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Diseases of esophagus

Esophagus: is a tubular – muscular structure that has inner mucosal layer consists of stratified squamous epithelum , and outer muscular layer made of both striated and smooth muscle.

- striated mostly appears in the upper third of esophagus
- smooth muscle mainly in the middle and lower parts.

-we have two main layers of muscle depending on arrangement:
- longitudinal (outer)
- circular (inner)

- like any organ esophageal diseases might be benign or malginant (neoplastic)
We'll start with the benign (non-neoplastic) oesophageal diseases:

**Esophageal Obstruction**

- Structural (mechanical) obstruction

- Functional obstruction (disruption of the coordinated waves of peristaltic contractions)  
  ---------  Esophageal dysmotility

- The normal function of esophagus is to transmit food from the oral cavity to the stomach by the smooth muscle contraction (peristalsis), so any problem with the peristaltic contraction will produce esophageal obstruction.

  - Esophageal obstruction is two types:  
    1) structural (mechanical): there is space occupying lesion or mass that obstruct the esophagus.
Functional obstruction: esophageal dysmotility – esophageal motility disorder

- Esophageal dysmotility:
  - Nutcracker esophagus:
  - Diffuse esophageal spasm:
  - Lower esophageal sphincter dysfuncition/hypertensive lower esophageal sphincter
• **Nutcracker esophagus:**

- High-amplitude contractions of the distal esophagus---- contractions proceed in a coordinated manner
- dysphagia, chest pain
Nutcracker esophagus: (benign)
- the problem here we have high amplitude contraction in coordinated manner.

- dysphagia and chest pain are the main symptoms in all esophageal motility disorders.

Dysphagia: difficulty swallowing, and can be on both solid food and liquid material.
- it starts as dysphagia of solid food, if the disease is not treated and the condition get more sever the dysphagia will include the liquid material.

Chest pain: mainly present in the MI and angina so you should consider that there might be esophageal dysfunction.
**esophageal motility study** (esophageal manometry)

Esophageal motility studies involve pressure measurements of the esophagus after a patient takes a wet (fluid-containing) or dry (solid-containing) swallow. Measurements are usually taken at various points in the esophagus.

- Esophageal dysmotility syndrome is classified according to esophageal manometry.

  manometry: is used to measure the function of the lower esophageal function by measuring:
  1) lower esophageal sphincter pressure
  2) contraction amplitude
  3) contraction duration

solid and liquid food
we are considered with the abnormal results: (contraction >180, duration > 7)

* Diagram of esophageal motility study in nutcracker esophagus: The disorder shows peristalsis with high-pressure esophageal contractions exceeding 180 mmHg and contractile waves with a long duration exceeding 6 sec.

nutcracker esophagus will gives us: (abnormal results )
1) high amplitude contracting ( more than 180 )
2) duration of the contraction is prolonged ( 7 sec or more )

*increased lower sphincter tone is seen in all dysmotality disorders
• **Diffuse esophageal spasm:**
  - repetitive, simultaneous contractions of the distal esophageal smooth muscle —— uncoordinated contractions of the esophagus, contractions that are of normal amplitude
  - Dysphagia, chest pain

In here we have normal amplitude and duration of contraction but the contraction is uncoordinated also with dysphagia and chest pain.

• Esophageal manometry tracing demonstrates diffuse esophageal spasm. Note the multiple uncoordinated contractions in the third tracing from the distal esophagus.

- This image shows the multiple uncoordinated contraction.
Barium swallow: test used to determine the cause of painful swallowing or any difficulty by using barium sulfate which will be shown on X-Rays.

- diffuse esophageal spasm will be shown as corkscrew. And it's mainly present in the distal part of the esophagus.

- corkscrew appearance is the diagnostic form of the diffuse esophageal spasm
• **Lower esophageal sphincter dysfunction:**
  - such as high resting pressure or incomplete relaxation.
- Termed hypertensive lower esophageal sphincter in absence of altered patterns of esophageal contraction that seen in disorders described above.

- When there is high lower esophageal tone, but we don't have the other features.
esophageal dysmotility may result in development of diverticulae:

- **Epiphrenic diverticulum** (above the lower esophageal sphincter)

- **Zenker diverticulum/pharyngoesophageal diverticulum** (above the upper esophageal sphincter) if large accumulation of food mass, regurgitation and halitosis.

