Esophageal Disease

No.25

Editing file

Objectives :

- ★ Describe the major components in the anatomy of the esophagus and physiology of swallowing.
- ★ Explain the pathophysiology and etiology of common esophageal disorders.
- ★ Name the common presenting symptoms and complications of reflux disease (GERD).
- ★ List the main points in the management of GERD.
- ★ Differentiate between oropharyngeal and esophageal dysphagia.
- ★ Explain the differences between anatomical and functional esophageal disorders.
- ★ Explain the pathophysiology of common causes of dysphagia.
- ★ List the common presenting symptoms, appropriate investigations and treatment options in different causes of dysphagia.

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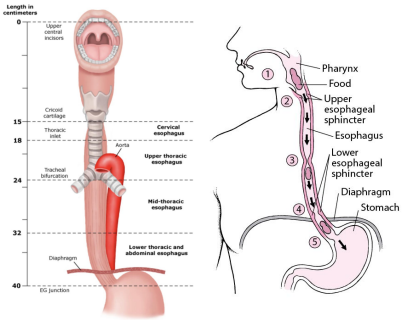
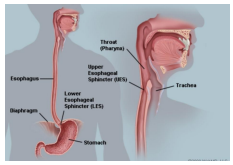
Introduction

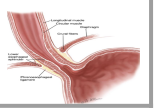
◀ Anatomy of the esophagus

- Muscular tube 20-25 cm long, located between the pharynx at the level of C6 and the stomach at the level of T11 and it crosses the diaphragm at T10 through the esophageal hiatus.
- it has an **UES & LES** that help empty food between swallows while preventing regurgitation of stomach contents
- The oesophagus is lined by **stratified squamous epithelium**, which extends distally to the squamocolumnar junction where the oesophagus joins the stomach.
- striated muscles make up the proximal ⅓.
- smooth muscles make up the distal ⅔.
- **how long does it take a bolus to pass the entire esophagus?**
  - **about 7-10 seconds.**



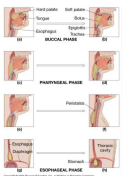


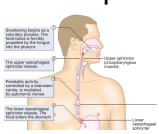
◀ Esophageal sphincters

Upper esophageal sphincter	Lower esophageal sphincter[1,2]
Functions to prevent **regurgitation** into oral cavity and larynx , prevent aspiration	A physiological sphincter comprised of **smooth muscles**
Restricts airflow into the esophagus during inspiration. When we take a breath UES will close so that all the air will go into the lungs rather than entering the esophagus.	Normally located within the diaphragmatic hiatus with ⅔ in the abdominal cavity and ⅓ in the thoracic cavity. **Can be displaced proximally by hiatus hernia[3]** (discussed later)
Composed of **striated muscles** and are under conscious control, used when breathing, eating, belching, and vomiting.	It maintains a high pressure zone between stomach and esophagus **(barrier to reflux)** 

◀ Physiology of swallowing[4]

UES opens and bolus is introduced into the esophagus. esophagus distends causing a contraction proximal to distal. propagating sequence.





Primary peristalsis: initiated in the pharynx **by a swallow**. the waves are slow moving and sweep the entire length of the esophagus.

Secondary peristalsis: initiated **by esophageal distention** by food. these waves are important to remove all the food from the esophagus if it has not been totally cleared by primary peristalsis

LES open at the end of swallowing to allow the food to pass to the stomach

Then it closes immediately to prevent food from coming back into the esophagus

1- not a true sphincter, **the diaphragmatic muscles (striated) contribute to the LES with the esophageal muscles (smooth).** ★

2- This sphincter remains tonically constricted (protects the esophagus from the stomach acidic juices) until the peristaltic swallowing wave passes down the esophagus and causes a "receptive relaxation" of the sphincter and emptying of the propelled food into the stomach.

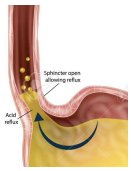
3- A hiatal hernia is when your stomach bulges up into your chest through an opening in your diaphragm.

4- Swallowing process starts in the mouth after chewing the food, the tongue moves against the palate pushing the bolus down into the pharynx, once the food reaches the pharynx the UES senses that and opens allowing the bolus to move down into the esophagus. When the bolus is inside the esophagus UES closes immediately. Then primary "main" peristalsis starts which is a continuation of the pharyngeal peristalsis and when it ends with a clear esophagus we are done, but whenever there is a remnant food or fluid after the primary peristalsis the secondary peristalsis takes place. What stimulates the secondary peristalsis? The pressure exerted by food into the esophageal wall.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

◀ Introduction

- Symptoms or complications resulting from the **reflux of gastric contents** into esophagus or beyond, into the oral cavity (including larynx) or lung, most common esophageal disorder
- **Transient lower esophageal sphincter relaxations (TLESRs[1])** are part of **normal physiology,** but occur more frequently in patients with GERD, allowing gastric acid to flow back into the oesophagus.
- Prevalence in Saudi Arabia **is 45%-50%**.
- **What causes GERD? LES problems**
  - **Change in its location: Hiatal hernia**
  - **Hypotensive LES: because of some foods like(spicy, acidic food, caffeine, soda, chocolate) , drugs**
  - **Increase Intra-abdominal pressure: (eg: Pregnancy, obesity, large meals, tumor,persistent cough),** LES can not overcome this increase in pressure



◀ Factors associated with GERD

1. **Pregnancy or obesity**
2. Fat, chocolate, coffee or alcohol ingestion, Large meals
3. **Cigarette smoking**
4. Drugs – antimuscarinic, calcium-channel blockers, nitrates and any drugs cause dilation of LES
5. **Hiatus hernia,** Systemic sclerosis
6. Treatment for achalasia

◀ SYMPTOMS OF GERD

Typical	★ **Heartburn is the major feature**. This is a burning chest pain that is **aggravated by bending, stooping and lying down**, all of which promote acid exposure and improved by sitting up due to gravity because gravity pulls the acid from the esophagus to the stomach. • **Regurgitation** of food and acid into the mouth occurs, **particularly on bending or lying flat.** This can lead to excess salivation in the mouth, commonly known as **water-brash, and need to swallow again**
Atypical	**Chest pain**, Early satiety, Nausea, Bloating, belching and **Globus sensation**[2]
ExtraEsophageal[3]	Cough, Nocturnal Asthma, Laryngitis, Sinusitis/recurrent otitis media, Dental erosions

◀ Classic features of GERD and cardiac ischaemic pain

★ **A cardiac cause should be excluded in patients with chest pain before starting GI evaluation[4].**

