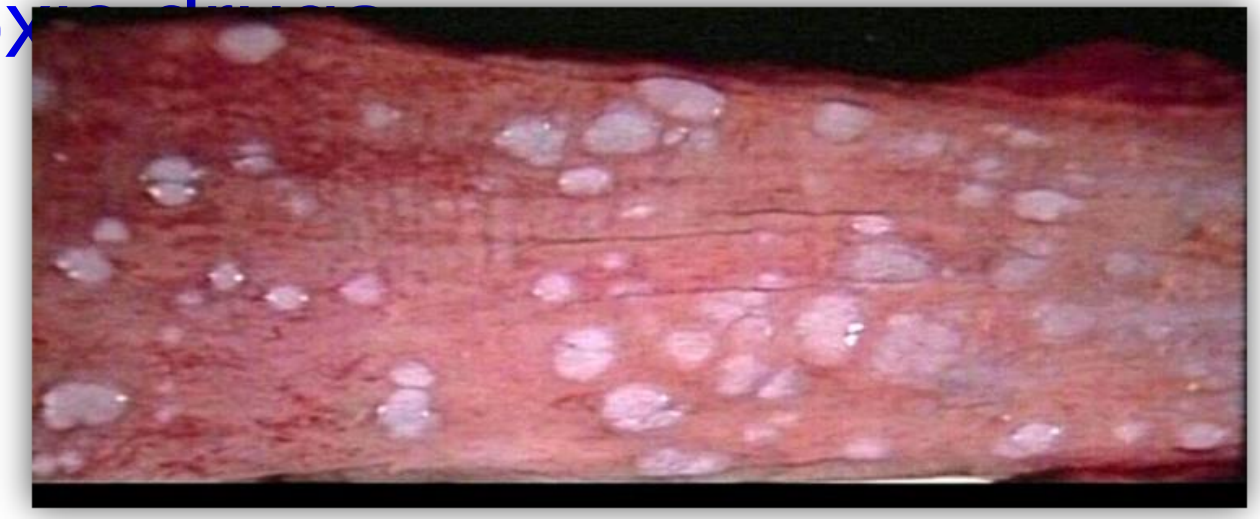
Especially in:* chronic debilitating diseases,

* Diabetes mellitus,

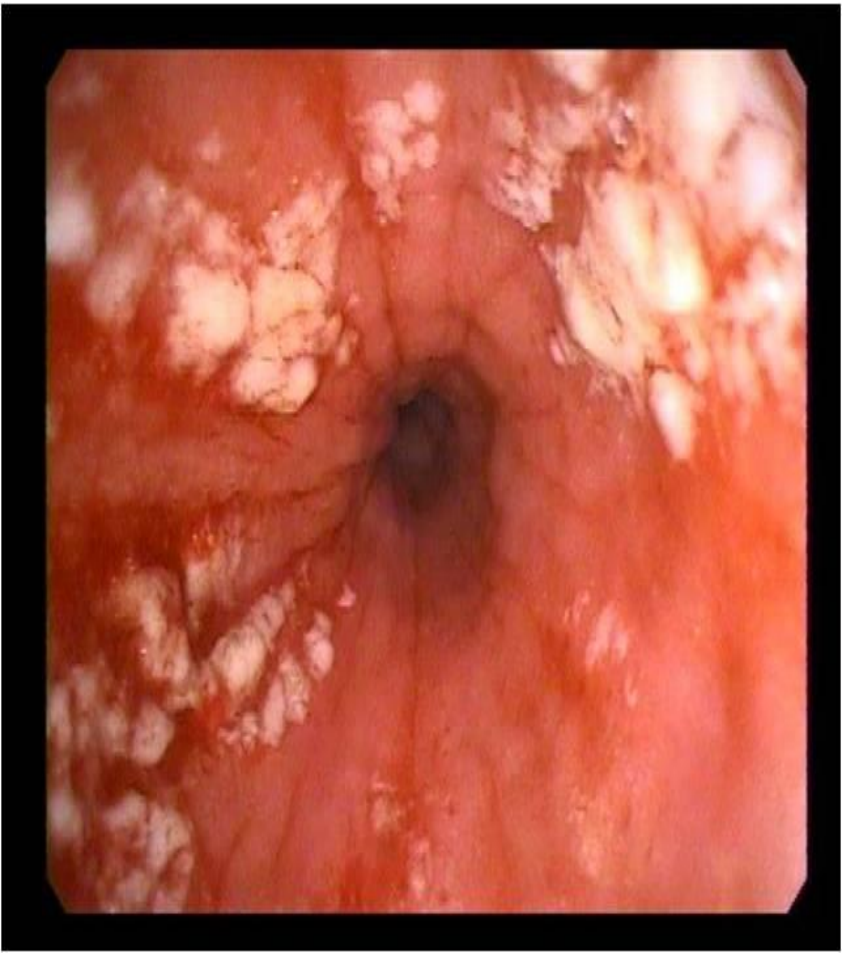
* AIDS,

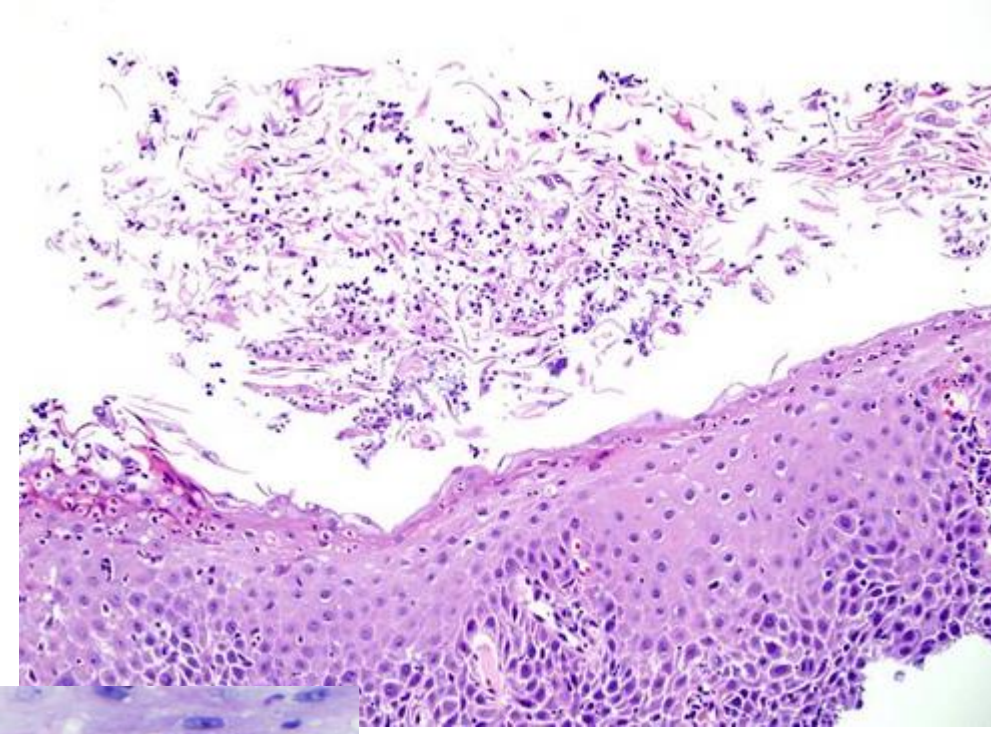
- Patients taking cytotoxic drugs
- **Morphologicaly:**

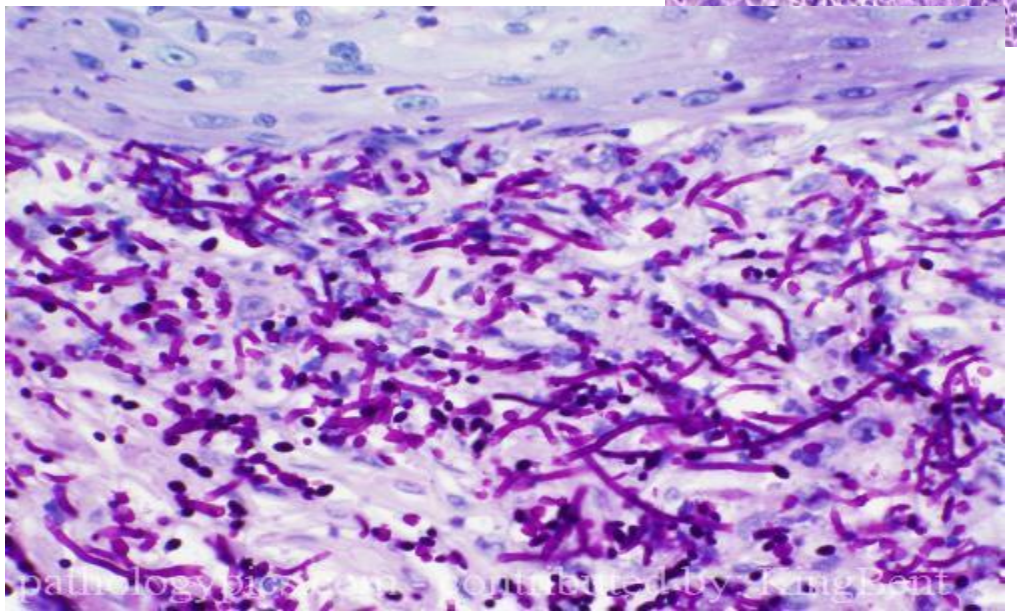
The esophagus covered by adherent gray –white pseudomembrane.



densely matted fungal hyphae and inflammatory cells covering the esophageal mucosa







2- Herpes simplex &cytomegaloviruses:

Morphologically it produces punched out ulcers.

