GIT Pathology

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Esophageal pathology

The Esophagus

It is a muscular tube of 23-25cm, **its function** is to:
1-conduct food and fluid from the pharynx to the stomach
2- Prevent reflux of gastric content by the presence of two physiological sphincters:
* Upper esophageal sphincter (UES) at the cricopharyngeal muscle.
* Lower esophageal sphincter (LES) proximal to the esophageo-gastric junction.

Histologically:

1- Mucosa: lined by non-keratinizing squamous epithelium.
2- Lamina propria
3- Submucosa: containing glands
4- Muscularis propria: The upper fourth of the esophagus consists entirely of striated muscle, the next quarter contains a mixture of striated and smooth muscle, and the lower half consists entirely of nonstriated muscle.
5- No serosal coat.

Congenital anomalies:

Several congenital anomalies affect the esophagus The most important are esophageal atresia and tracheoesophageal fistula.

Atresia: a thin, noncanalized cord replaces a segment of esophagus, causing a mechanical obstruction (i.e. congenital absence of the normal canal (blind end of the canal).
Usually atresia is associated with fistula (tracheoesophageal)

Tracheo-esophageal fistula:
Abnormal connection will lead to the passage of food from the esophagus to the trachea leading to aspiration pneumonia and suffocation from food.

Acquired lesions:

1. Stenosis consists of fibrous thickening of the esophageal wall. Although it may be congenital, it is more frequently the result of severe esophageal injury with inflammatory scarring, as from gastroesophageal reflux disease (GERD), radiation, scleroderma and caustic injury. Stenosis usually manifests as progressive dysphagia, at first to solid food but eventually to fluid as well.

2.Esophageal mucosal webs: are small, thin, eccentric lesion composed of a fibrovascular connective tissue and overlying epithelial mucosal membrane projects into the esophageal lumen. These are most common in the upper esophagus. **The triad of upper esophagealweb, iron-deficiency anemia, and glossitis is referred to Plummer-Vinson syndrome.**

3.Esophageal (Schatzki) rings: unlike webs are concentric plates of tissue protruding into the lumen of the distal esophagus. Esophageal webs and rings are encountered most frequently in women over age 40. Episodic dysphagia is the main symptom**.**

Lesions associated with motor dysfunction:

Coordinated motor activity is important for proper function of the esophagus. The major entities that are caused by motor dysfunction of the esophagus are

1. Achalasia
2. Hiatal hernia
3. Diverticula
4. Mallory-Weiss tear

1- Achalasia: Greek: does not relax):

Failure of the lower esophageal sphincter to relax in response to swallowing leading to accumulation of food in the more proximal esophagus causing its dilatation & inflammation (esophagitis)

It is characterized by three major abnormalities:

1. Incomplete relaxation of the LES in response to swallowing
2. Increased resting tone of the LES
3. A peristalsis (failure of peristalsis)

symptoms:

The symptoms become manifested in young adulthood
a- progressive dysphagia for solid and then to fluid.
b- Regurgitation of undigested food.

Etiology:

1- Primary: degeneration of nitric oxide–producing neurons that normally induce lower esophageal sphincter relaxation.
2- Secondary:
A-Chagas disease caused by Trypanosoma cruzi parasitic infection lead to destruction of the myenteric plexus, failure of peristalsis, and esophageal dilatation. Duodenal, colonic, and ureteric myenteric plexuses can also be affected in Chagas disease.
B- achalasia-like disease include: diabetic autonomic neuropathy; infiltrative disorders such as malignancy, amyloidosis, sarcoidosis and systemic sclerosis.
3-Occasional coexistence of achalasia with other autoimmune diseases suggest immune-mediated destruction of inhibitory esophageal neurons.

Complications:

* Aspiration of undigested food which may cause pneumonia
* Candida esophagitis
*Lower esophageal diverticuli
* Squamous cell carcinoma develops in 5% of cases.

2- 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 gastroesophageal 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.)

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

2.Rolling (Para esophageal) 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 hernia)
3. Strangulation and infarction (Para esophageal hernia)

3- Diverticula:

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.

- They may develop in three regions of the esophagus

A- Zenker (pulsion) diverticulum: located immediately above the upper esophageal sphincter.

B-Traction diverticulum: near the midpoint of the esophagus

C-Epiphrenic diverticulum immediately above the lower esophageal sphincter.

Symptoms:

* Regurgitation of large amounts of food stored in the diverticulum during the day (occasionally for days) is typical with no dysphagia
* Mass in the neck

Complications:

-Aspiration pneumonia

-Perforation

4- 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 severe vomiting.

Clinical presentation: Patients often present with hematemesis.

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 upper GIT 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 is the usual outcome. Rarely esophageal rupture occurs.

Esophageal varices

- 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 plexuses 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.

DDX for massive Upper GI bleeding:

Esophageal varices
Bleeding gastric ulcer
Bleeding duodenal ulcer

Esophagitis

- This term refers to inflammation of the esophageal mucosa.
- The causes are divided into:

1- infective
2- non infective

- **Infective causes:**

1- Fungal: most common Candida albicans*:*

Especially in:
* Chronic debilitating diseases,
* Diabetes mellitus,
* AIDS,
* Patients taking cytotoxic drugs.

Morphologically:

The esophagus covered by adherent gray –white pseudomembrane.

2- Viral: Herpes simplex &cytomegaloviruses

Morphologically it produces punched out ulcers.

3- Bacterial: 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 and drink)
* Drugs (tablets or capsules) when sticking in the esophagus
* Irradiation or chemotherapy.

2- 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 (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.
* 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)
- dysphagia
- heart burn
- regurgitation of sour-tasting gastric contents.

Complications:

1-Bleeding 2– Stricture 3- 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 is 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:

* leiomyoma (smooth muscle tumors) is the most common tumor.
* Mucosal polyp
* Squamous cell papilloma

2.Malignant tumors:

*Nearly all esophageal cancers are either squamous cell carcinoma or adenocarcinoma. Squamous cell carcinoma is more common worldwide.

Squamous cell carcinoma:

- Age: >45 years
- Male/ female: 4:1
- Geographically: most common in Iran, china, central Asia. Others: South Africa, Eastern Europe

Etiology and pathogenesis:

- Factors associated with the development of Squamous Cell Carcinoma are classified as:

1. Dietary

- Deficiency of vitamins (A, C, riboflavin, thiamine, and pyridoxine) & trace elements (zinc)
- Contamination of food stuff with fungus e.g. Aflatoxin.
- High content of nitrites/nitrosamines

2. Lifestyle

- Burning-hot food
- Alcohol consumption and Tobacco abuse

3. Esophageal Disorders

- Achalasia
- Plummer-Vinson syndrome
- Caustic esophageal injury

4. Genetic Predisposition

5. Previous radiation to the mediastinum

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.

Microscopically:

- Squamous cell carcinoma composed of nests of malignant cells that partially recapitulate the organization of squamous epithelium

Spread:

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

2- **Lymphatic**: to the regional lymph nodes

Rich lymphatic plexus and absence of serosa…easily metastasize

3- **Hematogenous**: distant organs

Adenocarcinoma:

- Typically arises in the background of Barrett esophagus and long-standing GERD.
- Usually occurs in the distal third of the esophagus and may invade the adjacent gastric cardia.

❖ **Grossly:**

- As is the case with squamous cell carcinomas, adenocarcinomas initially appear as flat raised patches that may develop into large nodular fungating masses or may exhibit diffusely infiltrative or deeply ulcerative features.

❖ **Microscopically:**

- Most tumors are mucin-producing glandular tumors exhibiting intestinal-type features.