Reflux Pain	Cardiac Ischaemia Pain
Rarely radiates to the arms	Gripping or crushing, Radiates to neck or left arm
Worse with **spicy food, hot drinks or alcohol**	Worse with exercise
Relieved by **antacids**	Accompanied by dyspnoea

1- In TLESRs the LES sphincter relax 3-6 time every hour for 3-10 sec to allow gases to go out of the stomach, otherwise bloating will occur (Considered as a protective mechanism
2- Very common, pt words "I feel something is stuck in my throat, and isn't relieved by eating or drinking" . It's a sensation caused by the irritation of the esophagus.
3- Happens when the reflux is so severe going beyond the esophagus.
4- All patient with chest pain must undergo cardiac evaluation first (ECG) before doing any GI evaluation. Even if they present with very suggestive signs and symptoms of GERD
-When the patient comes with typical symptoms; heartburn, regurgitation he mostly has GERD
But when come with atypical or extraEsophageal symptoms, he probably has GERD means it is a 50% 50% chance.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

◀ HOW TO DIAGNOSE GERD?

- The clinical diagnosis can usually be made **without investigation** and is often made using mainly:
  - **Typical symptom presentation.**
  - **Antisecretory responsiveness.** **(PPI responsive for 2 months and then see)**
- **In some cases you will need to go further with:**

Endoscopy

indications for endoscopy in suspected GERD:

1. **Alarm symptoms, like?**
   - Dysphagia, Weight loss, Positive family or personal history of esophageal malignancy
   - Hematemesis, Melena, Anemia.
2. **Non-Cardiac chest pain**
3. Screening **high risk patients for Barrett's:** overweight, white males, older than 50, chronic GERD (>7 y), smokers, Family history
4. Patients that are **unresponsive to PPI** or if they initially has response then relapse.

Ambulatory reflux monitoring (PH study[1])

Indications for ambulatory PH monitoring:

1. Suspected GERD with **normal Endoscopy** (to confirm GERD), 60% of patients will have negative endoscopy because the level of reflux wasn't enough to cause ulcers that we can see.
2. Persistent symptoms even with PPI[2] (To R/o other causes)

◀ MANAGEMENT OF GERD

Lifestyle changes	• Weight loss, Head of bed elevation at night, Smoking Cessation, eat small meals • Avoidance of meals 2-3 hours before bedtime for patients with nocturnal GERD. • Culprits: fatty foods, caffeine, chocolate, ETOH, spicy foods, carbonated beverages, peppermints, don't give the patient a list of food and drinks, just avoid what aggravates his condition
Medical Usually we do lifestyle changes & medical	★ **Proton pump inhibitors (PPIs)** are the most commonly used medications, **usually effective in resolving symptoms and healing esophagitis.** • **domperidone**, when dysmotility features are prominent, can be helpful. • **antacids** and **alginates** can also provide symptomatic benefit. • **H2-receptor antagonist** drugs, helpful in resolving symptoms without healing esophagitis.

Surgical	• **Fundoplication[3]: take a part of the stomach and wrap it around LES**, which can treat both hiatal hernia and GERD, the Indications for Fundoplication are:
	  ○ **Persistence reflex even with PPI's**
	  ○ **Barrett's esophagus**
	  ○ **The Patient doesn't need to continue his life with PPI**

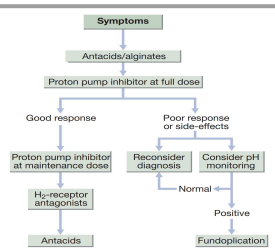
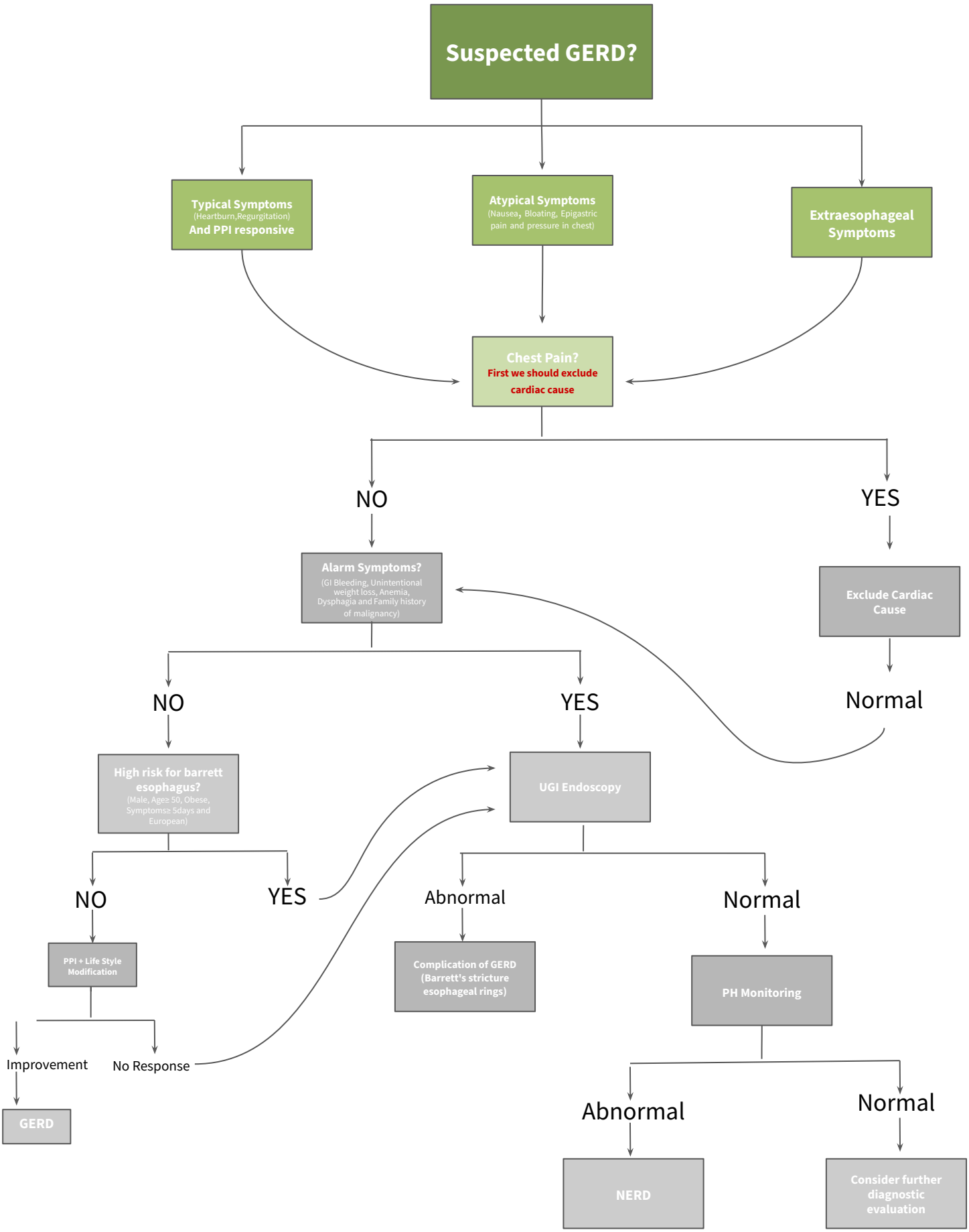


Fig. 21.30 Treatment of gastro-oesophageal reflux disease: a 'step-down' approach.