3-Bacteria:

Accounts for 10-15% of infective esophagitis .

- **Non infective causes:**

1- ***Acute esophagitis*** caused by:

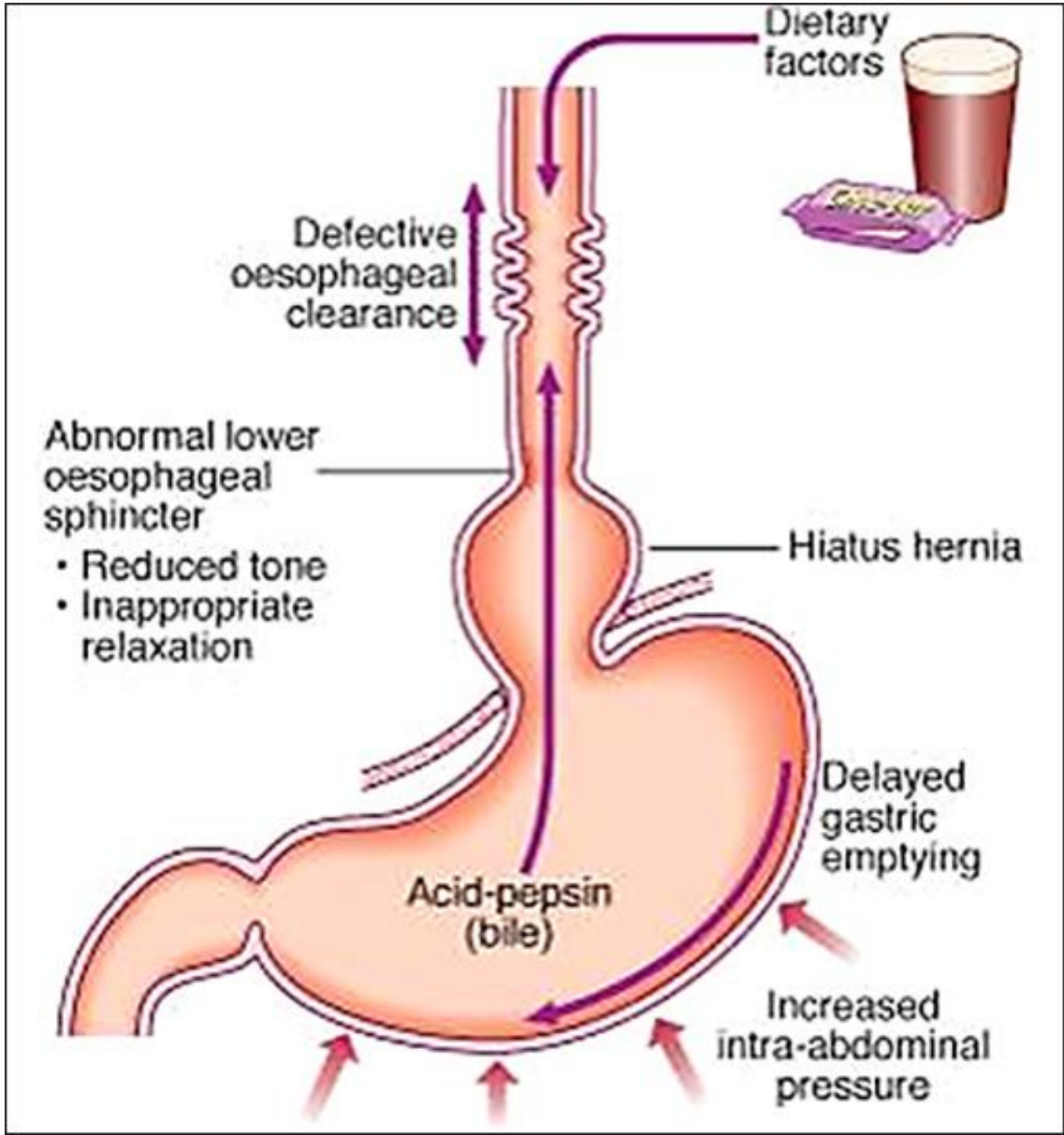
* Ingestion of mucosal irritants (alcohol, corrosive acid or alkali, hot food or drink)
* Drugs (tablets or capsules) when sticking in the esophagus
* Irradiation or chemotherapy.

- ***Reflux esophagitis***:

Is **reflux** of the gastric content into the lower esophagus in which the acid –peptic action of the gastric juice is the main cause of injury leading to inflammation of the **lower esophagus .**

Etiological and pathogenesis:

* Decrease efficacy of the esophageal anti-reflux mechanism especially of the LES(transient LES relaxation mediated via vagal pathways).
* Presence of sliding hiatal hernia.
* Slow esophageal clearance of the refluxed material.
* Decrease gastric emptying or gastric distention, by gas or food,
* abrupt increase in intraabdominal pressure(coughing, straining, or bending)… forceful opening of a relatively hypotensive LES
* Impaired reparative capacity of the esophageal mucosa by prolonged exposure to gastric juices.
* Other cause: Pregnancy, obesity alcohol and tobacco use,
* In many cases, no definitive cause is identified.



- **Clinical features:**

occur usually at the age > 40 years(it can occur at any age)

heart burn

dysphagia

Regurgitation of sour-tasting gastric contents.

- Complications:

-Bleeding

- Stricture

- **Barrett esophagus**

- **Barrett esophagus**

-Is a complication of long standing gastroesophageal reflux.

-Seen in 11% of reflux esophagitis cases.

-It is considered as a **premalignant condition**.

Morphologically:

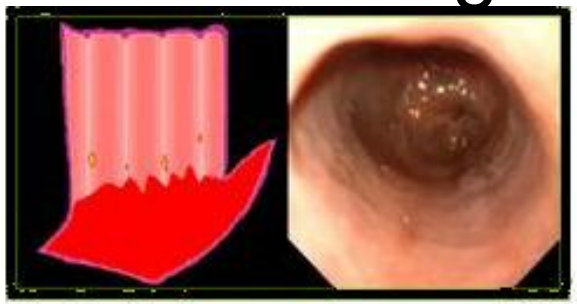
The distal part of the esophagus which is normally lined by squamous epithelium will be replaced by **metaplastic columnar epithelium containing goblet cells** due to prolonged injury, because the metaplastic columnar epithelium are more resistant to injury from refluxing gastric contents.

- **Complications:**

Metaplastic epithelium may be converted into dysplastic cells and then **adenocarcinoma** .

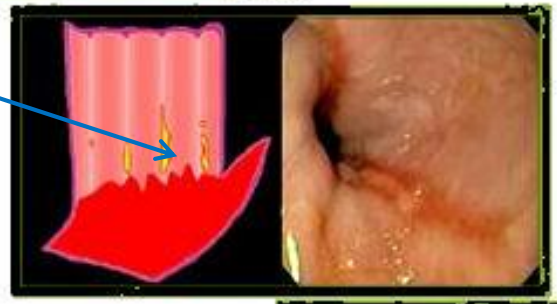
This is explained by: inflammation and ulceration may lead to ingrowth of stem cells which then differentiate into columnar epithelium which resists the acidic environment.