- uncoordinaton of smooth muscle contraction leads to diverticulum formation.

  Diverticulum: pouch or pocket of stretched or dilated tissue that develops along the esophagus, it's a protrusion of the mucosa and submucosa through a defect of the wall. Like hernia in the abdominal wall but internally.

  في أماكن بال خاصية اليفبدخل منها الأعصاب والأوعية الدموية déficit الجدار يكون ضعيف وسهل يصير فيه

- it's two types depending to the place .(on slide)

- the problem in the diverticulum that it leads to food impaction and regurgitation that causes accumulation of food and bad smell (halitosis).
**Mechanical obstruction:**
presents as progressive dysphagia that begins with inability to swallow solids - liquids.
can be caused by:
1- strictures/stenosis (chronic gastro-esophageal reflux, irradiation, caustic injury, cancer)
2- Cancer
3- Esophageal mucosal webs:
   - idiopathic
   - Paterson-Brown-Kelly or Plummer-Vinson syndrome — webs with iron-deficiency anemia, glossitis, and cheilosis
   - gastroesophageal reflux
   - chronic graft-versus-host disease
   - blistering skin diseases
4- Esophageal rings, or Schatzki rings

Mechanical obstruction means we have mass or true lesion that makes obstruction to esophagus.
- SCC might cause mechanical obstruction.
- Cancer might be adenocarcinoma (mass) or SCC
- Esophageal mucosal webs and esophageal rings are protrusions of mucosa with or without submucosa and muscular wall. Both happen in the upper and lower esophageal areas.
  - mucosa alone: esophageal mucosal webs
  - mucosa with submucosa and muscular layer: esophageal rings.

esophageal mucosal webs is an associated mass syndrome appears as (Paterson-brown Kelly), the patient comes with webs iron-deficiency anemia, glossitis, and cheilosis.
- mostly in females.
Achalasia is a functional obstruction of the esophagus, with chest pain, heart burn and dysphagia as the main symptoms.

- after examining the patient with manometry, you'll see this triad:
  1- incomplete LES relaxation upon swallow (<75%),
  2- increased LES tone ( >100 mm),
  3- aperistalsis of the esophagus. ( means no contraction at all, full relaxation and it's very important in diagnosis ).
Barium studies help in achalasia diagnosis, because of the LES tone is high and unable to relax, the esophagus will be dilated "any obstructed area will be dilated" >> bird's beak appearance < also this is a diagnostic feature.
Ganglions of the esophagus are found in two areas:
- submucosal plexus
- myenteric plexus

- In the primary achalasia there will be degeneration of ganglions that are responsible for relaxation for unknown reason.
- In Chagas disease the disease is generalized not only in esophagus (systemic infection).

**Primary achalasia:**
- The cause is unknown
- Is the result of distal esophageal inhibitory ganglion cell degeneration.
- Degenerative changes in the extraesophageal vagus nerve or the dorsal motor nucleus of the vagus may also occur.

**Secondary achalasia**
- May arise in Chagas disease, in which Trypanosoma cruzi infection causes destruction of the myenteric plexus.
- Other causes: diabetic autonomic neuropathy; infiltrative disorders such as malignancy, amyloidosis, or sarcoidosis; lesions of dorsal motor nuclei, particularly polio or surgical ablation; may also be driven by immune-mediated destruction of inhibitory esophageal neurons.
• Treatment modalities for both primary and secondary achalasia aim to overcome the obstruction, and include laparoscopic myotomy and pneumatic balloon dilatation. Botulinum neurotoxin (Botox) injection, to inhibit LES cholinergic neurons, can also be effective.

The treatment could be surgical (laparoscopic myotomy) or non-surgical.