1- PH study is never done before endoscopy.
2- Patients with functional esophagitis: the esophagus is hypersensitive and gets irritated from anything, not necessarily acid. We monitor the pH to know the nature of the reflux causing the irritation
3- An antireflux procedure in which the gastric fundus wrapped around the lower esophagus results in a narrowing of the distal esophagus -> preventing reflux. Patients undergoing Fundoplication must be evaluated first by endoscopy to make sure they have a reflux. If normal, do PH monitoring.
Barium swallow has no role in GERD diagnosis.

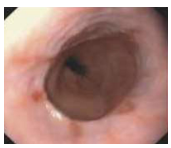
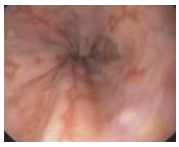
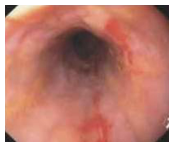
GASTROESOPHAGEAL REFLUX DISEASE (GERD)

◀ Extra



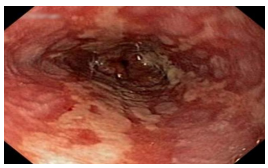
GASTROESOPHAGEAL REFLUX DISEASE (GERD)

◀ Complications

01 Erosive Esophagitis:

- AKA Ulcerative esophagitis, if left untreated it will lead to **Barrett's Esophagus**.
- **Treatment** : proton pump inhibitors (PPIs)

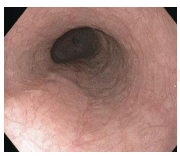


02 ★ Important

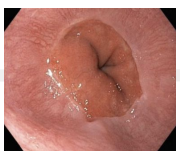
Barrett's Esophagus: not common about 2% of GERD patients[1]

- **Metaplastic** columnar epithelium (gastric and intestinal) replaces the stratified squamous epithelium in the distal esophagus in order to accommodate the acids, it's More common in Men.
- **Risk Factors for Barrett's :**
  - Chronic (>5 years) GERD symptoms, Advancing age (>50 years).
  - Male gender, Caucasian race, Family history
  - **Tobacco usage**, Central obesity.
  - **Alcohol doesn't increase risk of Barrett's**
- **Diagnosis:** endoscopy and biopsy
- Barrett's itself isn't a cancer
- The risk of **cancer** seems to relate to the **severity and duration of reflux** rather than the presence of Barrett's oesophagus per se.

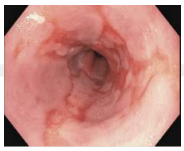
When left untreated



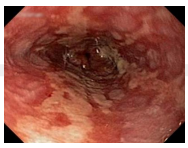
Normal esophagus



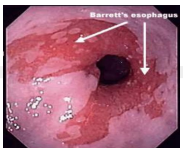
Normal transitional zone between the esophageal muscles "light pink" and gastric muscles which can tolerate acids



Uncomplicated GERD: edema, loss of vasculature, linear erythema

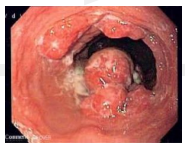


Esophagitis ulceration + exudate



Barrett's: metaplasia[2]
Benign, the cells of the stomach start migrating the esophagus

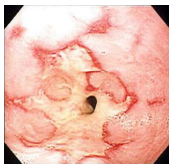
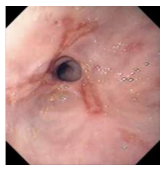
If Barrett's not treated> Low grade dysplasia, if not treated> High grade dysplasia: change from benign to malignant



Adenocarcinoma[3]

03 Peptic Stricture: Causes narrowing of the lumen

- They usually occur in patients **over the age of 60** and present with **intermittent dysphagia for solids**, which worsens gradually over a long period.
- Mild cases may respond to **PPIs alone**.
- More severe cases need **endoscopic dilation** and **long-term PPI therapy**.
- **Surgery** is required if medical treatment fails.

A lot of inflammation going on → Fibrosis→ Benign stricture "reversible by controlling the reflux first, then endoscopic dilatation"

1- The incidence of barrett's is very low, that's why we don't screen every patient with GERD, unless if he\she has multiple risk factors
2- PPi's have the capability to stop the progression of Barretts's metaplasia to dysplasia, But has no benefit once patients develop dysplasia (even low grade).
3- A patient has cancer in the lower esophagus, what is the most likely type ? Adenocarcinoma
What is the most likely risk factor ? Barrett's esophagus secondary to GERD