Pathogenesis & Stages:

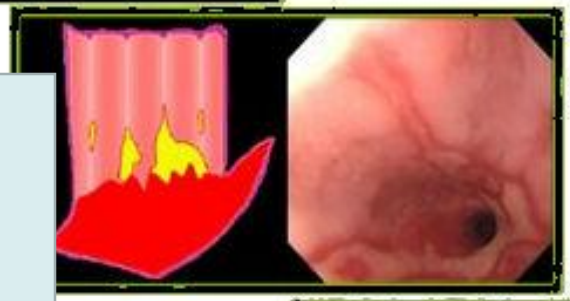


A

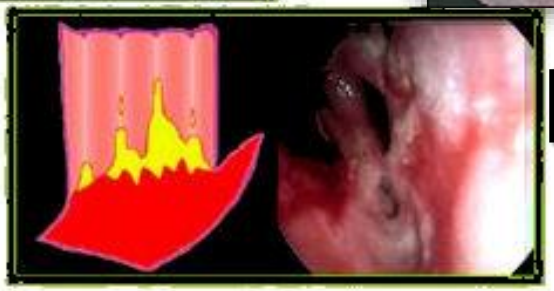
Basal Hyperplasia



B



C



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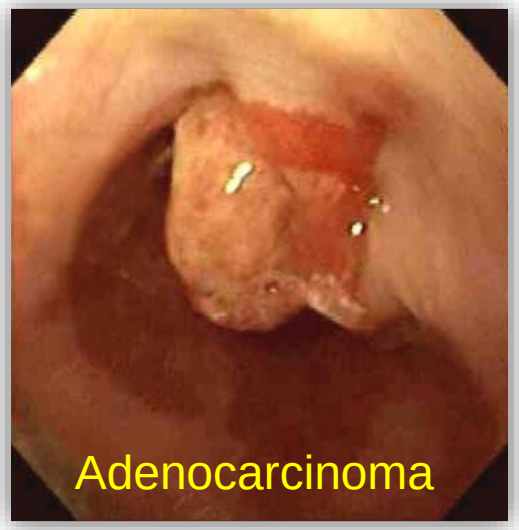
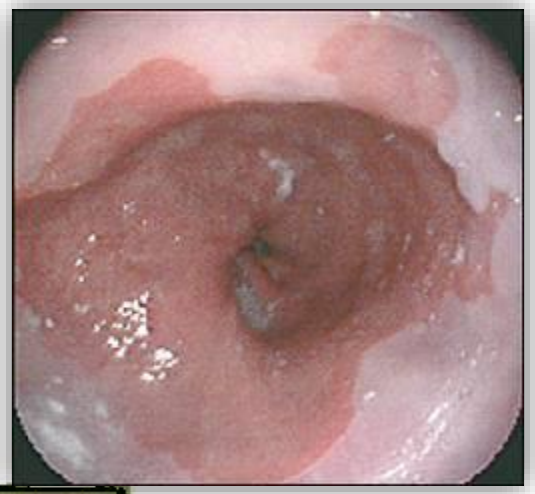
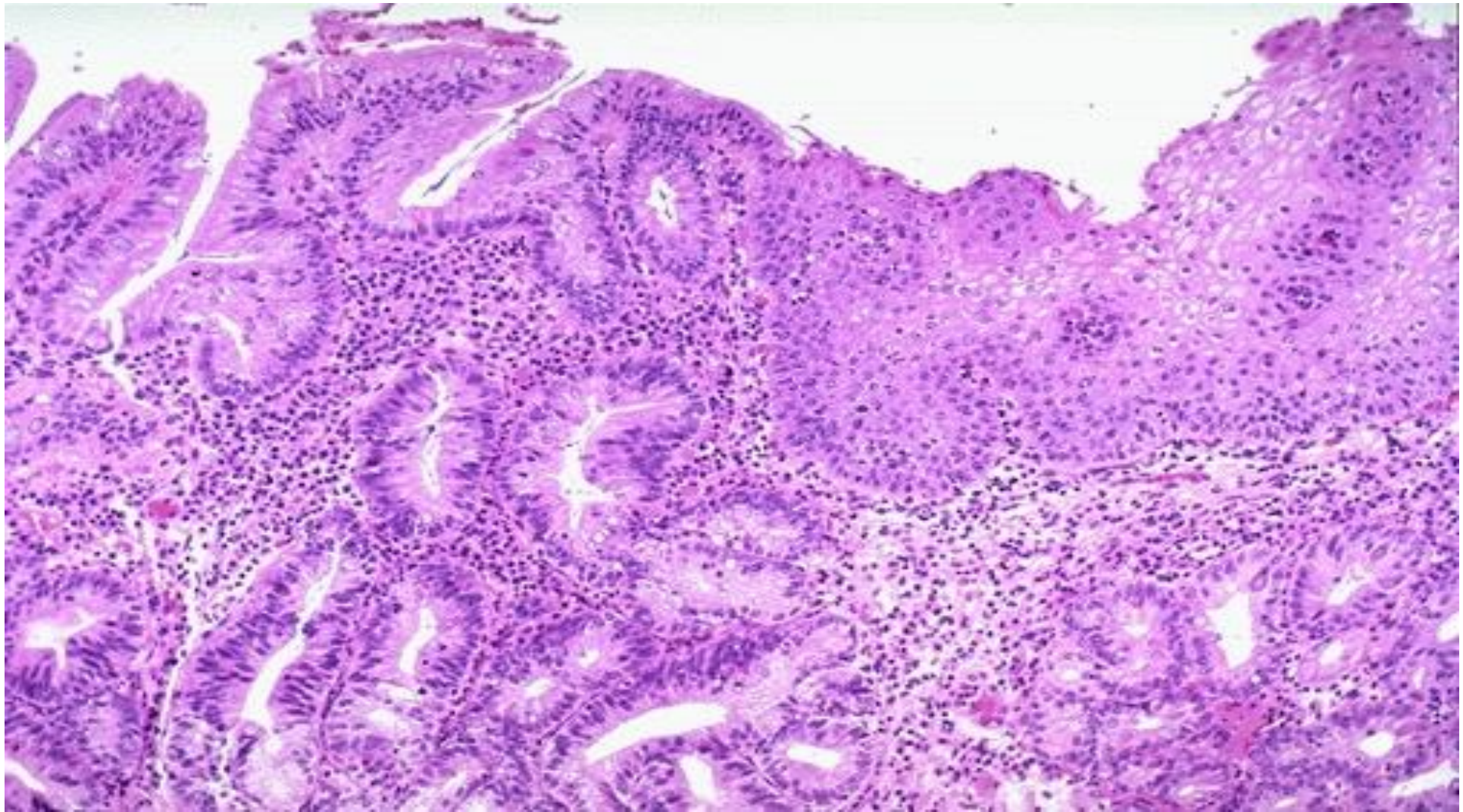




1. Acid reflux Symp.
2. Inflammation
3. Regeneration (basal).
4. Metaplasia (Barretts)
5. Mild Dysplasia
6. High grade Dysplasia
7. Adeno-Carcinoma