**Lacerations**

**Mallory-Weiss tears:**
- mucosal tears at the gastroesophageal junction
- Most often associated with severe vomiting secondary to acute alcohol intoxication

**Boerhaave syndrome:**
transmural tear and rupture of the distal esophagus------- lethal

In the Mallory-Weiss tears only the mucosal and submucosal layers are defected. The severe vomiting leads to laceration in the lower esophagus. While in Boerhaave all 3 layers are included.
Esophagitis

- Chemical and Infectious Esophagitis

- Reflux Esophagitis

- Eosinophilic Esophagitis

Esophagitis: inflammation in esophagus

May be:

- 1) Chemicals like patients who take drugs like akamin durg (young patients who take akamin which is antibiotic)

There is places in esophagus that constrict and if there is impaction to a pill in these places > esophagitis

symptoms of esophagitis مع ال heartburn يمكن ييجي حدا عنده بتنصحه يشرب كميه كبيره من الماء مع حبة الدوا عشان ما يصيرلها esophagus بل impaction

- Infections like herpes virus, fungal esophagitis

2) reflux esophagitis: most common form

3) eosinophilic esophagitis >> caused by allergy
Reflex Esophagitis

- Reflex of gastric contents into the lower esophagus is the most frequent cause of esophagitis.
- The associated clinical condition is termed gastroesophageal reflux disease (GERD).

- Pathogenesis:
  - The most common cause of gastroesophageal reflux is transient lower esophageal sphincter relaxation mediated via vagal pathways.
  - Conditions that decrease lower esophageal sphincter tone or increase abdominal pressure and contribute to GERD include: alcohol and tobacco use, obesity, central nervous system depressants, pregnancy, hialtal hernia, delayed gastric emptying, increased gastric volume, coughing, and straining.
  - Reflux of gastric juices is central to the development of mucosal injury in GERD. In severe cases, reflux of bile from the duodenum may exacerbate the damage.

- The lower esophageal sphincter stays relaxed which cause regurgitation of gastric content (back to esophagus)

- There is some risks that may increase the reflux esophagitis but if the esophagus is normal it will not affect it these are :

  (alcohol, tobacco, obesity..(on slide) )
This picture shows the difference between GERD and eosinophilic esophagitis:

A: the first manifestation of gastric reflux in esophagus is the appearance of eosinophilic infiltration (eosinophilic inflammatory cells first then they will become lymphocytes) in lower esophagus

B: large amounts of eosinophilic infiltration in the middle and upper esophageal region
- **Clinical Features:**
  - GERD is most common in individuals older than age 40 but also occurs in infants and children.
  - The most frequent clinical symptoms are heartburn, dysphagia, and regurgitation of sour-tasting gastric contents.
  - chest pain
  - Complications: ulceration, hematemesis, melena, stricture development, and **Barrett esophagus**.
  - Treatment: proton pump inhibitors, H2 histamine receptor antagonists

It's not necessarily for the patient to come with heart burn, they may come with bitter taste in their mouths this is also a symptom (26;40 )

**Hematemeses:** upper GI bleeding (blood vomiting)

**Melena:** lower GI bleeding

Treatment: increase the activity of the stomach to release the gastric content
• **Hiatal hernia:**
  - can give rise to symptoms, such as heartburn and regurgitation of gastric juices, that are similar to those of GERD.
  - It is characterized by separation of the diaphragmatic crura and protrusion of the stomach into the thorax through the resulting gap.
  - Congenital hiatal hernias are recognized in infants and children, but many are acquired in later life.

Hiatal hernia : type of hernia >> protrusion of stomach and gastroesophageal junction > this may cause GERD

### Esophageal Varices

• Venous blood from the GI tract passes through the liver, via the portal vein, before returning to the heart.
• Diseases that impede this flow cause portal hypertension and can lead to the development of esophageal varices

- Esophageal varices: dilated mucosal or submucosal of the esophagus.
- GI blood will pass through the liver and portal circulation to detox the blood from the materials, anything that
causes block of the venous blood through this circulation will lead to portal hypertension.

- When the portal HTN is developed, blood will try to drain via collateral vessels (small vessels connect between large vessels like portal vein and GI vessels and the only work when you have venous obstruction)

- varices might lead to GI bleeding.

• 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.

- esophageal varices might be a consequence of portal HTN, that commonly due to liver cirrhosis .
- bleeding depends on how much varices are extended, they might be large bleeding, or minute bleeding.
• Clinical Features:
- 25-40% of patients with cirrhosis develop variceal bleeding.
- **Variceal hemorrhage is an emergency** that can be treated medically by inducing splanchnic vasoconstriction or endoscopically by sclerotherapy (injection of thrombotic agents), balloon tamponade, or ligation.
- Despite these interventions, 30% or more of patients with variceal hemorrhage die as a direct consequence of hemorrhage such as hypovolemic shock, hepatic coma, or other complications.
- more than 50% of patients who survive a first variceal bleed have recurrent hemorrhage within 1 year, and this carries a mortality rate similar to that of the first episode.
Barrett esophagus is a consequence of GERD, when there is metaplasia in the lining of the esophagus.