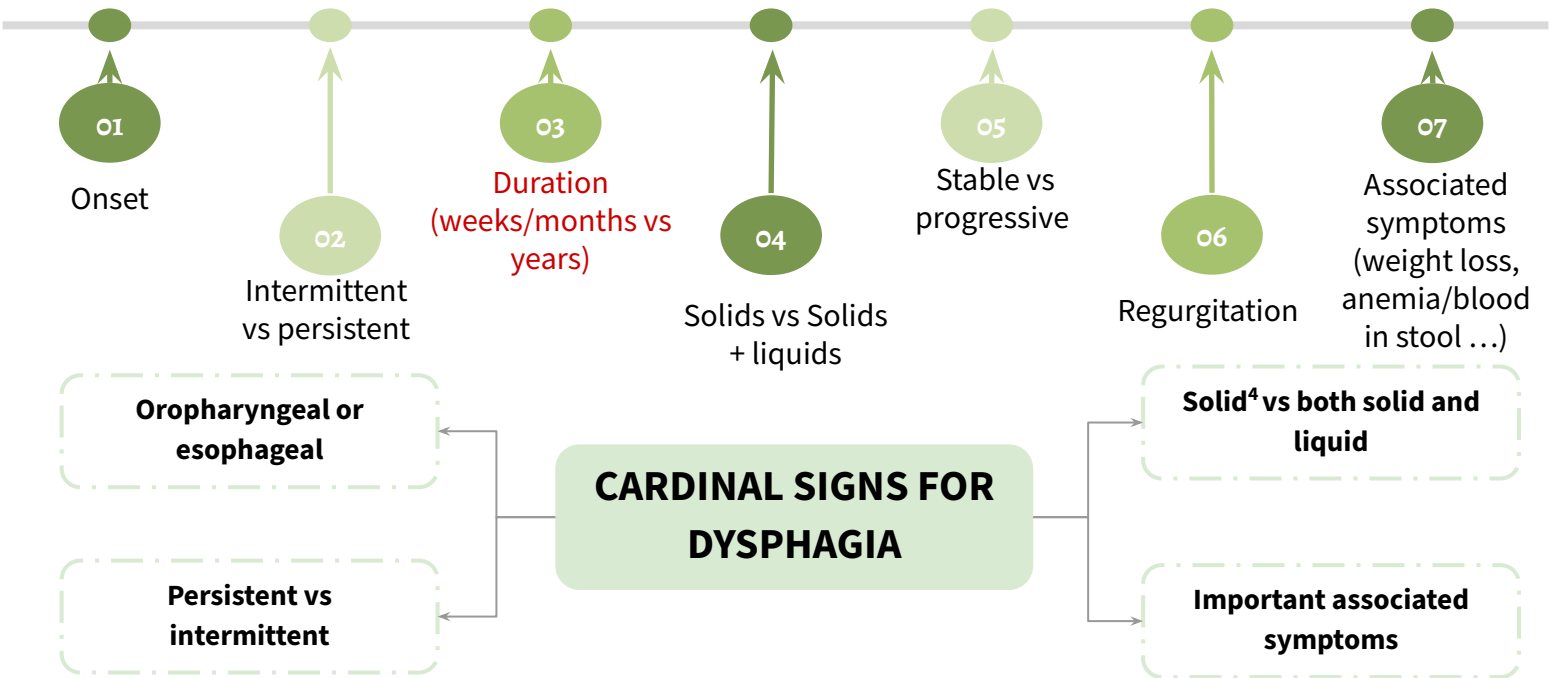
DYSPHAGIA

Introduction

- Subjective sensation of difficulty or abnormality of swallowing.
- **Oropharyngeal vs Esophageal dysphagia[1].**

Oropharyngeal Dysphagia	Esophageal Dysphagia [2]
Recurrent Pneumonia (Aspiration)	
Weight loss	
Coughing or choking with swallowing.	**Usually no problem with initiation of swallowing.**
Difficulty initiating swallowing.	Sensation of food getting **Stuck in the chest.**
Change in **Voice** or **Speech**	Can have **Pain in the chest** from food bolus.
Nasal regurgitation.	**Oral** or **Pharyngeal** regurgitation.
Systemic **Neurologic** (Such as Stroke) or **Myopathic** (Such as dermatomyositis) Syndromes. Because it's a striated voluntary muscles	**Functional** or **Anatomical** abnormalities in the esophagus **Functional causes:** • Achalasia. • Secondary dysphagia.[3] • Esophageal spasm. • Eosinophilic esophagitis. **Anatomical causes:** • Esophageal strictures • Esophageal ring. • Esophageal web. • Malignancy.

HISTORY TAKING IN DYSPHAGIA



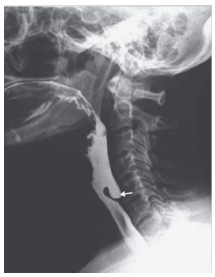
1- it's very imp to differentiate between them, we should ask about it in history taking then continue with usual HPI
2-In esophageal dysphagia, function refers to the "movement" While anatomy refers to obstruction caused by cancer/strictures
3- Secondary to rheumatological diseases as in scleroderma; which affects smooth muscles including esophageal muscles impairing their movement
4- Solids goes more with **anatomical** causes which make the esophageal lumen narrow allowing only fluids to pass through, while when they have solid and fluid dysphagia it means there are a **functional "motility relate"** cause so esophagus can't deliver both, if the patient said it was initially solids but now even liquids it is **anatomical** cause also.

Oropharyngeal Dysphagia

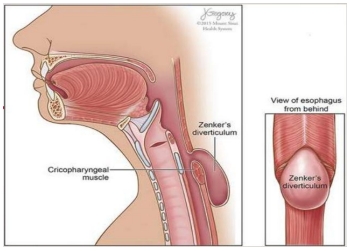
◄ Zenker's diverticulum

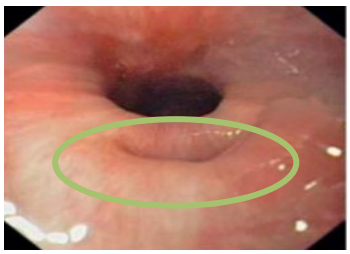
Introduction	• AKA **cricopharyngeal diverticulum**, Sac like, outpouching of the mucosa and submucosa in the area of **muscular weakness**[1] in the hypopharynx between the inferior constrictor and cricopharyngeus muscle **above the UES.** • An area of weakness known as **Killian's dehiscence** allows a pulsion diverticulum to form.
Cause[2]	• **Hypertensive / noncompliance of the Upper esophageal sphincter**
Symptoms[4]	• **Oropharyngeal dysphagia symptoms.** • **Food undigested on the pillow at night or after meals.** • **Halitosis**[3]. • **Weight Loss** happens in late stages (the diverticulum becomes big & obstructs the esophagus), so becoming oropharyngeal & esophageal • Occasionally, patients present with **recurrent pneumonia** following aspiration of food into the trachea. • They may also complain that a **gurgling sound** is heard in the neck following a swallow as liquid and food collect in the pouch.

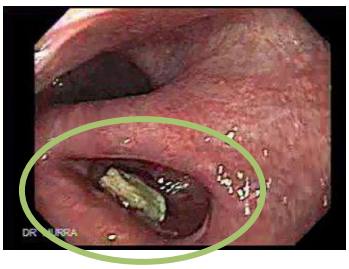
- **Diagnosis:**
  - **Video swallow testing**
  - **Modified barium swallow.**
  - **Endoscopy may be hazardous**, since the instrument may enter and perforate the pouch.(Never do)



- **Treatment:**
  - **Surgical**, either via an external approach through the neck where the pouch is excised or, **more commonly, via endoscopy with stapling of the party wall**



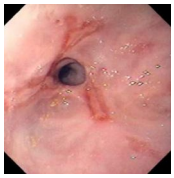




1- The disease is common among elderly cause muscles become even weaker with age
2- Tense UES → they are pushing the bolus against a closed sphincter → a high pressure will be exerted on the pharyngeal wall causing the weak area to protrude and form a sac where food and fluid accumulate
3-Halitosis is considered as a "**characteristic feature**" of Zenker's diverticulum that every physician must keep an eye on secondary to retention of undigested food.
4- In late stages of zenker's diverticulum, Patients may present with symptoms of both oropharyngeal and esophageal dysphagia as it begins to obstructs the esophagus.