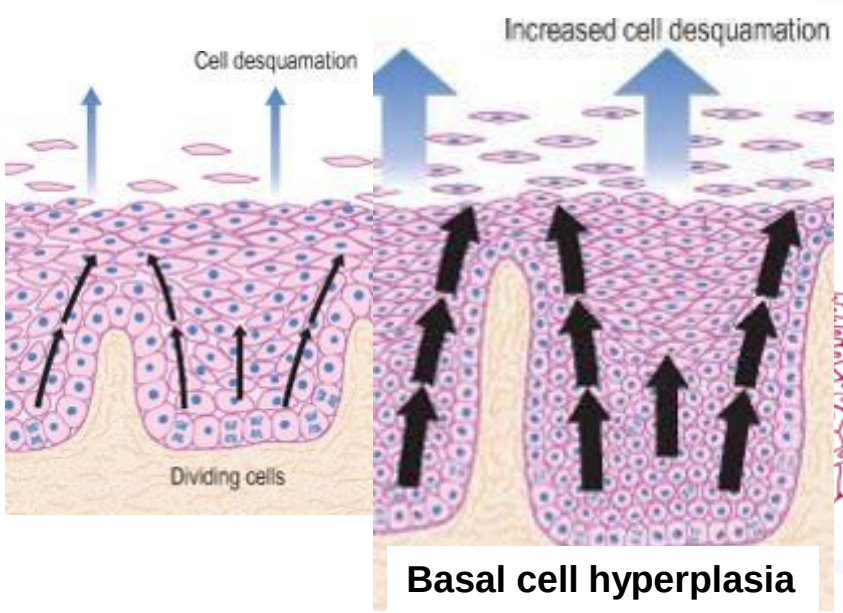
1: 85-92

Barrett esophagus

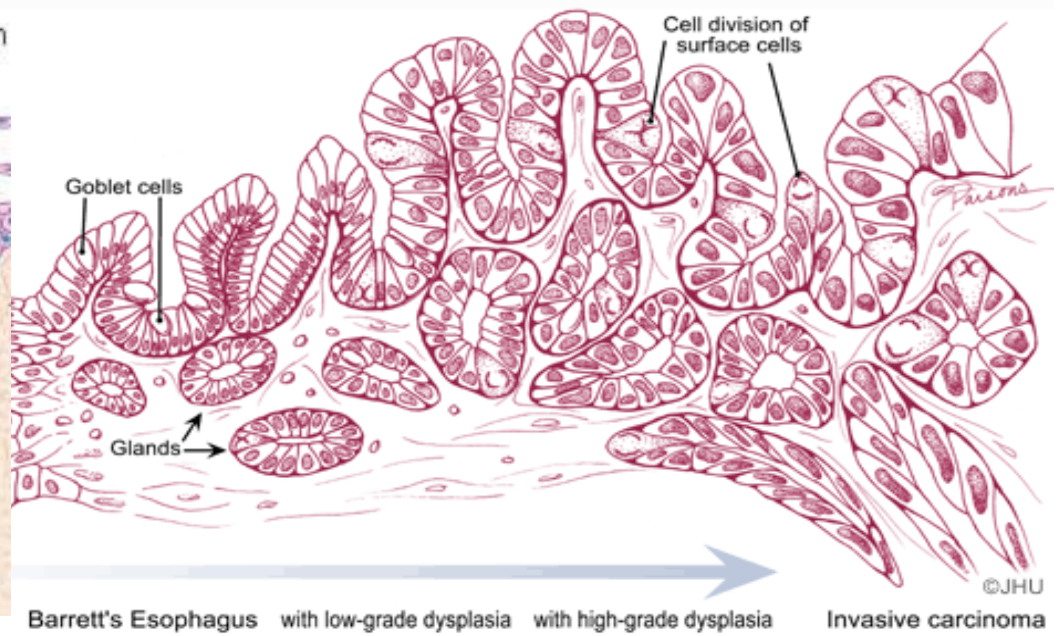


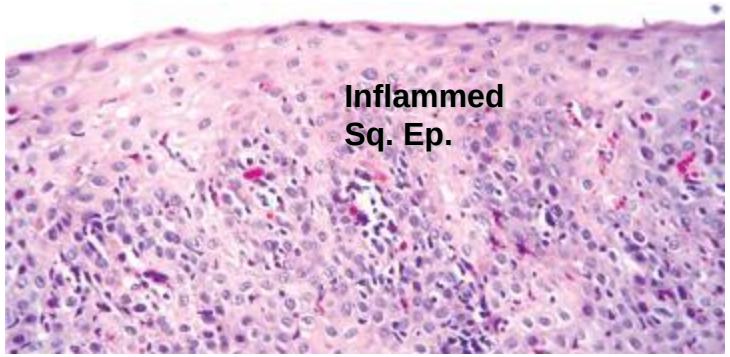
GERD: Pathogenesis.

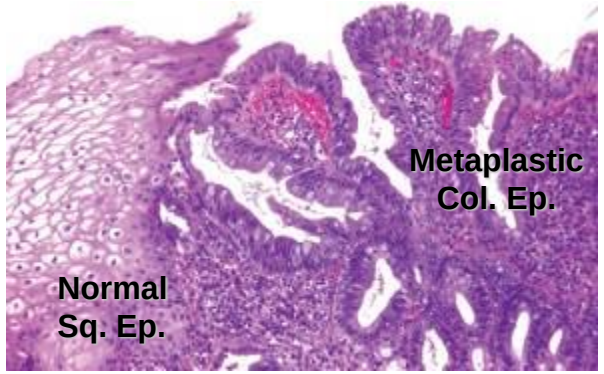
Normal → Hyperplasia → Dysplasia → Carcinoma