These patients treated prophylactically with beta-blockers to reduce portal blood flow and with endoscopic variceal ligation. It is important to recognize that cirrhosis patients with small varices that have never bled are at relatively low risk for bleeding and death.

Barrett Esophagus

- Barrett esophagus is a complication of chronic GERD that is characterized by intestinal metaplasia within the esophageal squamous mucosa.
- Occur in as many as 10% of individuals with symptomatic GERD.
- Most common in white males and typically presents between 40 and 60 years of age.
- The greatest concern in Barrett esophagus is that it confers an increased risk of esophageal adenocarcinoma.
- The presence of dysplasia, a preinvasive change, is associated with prolonged symptoms, longer segment length, increased patient age, and Caucasian race.

- Barrett esophagus is a consequence of GERD, when there is metaplasia in the lining of the esophagus.
- The normal lining of esophagus is stratified squamous, when there are goblet cells developed in the lining of intestinal metaplasia.
- Has the same of symptoms of GERD.
- If the symptoms are prolonged, you do an endoscopy.
- We do something called observation therapy, which is watching the patient each 6 months – to make sure there is no cancer formation (adenocarcinoma).

*Clinical Features:*

**Barrett esophagus can only be identified thorough endoscopy and biopsy, which are usually prompted by GERD symptoms.**
**Esophageal Tumors**

- The vast majority of esophageal cancers fall into one of two types:
  1. adenocarcinoma
  2. squamous cell carcinoma

Squamous cell carcinoma is more common worldwide.

Esophageal tumors may be:

1) Benign: includes the lining of esophagus like liomyoma (smooth muscle tumor come from the muscle wall of esophagus)
2) Malignant: (which we'll take about)
   2 types: adenocarcinoma and SCC
   The SCC is the most common form of malignant tumor of esophagus
Adenocarcinoma

• Most esophageal adenocarcinomas arise from Barrett esophagus.
• increased rates of esophageal adenocarcinoma

risk factors:
- gastroesophageal reflux/Barrett esophagus
- tobacco use
- Exposure to radiation

risk is reduced by:
- diets rich in fresh fruits and vegetables.
- Some serotypes of Helicobacter pylori

Adenocarcinoma occur in lower esophagus as a progression of barret esophagus

- the tobacco and alcoholism risk factors affect SCC more than adenocarcinoma

-some types of h.pylori increase HCL secretion and some decrease here we mean the h.pylori that decrease the HCL
• occurs most frequently in Caucasians and shows a strong gender bias, being sevenfold more common in men.

• **Pathogenesis:** Molecular studies suggest that the progression of Barrett esophagus to adenocarcinoma occurs over an extended period through the stepwise acquisition of genetic and epigenetic changes.
Clinical Features:
- pain or difficulty in swallowing, progressive weight loss, hematemesis, chest pain, or vomiting. Occasionally discovered in evaluation of GERD or surveillance of Barrett esophagus.

As a result of the advanced stage at diagnosis, overall 5-year survival is less than 25%.

Squamous Cell Carcinoma

- Risk factors include:
  - Alcohol and tobacco use (polycyclic hydrocarbons, nitrosamines)
  - Poverty
  - Caustic esophageal injury
  - Achalasia
  - Plummer-Vinson syndrome
  - Diets that are deficient in fruits or vegetables
  - Frequent consumption of very hot beverages
  - Previous radiation to the mediastinum
  - HPV infection has also been implicated in esophageal squamous cell carcinoma in high-risk areas
  - Fungus contaminated foods

More common in upper and middle esophagus

And more in males
• Clinical Features:

- The onset of esophageal squamous cell carcinoma is insidious and most commonly presents with dysphagia, odynophagia (pain on swallowing), or obstruction.

- Weight loss

- Hemorrhage and sepsis may accompany tumor ulceration,

- Occasionally, the first symptoms are caused by aspiration of food via a tracheoesophageal fistula.

- The overall 5-year survival rate in the United States remains less than 20%, and varies by tumor stage and patient age, race, and gender.