Esophageal Dysphagia

◀ 1- ESOPHAGEAL STRICTURE



Causes:

- Benign peptic stricture caused by GERD
- Malignant stricture
- Iatrogenic? eg: Suicidal ingestion of acids, or radiation therapy.

Diagnosis:

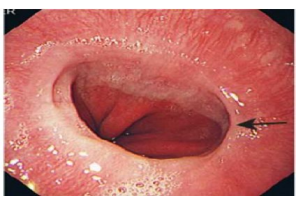
- **Barium[1], endoscopy, and a biopsy to determine whether it's benign or malignant**

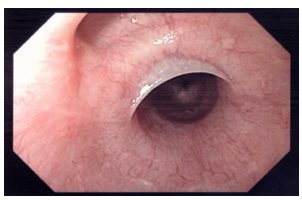
Treatment:

- Depends on the cause: 3 months history of dysphagia? most likely secondary to GERD treat it with PPI's. Didn't work? then its either secondary to fibrosis > Dilation, or cancer > resection
- Benign > dilation , malignant> treat the cancer

◀ 2- ESOPHAGEAL RINGS AND WEBS ★

	Esophageal ring	Esophageal web
Site	Common in the **lower** esophagus (above LES)	Usually in **upper** esophagus (cervical)
Composed of	Connective tissue + muscularis mucosa	**Thin membrane**
Cause	Caused by ? **GERD**	Unknown cause? Genetic
Seen in	Schatzki's ring	**Plummer vinson syndrome[2] (triad[3])**
Risk of cancer	No cancer risk	**PVS** has increased risk for **squamous cell cancer** in the esophagus, hypopharynx and oral mucosa.
Diagnosis	**barium, endoscopy**	
Initial treatment	**PPI** daily for GERD	**Iron replacement** therapy if PVS & be aware of risk of cancer
When unresponsive	endoscopic dilation if needed (persistent symptoms or severe narrowing)	





1-Role of thumb, any patient with dysphagia we do **Barium first** (if no barium in MCQs choose endoscopy) ,If the patient has reflux and dysphagia, also start with barium because of dysphagia. ★
In PVS, if you start with endoscopy you will rupture the web and won't see it, you will miss the diagnosis while the patient is at risk of cancer!
2-Supplementing PVS patients with iron will make esophageal web disappear, however they need continuous surveillance as they have an increased risk of esophageal squamous cell carcinoma
3-the triad of Plummer vinson syndrome are: **Female, iron deficiency anemia, dysphagia secondary to esophageal web**

ESOPHAGEAL DYSPHAGIA

ESOPHAGEAL MOTILITY DISORDERS CAUSING DYSPHAGIA

Esophageal spasms
Not common

Aperistalsis in esophagus (connective tissue disease)
(Scleroderma)
Patient presents with joint pain and dry mouth

Achalasia

◀ 3-Achalasia

Higher risk for Esophageal squamous cell carcinoma (squamous cell carcinoma usually arises from upper and middle third of the esophagus)because of the achalasia itself (genetic predisposition), also has risk of **adenocarcinoma** (less common) because of the complications of achalasia (food & liquids stuck and cause irritation of LES).

Achalasia	
Primary Achalasia[1]	• **Pathophysiology:** Failure of lower esophageal sphincter to relax (lacking ganglion cells in the myenteric plexus in the distal esophagus[2]). • Males = Females **Characterised by:** • A hypertonic lower esophageal sphincter, which fails to relax in response to swallowing waves. • Failure of propagated esophageal contraction, leading to progressive dilatation of the gullet. **Etiology[3]:** • Immune mediated. • Genetic predisposition.
Secondary Achalasia[4] Hints for secondary Achalasia: • Travel history to South/Latin America • Cardiac or GI symptoms (e.g. constipation)	• **Chagas disease**, Parasitic Infection by **Trypanosoma cruzi** (common in latin America), Result in achalasia with features of diffuse enteric myenteric destruction, **including megacolon, heart disease (cardiomyopathy), and neurologic disorders.** • **Diagnosis**: serology testing. • **Treatment**: Anti trypanosoma like **Benznidazole** (GI symptoms managed symptomatically[5])
Pseudoachalasia[6]	• **Achalasia symptoms and similar diagnostic findings.** • **Due to Malignancy** (tumors in the gastric cardia or those infiltrating the myenteric plexus like adenocarcinoma of gastroesophageal junction, pancreatic, breast, lung, or hepatocellular cancers) • **When to suspect (rapid onset over weeks-months[7], elderly[8] and risk factors for cancer)** • Need to get **CT scan or endoscopic US** for further workup.

1-Primary achalasia is the commonest, Patients present with history of esophageal dysphagia that persist for years (2-3 years history)
2- Which has an inhibitory effect on LES.
3- The cause is not well known yet.
4- The main feature of secondary achalasia is that it is **multi-system** (unlike primary) and not restricted to the esophagus only. It is **Important** to look for and ask about **travel history** in secondary achalasia
5- Symptoms are reversible following anti trypanosoma treatments except for the esophagus(irreversible damage), thats why its managed symptomatically
6- in pseudo achalasia, CT scan or endoscopic US is a MUST to roll-out carcinoma specially in high risk group (but it has to be done after the usually workup approach: (barium, endoscopy, and manometry to diagnose and confirm achalasia first)
7- Keep in mind that even 8 Months in Pseudo-achalasia is considered as rapid onset in comparison to primary achalasia (which usually takes 2-3 years, some patients 4-6 y)
8- Above 50, or even if young with Family history of cancer

ESOPHAGEAL DYSPHAGIA

◄ 3-Achalasia (Cont.)