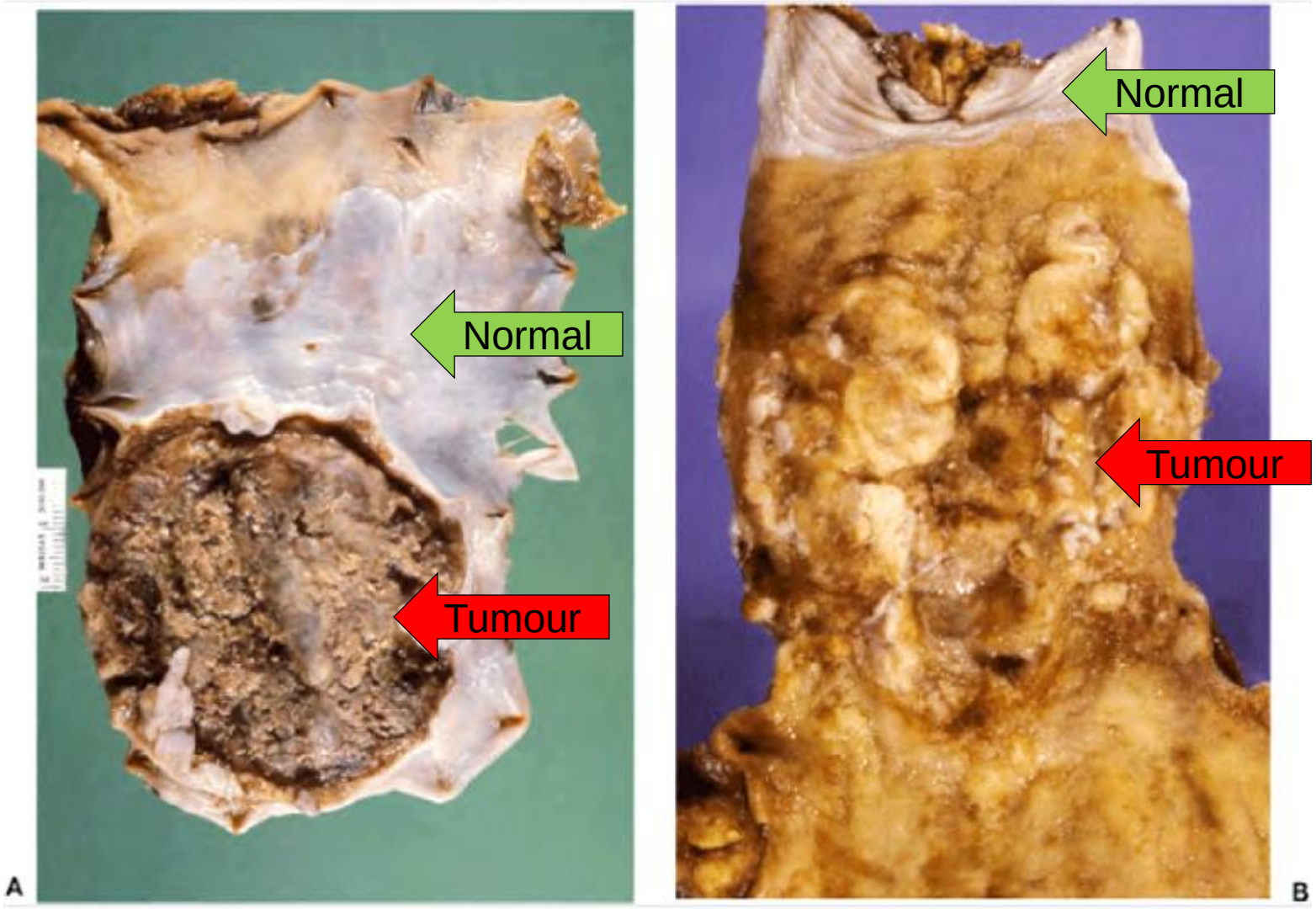
Basal cell hyperplasia







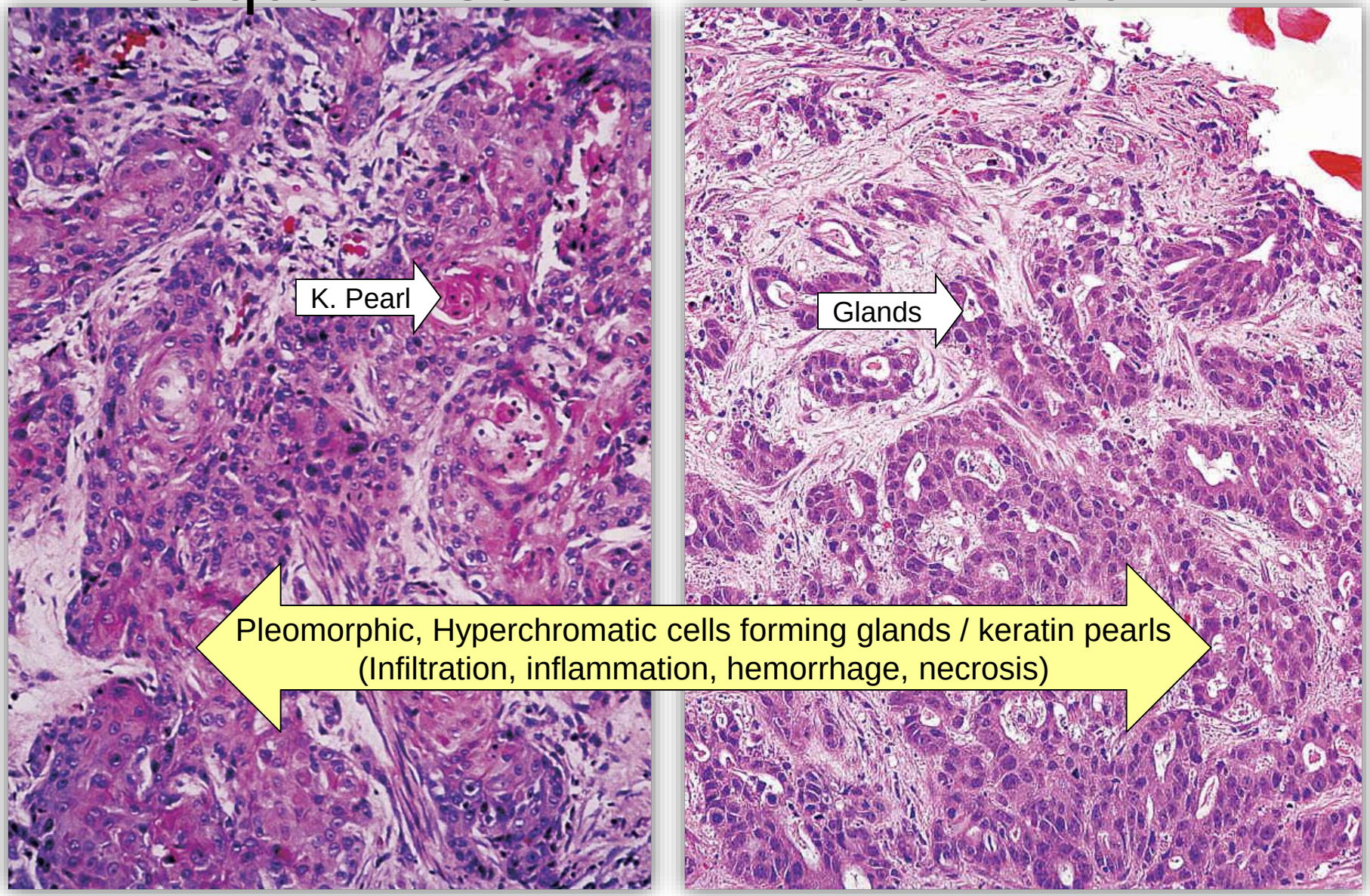
Squamous Carcinoma - Adenocarcinoma.

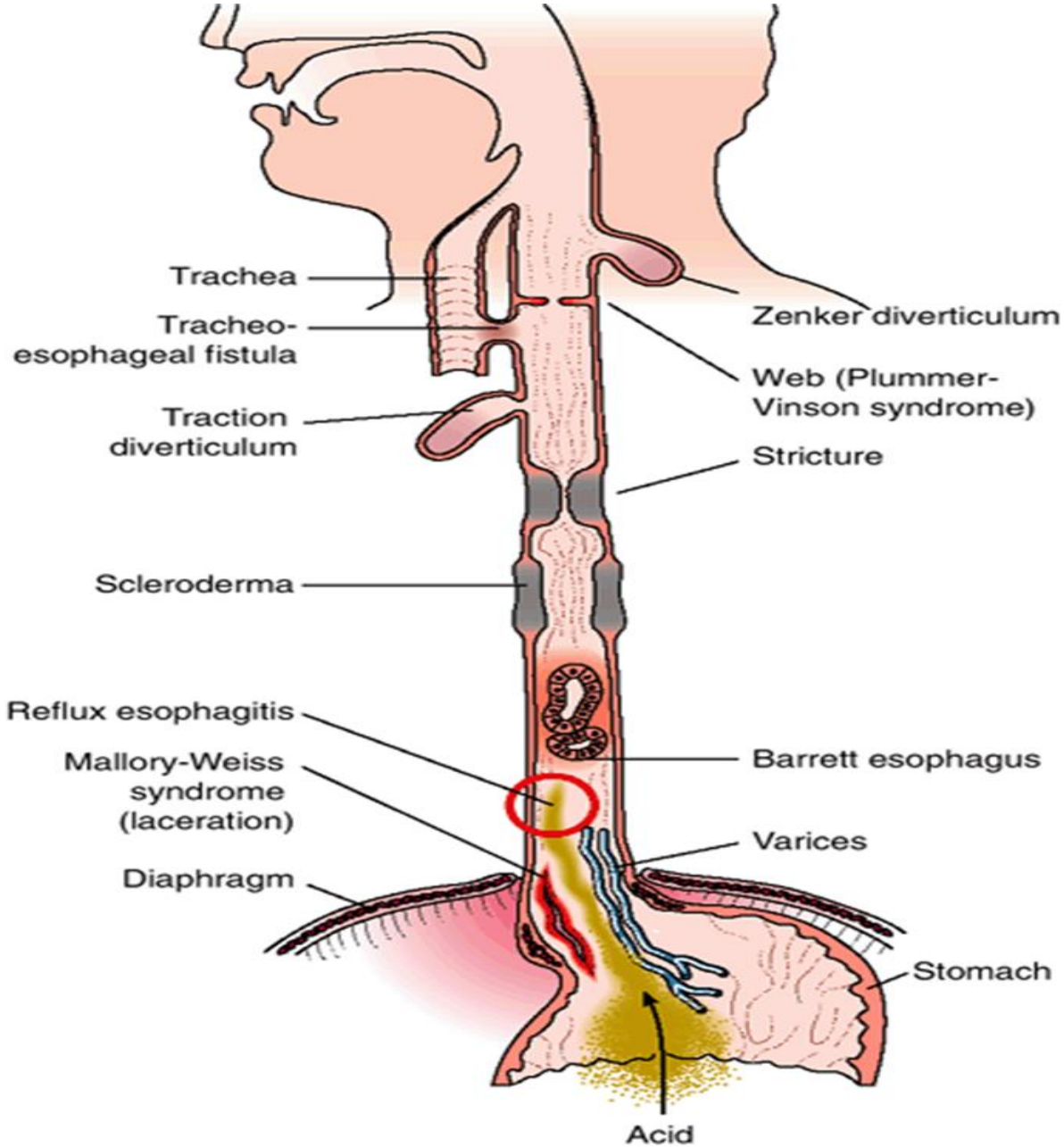


- More common
- Upper end
- Tobacco, diet, toxins.

- less common
- Lower end
- Reflux disease (Barretts)

Squam. Ca. - Adeno. Ca.




Trachea
Tracheo-esophageal fistula
Traction diverticulum
Scleroderma
Reflux esophagitis
Mallory-Weiss syndrome (laceration)
Diaphragm
Zenker diverticulum
Web (Plummer-Vinson syndrome)
Stricture
Barrett esophagus
Varices
Stomach
Acid

Tumors

Benign tumors:

e.g* leiomyoma (smooth muscle tumors)
is the most common tumor.

* Mucosal polyp

* Squamous cell papilloma

Malignant tumors:

Squamous cell carcinoma 90%

Adenocarcinoma, carcinoid, undifferentiated 10%

Squamous cell carcinoma:

Age : >50 years

Male/ female: 2:1-20:1

Geographically: most common in Iran, china, central Asia(?genetic,?certain food: very hot fluids)

Others: South Africa, Eastern Europe

- **Etiology:**

1- Dietary:

- Deficiency of vitamins & trace metals e.g vitamin A, C, Zinc.
- Contamination of food stuff with fungus e.g Aflatoxin .
- Nitrosamines and other nitrogenous compounds

2- Life style:

- Alcohol
- Tobacco

3- Esophageal disorder:

- Reflux esophagitis which may predispose to Barrett esophagus.
- Achalasia
- Plummer Vinson syndrome
- 4- Genetic predisposition

5-Thermal injury (eating hot food)

6-Viruses (HPV 16, 18)

Site:

