**GIT PATHOLOGY Lec 2 Dr Methaq Mueen**

**Esophageal pathology**

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.

*Oesophageal****diverticula****are classified according to the mechanism of formation into:****traction diverticula****: occurs secondary to pulling forces on the outer aspect of the oesophagus.****pulsion diverticula****: occurs secondary to increased intraluminal pressure (e.g. Zenker****diverticulum***

**Types of esophageal diverticulae**:

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

a. upper, pulsion (Zenker diverticulum), located immediately above the upper esophageal sphincter (hypo pharyngeal 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).

**Mic.** Zenker diverticula are lined with stratified squamous epithelium with a thin lamina propria. No muscular layer exists. Fibrosis surrounding the diverticulum is common.

The typical symptom is regurgitation of food eaten some time previously (occasionally days), in the absence of dysphagia.

Recurrent aspiration pneumonia may be a serious complication. When symptoms are severe, surgical intervention is the rule.

b.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.

c. Epiphrenic diverticulum immediately above the LES.

 Causes:

*An epiphrenic diverticulum occurs from 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

 **Signs and symptoms:**

 \* Food regurgitation with no dysphagia

 \* Mass in the neck

 **Complications:**

 -Aspiration pneumonia

 -Perforation

***3-hiatus hernia***

It is a protrusion (herniation ) of the gastric segment above the diaphragm into the thorax.

* 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.

**Causes:**

1. Abnormal short esophagus.
2. Esophageal spasm and traction of the stomach.
3. Repeated episodes of increased intra abdominal pressure (coughing, vomiting, ---etc.)

**2-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.

 See the diagrams at the end of the lecture.

Complications of hiatal hernias include

1. Ulceration, bleeding and perforation (both types)

2. Reflux esophagitis (frequent with sliding hernias)

3. Strangulation of paraesophageal hernias

***4- Esophageal laceration***

 Also called **Mallory Weiss** syndrome .

* + - It consists of longitudinal tears in the esophagus at the gastroesophageal junction.
		- They are encountered most commonly in alcoholics,since they are susceptible to episodes of excessive vomiting.

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

prognosisThese tears are superficial and do not generally require surgical intervention; healing tends to be rapid and complete.

**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

Differential diagnosis upper GIT bleeding:

Esophageal varices

Bleeding gastric ulcer

Bleeding duodenal ulcer

Gastric leiomyoma

  **Esophagitis**

It is inflammation of the esophagus.

The causes are divided into:

1. infective
2. non infective

**Infective causes:**

1. ***Candida albicans:***

Especially in:\* chronic debilitating diseases,

\* Diabetes mellitus,

\* AIDS,

\* Patients taking cytotoxic drugs.

**Morphologicaly:**

The esophagus covered by adherent gray –white pseudomembrane.

1. ***Herpes simplex &cytomegaloviruses:***

Morphologically it produces punched out ulcers.

 ***3-Bacteria***:

 Accounts for 10-15% of infective esophagitis .

**Non infective causes:**

*1-* ***Acute esophagiti****s* caused by:

 \* Ingestion of mucosal irritants ( alcohol, corrosive acid or alkali, hot food and drink)

 \*Uremia (chronic renal disease)

 \* Drugs (tablets or capsules) when sticking in the esophagus

 \* Irradiation or chemotherapy.

1. ***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 factors:**

\* Decrease efficacy of the esophageal anti-reflux mechanism especially of the LES.

\* Presence of sliding hiatal hernia.

\* Slow esophageal clearance of the refluxed material.

\* Decrease gastric emptying.

\* Impaired reparative capacity of the esophageal mucosa by prolonged exposure to gastric juices.

**Clinical features:**

* occur usually at the age > 40 years
* dysphagia
* heart burn
* regurgitation

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.



**Tumors**

1. **Benign tumors:**

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

 \* Mucosal polyp

 \* Squamous cell papilloma

1. **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

 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 .

2-Life style:

* Alcohol
* Tobacco
1. Esophageal disorder:
* Reflux esophagitis which may predispose to Barrett esophagus.
* Achalasia
* Plummer Vinson syndrome
1. Genetic predisposition
2. Thermal injury (eating hot food)
3. 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. See the diagrams at the end of the lecture

**Microscopically:**

95% are squamous cell carcinoma

5% adenocarcinoma (lower 1/3)

**Spread:**

* 1. **Local** spread: it may invade the respiratory tree, aorta, pericardium, mediastinum (specially in the ulcerative type).
	2. **Lymphatic**: to the regional lymph nodes

***Rich lymphatic plexus and absence of serosa…easily metastasize***

* 1. **Hematogenous**: distant metastasis to the lung , liver …..etc.