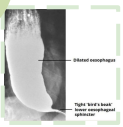
➔ Symptoms of Achalasia:

- **Dysphagia to solids and liquids[1]**
- **Regurgitation[2] of undigested food**
- **Heartburn[2]**
- **Chest pain[3]**
- **Weight loss**

In pseudoachalasia:
rapid weight loss because of cancer + achalasia

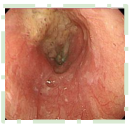
➔ Diagnosis of Achalasia:

01



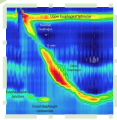
Barium swallow (First Step):
- Dilated esophagus
- Tight LES
- **BIRD BEAK APPEARANCE**

02



Endoscopy
- Normal with some resistance at the LES
- Dilated esophagus. If advanced
- **Retained saliva, liquid, and food in the esophagus** without mechanical obstruction from stricture or mass

03



Esophageal Manometry[4]:
- Shows aperistalsis of the esophagus and failure of relaxation of the LOS.
- **Confirmatory test**

04

To rule out malignancy when you suspect pseudoachalasia:
- **CT** and **US** AFTER confirming the presence of achalasia symptoms and findings

➔ Management of achalasia:

❖ Most common complication of achalasia treatment? GERD

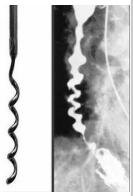
Medical therapy[5]	Endoscopic therapy
Calcium channel blocker	Pneumatic dilatation[6] (effective/ longer term/ more complications)
Nitrates	Botox injection at LES. (effective/ short term 3-6 months)
Antimuscarinic agents	Peroral endoscopic myotomy (POEM): same as Heller's myotomy but from inside by endoscopy
Surgical (Heller's myotomy)	
Cut in the cardia muscle Performed laparoscopically or as opened surgery, accompanied by a partial fundoplication anti-reflux surgery	

1- Because it is a functional dysphagia "motility related"
2- Why do they have the typical symptoms of GERD even though there is no reflux? due to the acidity of accumulated food, that's why they might be mistaken as GERD
3- Some patients experience severe chest pain due to esophageal spasm; it might be misdiagnosed as cardiac pain.
4- A catheter that measures the esophageal and LES pressure, it is never the answer in exam before endoscopy.
5- Medical therapy for achalasia almost never work, however we can describe it for elderly patients with comorbidities that prevent them from doing any endoscopic or surgical procedure

ESOPHAGEAL DYSPHAGIA

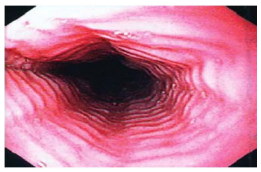
◀ 4- Diffuse esophageal spasm (DES)

★ Diffuse esophageal spasm (DES) (corkscrew esophagus)



- A severe form of esophageal dysmotility that presents in late middle age with **episodic retrosternal chest pain (that can mimic angina)** and **transient dysphagia**.
- Could occur as a response to gastroesophageal reflux.
- **Can be precipitated by drinking cold liquids.**
- On barium swallow, the appearance may be that of a **corkscrew esophagus.**
- **Nutcracker**, a variant of diffuse esophageal spasm, is characterized by **very high-amplitude peristalsis** within the esophagus.
- Treatment is based on PPI when gastroesophageal reflux is present, antispasmodics, nitrates, calcium channel blocker (nifedipine) and GABA agonists (baclofen) are also used.
- Occasionally, balloon dilatation or longitudinal esophageal myotomy is necessary.
- DES and nutcracker can be distinguished only by manometry.

◀ 5- Eosinophilic Esophagitis

Eosinophilic Esophagitis[1]	
Overview	• Chronic inflammation due to **immune-mediated disease** resulting in eosinophilic infiltration of esophagus • **No malignancy Potential.** • Commonly present with food impaction. Main symptom is dysphagia. • **History of allergies** is seen in >50% of these patients.
Diagnosis	**Endoscopy:** • Can be normal. • Strictures. • linear furrows. ★ **Trachealization of the esophagus.** • **BIOPSY shows 15 or more eosinophils**/hpf on microscopy (from proximal and distal esophagus[2]) "Trachealization" 
Treatment	• **PPI[4]** first for 8 weeks then repeat Endoscopy if Eosinophils is still 15 or more: ○ **Corticosteroids[3]** (swallowed fluticasone/ budesonide/betamethasone)for 8-12 weeks. • If symptoms persists repeat endoscopy if there's a ring try dilation. • **Leukotriene inhibitor (montelukast) for refractory symptoms.** • Elimination diets for children.

★ A case from Dr. : 27 yo young male with history of any type of allergy(seasonal, eczema,asthma, contact dermatitis, allergic rhinitis/sinusitis with history of dysphagia , what is the diagnosis? Eosinophilic esophagitis

1- Patients with Eosinophilic esophagitis may present with history of allergies eg: Asthma, allergic rhinitis, food allergies
2- Biopsy **MUST** be taken from both ends of the esophagus as refluxes may also cause eosinophilia
3- Corticosteroids are initiated if PPI didn't work, it has to be swallowed (the patient has to use corticosteroid inhaler(not tablets!) but instead of inhaling the substances he swallows them)
4- Why do we give them PPIs? cause one of the eosinophilic esophagitis causes is reflux

ODYNOPHAGIA[1]+/- DYSPHAGIA

◀ Acute Esophagitis

★ Pill-induced[2]

What is the diagnosis? Acute esophagitis and the cause?

Antibiotics (Tetracycline/Doxycycline):

- 15 years old female which started taking a medication for her acne recently coming with 2-3 days of painful swallowing
- Or Patient with infection and an antibiotic was prescribed for him and now he is coming for painful swallowing

Bisphosphonate (alendronate):

- Elderly post-menopausal lady diagnosed with osteoporosis was given a medication and now she is complaining of odynophagia

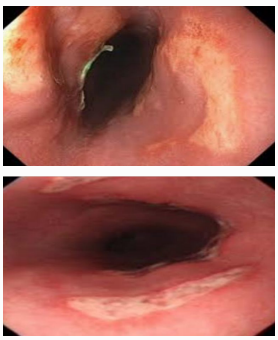
Potassium

- A HF patient on lasix and diuretics with hypokalemia using potassium supplements

Treatment :

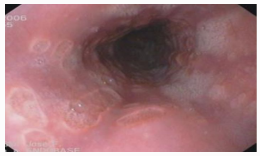
- Stop the medication and give them PPI

"Kissing ulcer appearance"

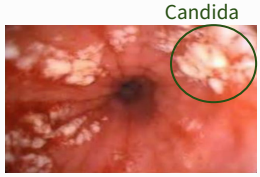


Infections

- **Immunocompromised patients**
  - HIV, DM, on chemotherapy
  - Patients on corticosteroid therapy are at risk of "Infection induced" acute esophagitis
- **Viral** (CMV, HSV)
  - Treatment: antiviral
- **Candida** (more common)
- Common in DM, or in asthmatic using inhaler steroids
  - Treatment: anti fungal



Candida



1- it is typically a pain when swallowing, Usually it is an acute presentation cause they won't tolerate the pain

2- The comments site for pill induced acute esophagitis is at the aortic constriction of the esophagus as it is considered as the tightest area of the esophagus where pills can get stuck(while it is big and acidic mostly), (produce kissing ulcer appearance), for that when you prescribe these medications, advise the pt to drink a lot of water and stay in upright position at least for 30 min after taking the pill.