50% occur in the middle third

30% occur in the lower third

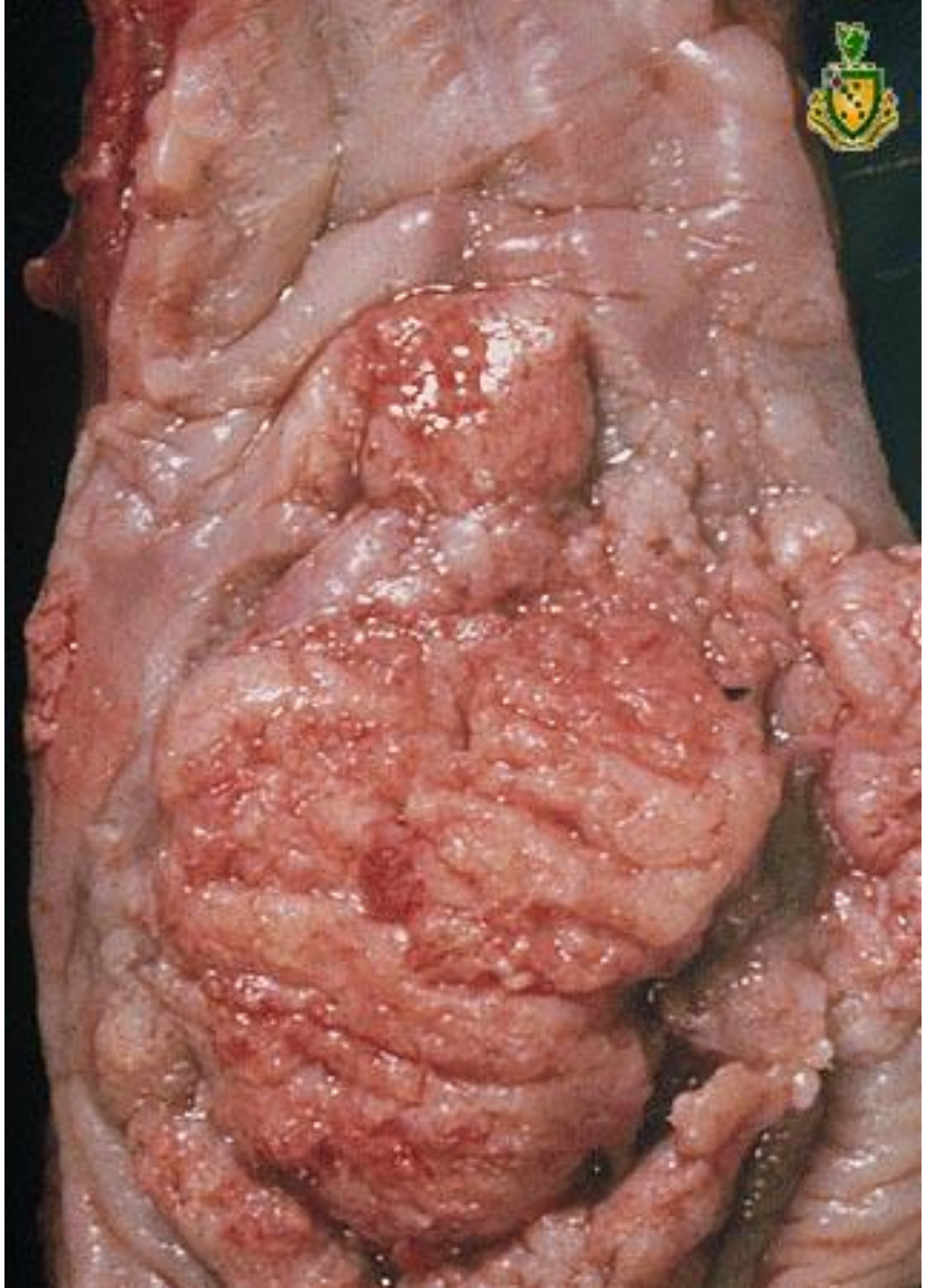
20% occur in the upper third

Gross appearance:

60% are polypoidal and may cause obstruction to the lumen

25% are ulcerative

15% may show diffuse thickening of the wall leading to its narrowing.

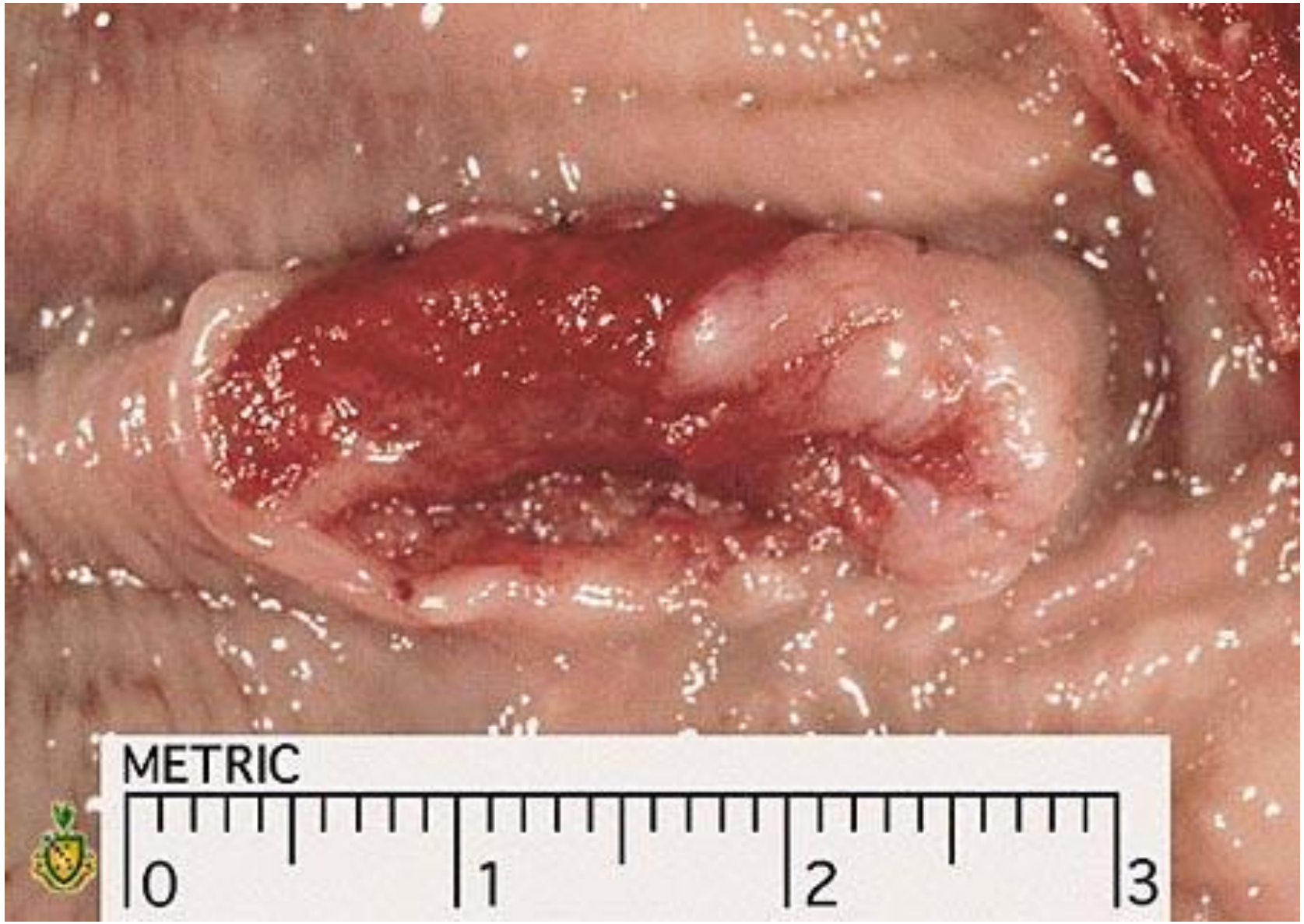


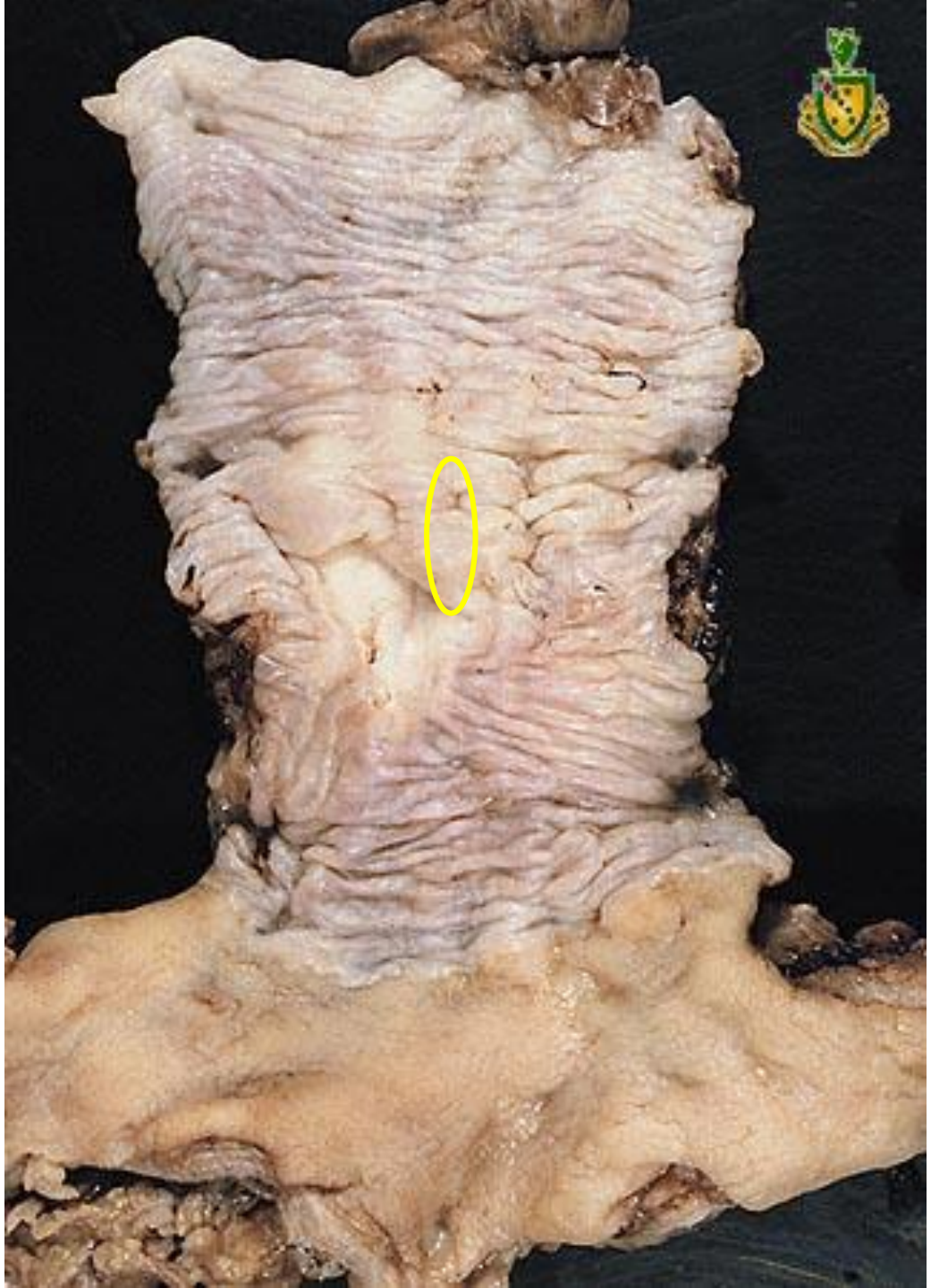
EXOPHYTIC

OR

FUNGATING CARCINOMA

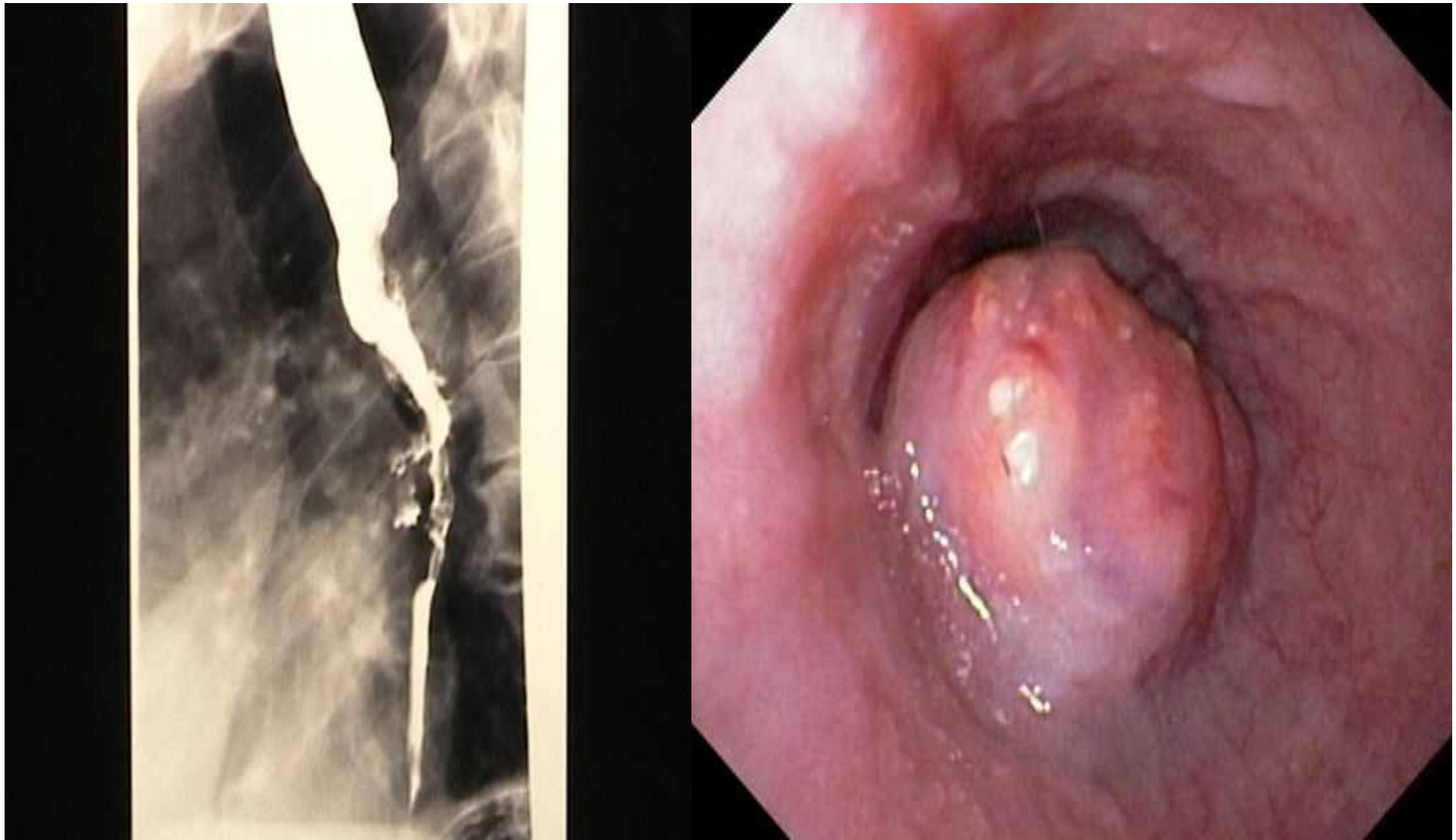
ULCERATING CARCINOMA





INFILTRATIVE PATTERN

Squamous cell carcinoma - Esophagus



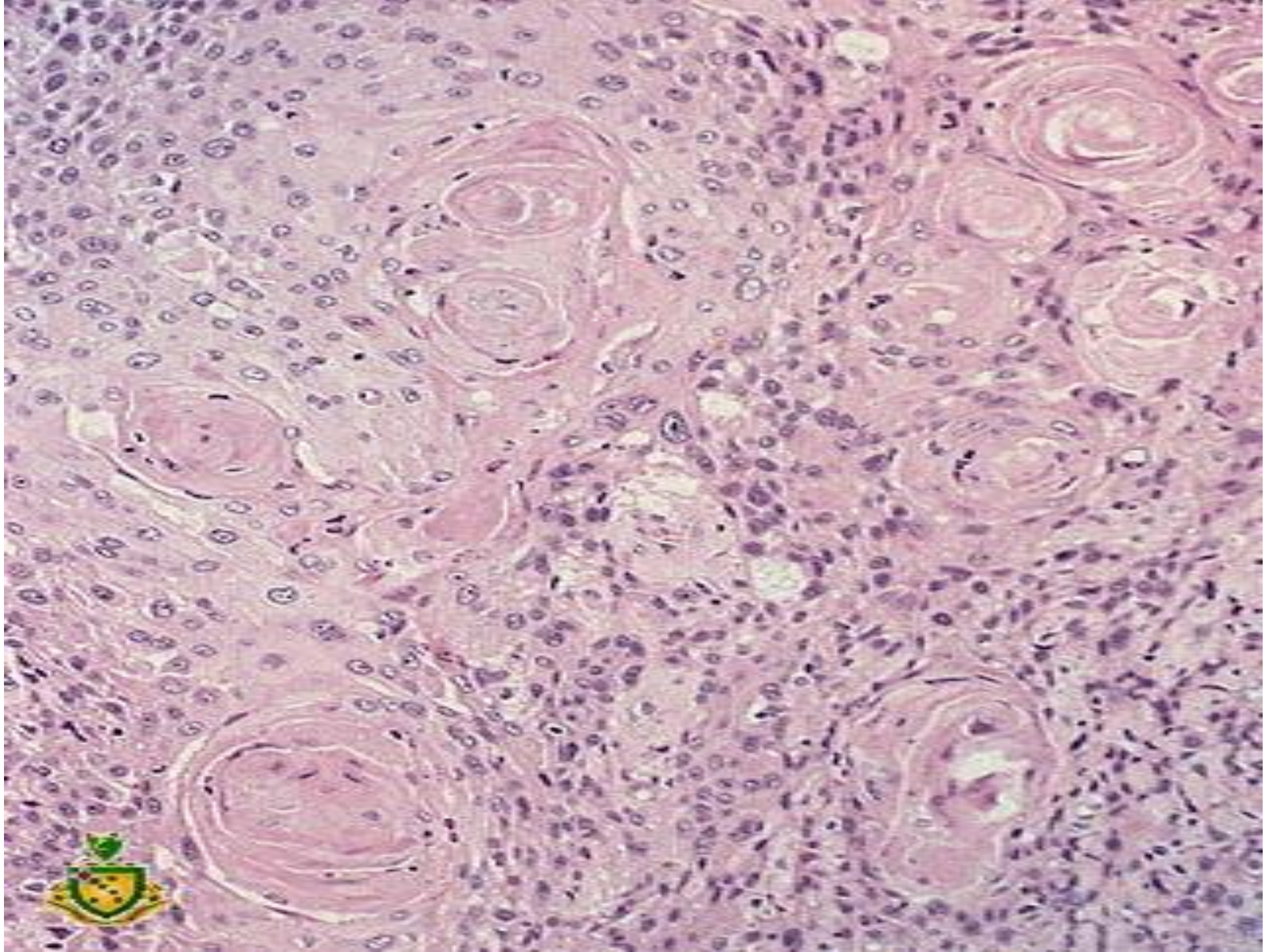
- **Microscopically:**

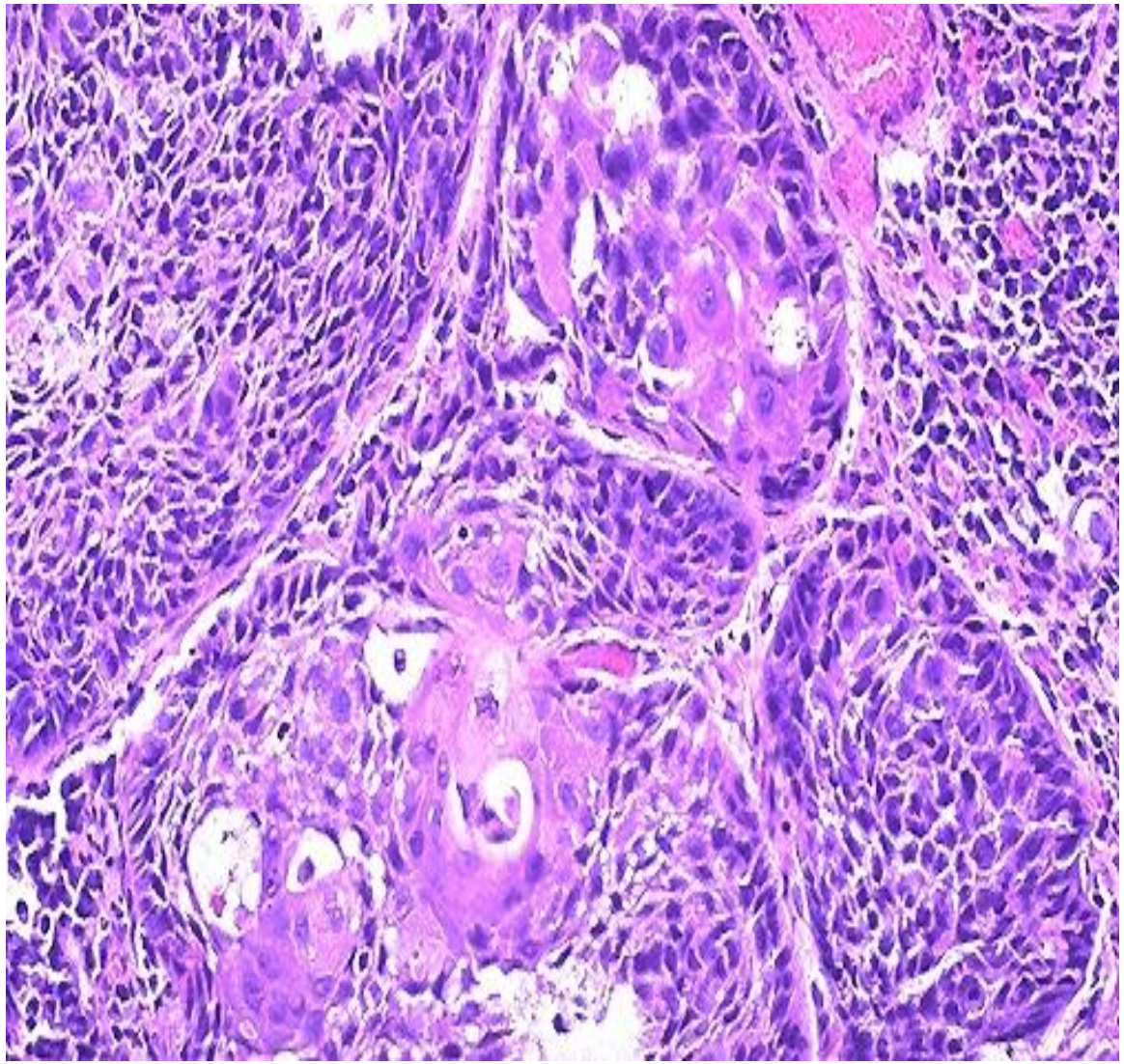
95% are squamous cell carcinoma

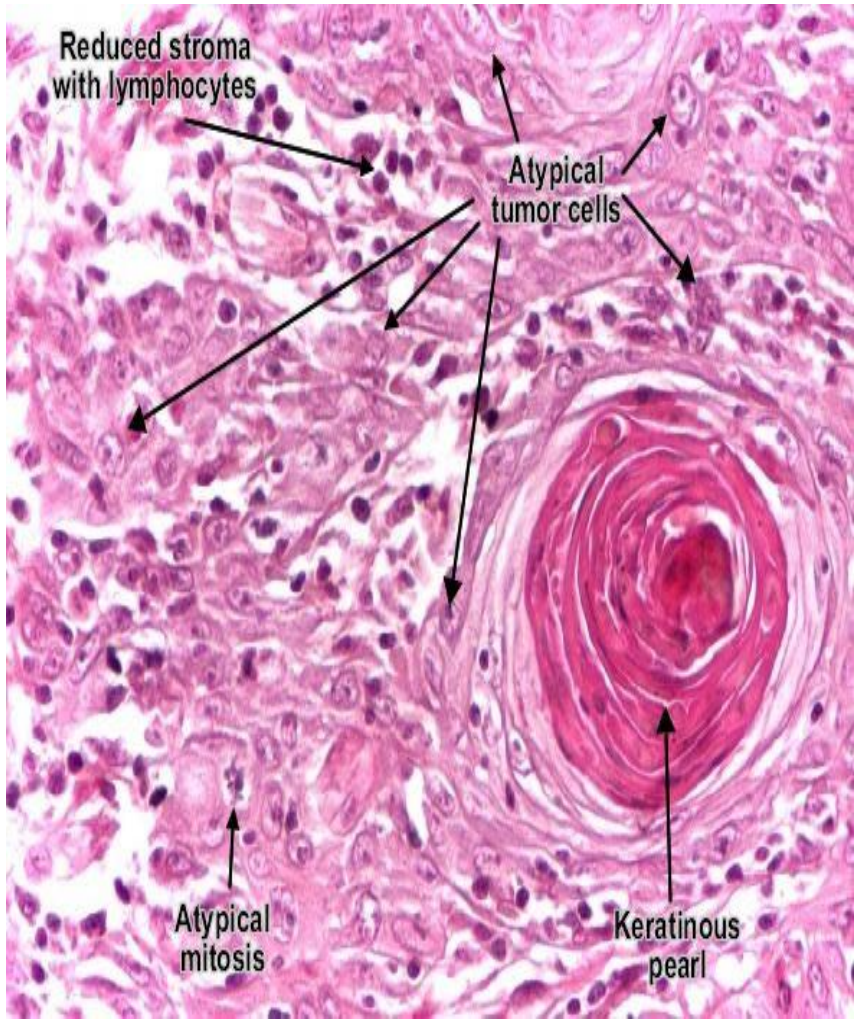
5% adenocarcinoma (lower 1/3)

metastasis to the lung , liver

…..etc.






Reduced stroma
with lymphocytes
Atypical
tumor cells
Atypical
mitosis
Keratinous
pearl

- **Spread:**

1- Local spread: it may invade the respiratory tree, aorta, pericardium, mediastinum(specially in the ulcerative type).

2- Lymphatic spread: to the regional lymph nodes

Rich lymphatic plexus and absence of serosa…easily metastasize

3- Hematogenous spread : distant organs

THANK YOU