Summary

GERD	
Sx	• **Typical**: heartburn (the major feature), regurgitation • **Atypical**: nausea, chest pain, bloating and belching, globus sensation, early satiety. • **extra-esophageal**: sinusitis / recurrent otitis media, cough, asthma, dental erosions, laryngitis.
Dx	• **clinical**: typical symptom presentation, antisecretory responsiveness. • **endoscopy**: in case of alarm symptoms, non cardiac chest pain, screening high risk patients for Barrett's, Pts unresponsive to PPI. • **24h pH monitoring and motility**: suspected GERD with normal endoscopy, Persistent symptoms even with PPI.
MGT	• PPI, antacids and alginates, domperidone, H2 receptor antagonist.
Cx	• **peptic strictures**: >60, intermittent dysphagia for solids which worsens gradually. • **erosive esophagitis.** • **Barrett's esophagus**: white men >50, chronic GERD symptoms, Fx history, tobacco, central obesity, Risk for adenocarcinoma.

Zenker's diverticulum	
Sx	• oropharyngeal dysphagia symptoms, undigested food on the pillow at night or after meals, Halitosis, Weight loss in late stage
Dx	• modified barium swallow, video swallowing testing
Tx	• surgical Either via external approach (through the neck) or endoscopy (more commonly) with stapling of the party wall.

eosinophilic esophagitis	
Sx	• **dysphagia** or food bolus obstruction • **chest pain and heartburn** caused by the eosinophil induced esophageal inflammation • History of **allergy** seen in >50% of patients.
Dx	• **endoscopy**: can be normal / strictures / linear furrows / trachealization of the esophagus
Tx	• **PPI**: used first for 8 weeks then repeat endoscopy. • **corticosteroids**: swallowed fluticasone / budesonide / betamethasone • **montelukast**: for refractory symptoms

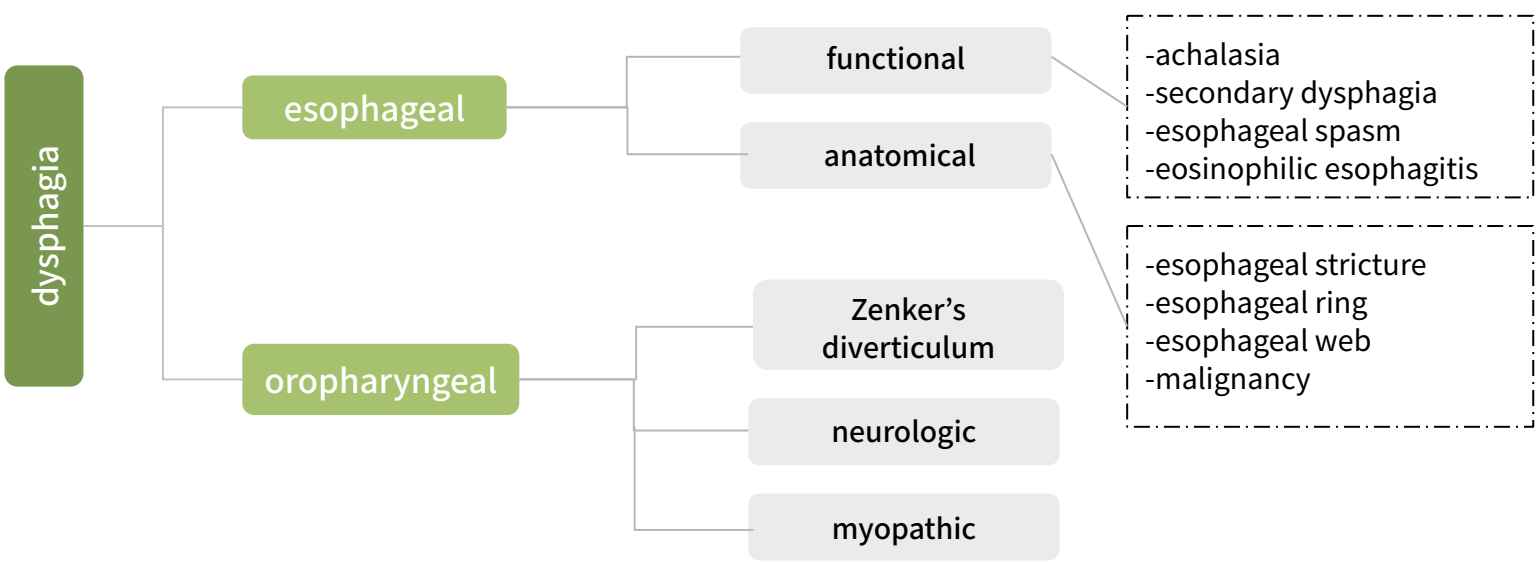
Esophageal webs and rings	
Esophageal ring	**Esophageal web**
Common in the lower esophagus (above LES)	Usually in upper esophagus (cervical)
Connective tissue + muscularis mucosa	Thin membrane
Caused by ? GERD	Unknown cause ?genetic
Schatzki's ring	Plummer vinson syndrome (triad)
No cancer risk	PVS has increased risk for squamous cell cancer in the esophagus, hypopharynx and oral mucosa.
Diagnosis: barium, endoscopy	
Treatment: endoscopic dilation if needed (persistent symptoms)	
PPI daily for GERD	Iron replacement therapy if PVS

Summary

Esophageal strictures	
causes	• Benign peptic strictures, malignant strictures, iatrogenic.
Dx	• Endoscopy.
Tx	• Depends on the cause: 3 months history of dysphagia? most likely secondary to GERD treat it with PPI's. Didn't work? then its either secondary to fibrosis > Dilation, or cancer > resection

Achalasia	
• **Primary achalasia**: Hypertonic LES which fails to relax in response to swallowing waves, and failure of propagated esophageal contraction leading to progressive dilation of the gullet. • Immune mediated and genetic predisposition.	
Sx	• dysphagia (develops slowly,initially intermittent, solids and liquids), chest pain(due to esophageal spasm), regurgitation and pulmonary aspiration, heartburn and weight loss.
Dx	• **barium esophagography** shows dilatation of the esophagus narrowing into a "beak-like" pattern at the lower end. • **esophageal manometry** shows increased LES resting pressure + no/partial relaxation, low amplitude contraction, no propagation. • **endoscopy** normal with some resistance at the LES, dilates esophagus and retained saliva, liquid and food in the esophagus.
MGT	• **drugs:** nitroglycerin / calcium channel blocker / antimuscarinic agents. • **endoscopic:** forceful pneumatic dilatation / botulinum toxin injection / peroral endoscopic myotomy. • **surgical myotomy (Heller's operation).**

Diffuse esophageal spasm (corkscrew esophagus)	
Sx	• Episodic retrosternal chest pain and (can mimic angina), dysphagia, precipitated by drinking cold liquids.
Dx	• **Barium swallow**: appearance maybe that of a corkscrew esophagus.
Tx	• PPI when GER is present.
	• Antispasmodics, Nifedipine (CCB) and nitrates.



Lecture Quiz (Dr's slides)

Q1: A 42 y/o male presents for evaluation of heartburn. He denies any dysphagia or weight loss. He has no other medical conditions and is currently not taking any medications. You suspect that he may have GERD. Which of the following describes the role of upper endoscopy in the evaluation of this patient?

A- He should undergo an Esophagogastroduodenoscopy with biopsy to ensure a more serious condition is not missed
B- He should undergo an Esophagogastroduodenoscopy only if he has no improvement in his symptoms after an empiric trial of twice daily PPI therapy.
C- He should undergo ambulatory PH monitoring while on PPI therapy to ensure the medication is working
D- He should undergo both an Esophagogastroduodenoscopy and ambulatory PH monitoring prior to the initiation of any therapy.

He has typical symptoms of GERD, no alarm symptoms of GERD, never tried PPI , no risk factors for barrett's
So the answer is: B

Q2: A 45-year-old man is evaluated for a 2-month history of a burning sensation starting in his stomach and radiating into his chest, usually occurring 4 to 5 times weekly. He says that he usually eats dinner late and then goes to sleep. He often wakes up with a sour taste in his mouth. He reports no dysphagia or unintentional weight loss. He takes no medication. On physical examination, vital signs are normal; BMI is 34. The remainder of the examination, including abdominal examination, is unremarkable. What would be the next step in his management?

A- PH testing
B- Barium esophagography
C- Empiric trial of proton pump inhibitor
D- Esophagogastroduodenoscopy

Q3: - A 56-year-old woman is evaluated for chest discomfort after meals occurring intermittently over the preceding month. She describes a sensation of heaviness on her chest, and says that she also notices this pain sometimes while walking up stairs. She reports no nausea, dysphagia, or reflux. She has been taking ranitidine with minimal relief of symptoms. She also takes atorvastatin for hyperlipidemia. She smokes half a pack of cigarettes daily. On physical examination, her blood pressure is 140/90 mm Hg and other vital signs are normal; BMI is 34. The remainder of the examination, including abdominal examination, is unremarkable. What is your next step?

A- Barium esophagography
B- Electrocardiography
C- Empiric trial of a proton pump inhibitor
D- Esophagogastroduodenoscopy

She has chest pain, noticed sometimes when walking upstairs, she's obese, smoker, has hyperlipidemia so most likely it is a cardiac cause
Don't forget (any chest pain beside heartburn > we need to roll out cardiac causes
So the answer is: B

Answers: Q1:B | Q2:C | Q3:B

Lecture Quiz (Dr's slides)

Q4: - A 75-year-old man is evaluated for progressive dysphagia of 8 months' duration for both solids and liquids and the necessity to induce vomiting several times each month to relieve his symptoms. He also has experienced chest pain and heartburn symptoms. He has lost approximately 6 kg (13 lb) of weight over the preceding 3 months and a total of 9 kg (20 lb) since his symptoms began. He has a long history of cigarette and alcohol use. His medical history and review of systems is otherwise negative. He has no travel history outside the northeastern United States. He takes no medication. On physical examination, vital signs are normal; BMI is 23. He appears thin and tired. The remainder of the physical examination is unremarkable. Esophagogastroduodenoscopy findings reveal retained saliva, liquid, and food in the esophagus without mechanical obstruction. Manometry demonstrates incomplete lower esophageal relaxation and aperistalsis. What is the most likely diagnosis?

A- Achalasia
B- Pseudoachalasia
C- EOE
D- Chagas disease

It is achalasia, but we need to know is it primary,secondary , or pseudoachalasia?
2ry achalasia will be rolled out because no history of recent travel to Latin America, but it's is a short duration (8 months) he is elderly and has weight loss, has risk factor: long history of cigarette smoking
So the answer is: B

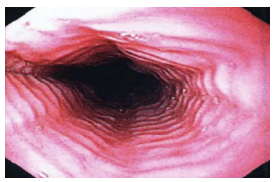
Q5: A 52-year-old man is evaluated for dysphagia of 3 months' duration. He reports regurgitating undigested food soon after eating solid food, occasional coughing and choking after swallowing, and chronic halitosis. He reports no weight loss or chest pain. He drinks two beers weekly and does not smoke. On physical examination, vital signs are normal; BMI is 25. The remainder of the examination, including abdominal examination, is unremarkable. What is your next step?

A- Barium esophagram
B- Esophagogastroduodenoscopy
C- Manometry
D- PH study

The answer is: A ,because this is a dysphagia so the barium is first
What is the diagnosis? Zenker's diverticulum because the symptoms of oropharyngeal dysphagia

Q6: A 25 year old man is evaluated for a sensation of solid food "sticking" several times per week. He reports that he sometimes forces himself to vomit when he feels food "stuck" in the esophagus, but he has never gone to the emergency department. He takes a multivitamin and is generally healthy.On physical examination, vital signs and other findings, including those of an abdominal examination, are unremarkable. Esophagogastroduodenoscopy findings are shown. Biopsies of the esophagus show more than 18 eosinophils/hpf. Which of the following is the most likely diagnosis?

A- Achalasia
B-Eosinophilic esophagitis
C- Gastroesophageal disease
D- Pill induced esophagitis



The patient has dysphagia, he's young, trachealization, eosinophils more than 15
So the answer is: B
(But this patient is from the 50% who don't have allergy history)

Answers: Q4:B | Q5:A | Q6:B

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