

Esophageal diseases

Objectives:

- 1. Surgical anatomy.
- 2. Symptoms of esophageal diseases: a. Dysphagia | b. Pain. | c. Regurgitation.
- 3. Examination.
- 4. Investigation: a. Blood test. | b. Radiology. | c. Endoscopy. | d. Computed tomography. | e. Ultrasonography. | f. Laparoscopy. | G. Manometry and pH studies.
- 5. Impacted foreign bodies.
- 6. Corrosive oesophagitis.
- 7. Perforation.
- 8. Motility disorder: a. Achalasia. | b. Diffuse esophageal spasm. | c. Nutcracker oesophagus.
- 9. Plummer-vinson syndrome.
- 10. Pouches.
- 11. Gastro-oesophageal reflux: a. Hiatus hernia. | b. Barrett's oesophagus.
- 12. Tumours of the oesophagus: a. Benign tumours. | b. Carcinoma of the oesophagus.

Resources:

- Davidson's.
- Slides.
- Surgical recall.
- 435 Teamwork

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The ones in <u>Grey</u> Dr. Sami did not explain during the lecture **but mentioned in the slides & objectives.**Dr. Sami said that the exam will be directly from the slides.

COLOR INDEX:

NOTES, IMPORTANT, EXTRA, DAVIDSON'S

EDITING FILE









Basic Review (Read it to refresh your mind)

Esophagus:

• Length: about 25 cm long

• Extension:

(1) starts at cricoid cartilage - C6 (2) passes through the diaphragm to join the stomach at T10 (3) final 2-4 cm lie within the peritoneal cavity, terminates at T11 by entering the cardiac orifice of the stomach.

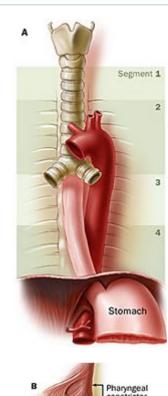
• The esophagus is a tubular structure with **four** layers:

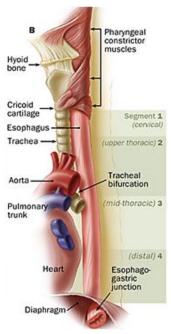
Mucosa	Submucosa	Musculosa externa	Outer layer
Lined by Nonkeratinized Stratified squamous epithelium.	Mucous glands and lymphatics. [Meissner's plexus: nerve fibers and nerve cells]	Outside: longitudinal [Auerbach's (myenteric) plexus in between the 2 layers] Inside: circular • Upper 1/3: both are skeletal. • Middle 1/3: inner is smooth, outer is skeletal. • Lower 1/3: both are smooth.	Adventitia: upper two thirds Serosa: lower one third

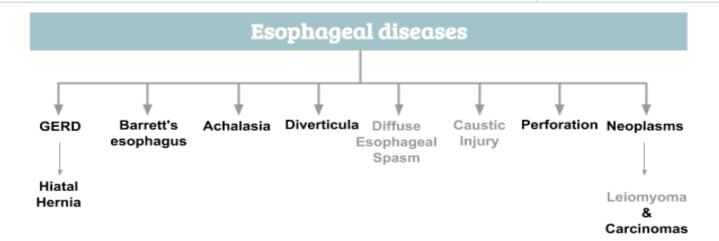
• Parts: cervical, thoracic and abdominal portions

Region	Cervical	Thoracic	Abdominal
Arterial supply	Inferior thyroid artery	Thoracic aorta (bronchial arteries and branches)	Inferior phrenic & left gastric arteries (from celiac trunk)
Venous drainage	Inferior thyroid veins	Hemiazygos & azygos veins (systemic circulation).	Left gastric vein (portal circulation) Note: Connection between these veins are important in the formation of varices.
Lymph drainage	Deep cervical nodes.	Superior & inferior mediastinal nodes.	Celiac lymph nodes
Nerve supply	Sympathetic:preganglionic (T5&T6) postganglionic (cervical & coeliac ganglia) Parasympathetic: glossopharyngeal vagus and recurrent laryngeal nerves		
Constric- tions	Pharyngeal- esophageal At junction with pharynx.	Aorto-bronchial Crossing of aortic arch & left main bronchus	Diaphragmatic (LES) At junction with stomach

- Esophagus has two sphincters:
- **Upper sphincter:** cricopharyngeus.
- **Lower sphincter:** cannot be defined anatomically, 3-5 cm high-pressure area located in the region of esophageal hiatus of the diaphragm.









GASTROESOPHAGEAL REFLUX DISEASE Helpful video 10:05

Definition

It is often a chronic and relapsing condition characterized by <u>symptoms</u> OR <u>mucosal damage</u> produced by abnormal reflux of gastric contents into the esophagus when pressure of the high-pressure zone in the distal esophagus is too low to prevent gastric contents from entering the esophagus.

(LES has the primary role of preventing reflux) (the most common esophageal disease).

Epidemiology

- •About 44% of the US adult population have heartburn at least once a month.
- •14% of Americans have symptoms weekly.
- •7% have symptoms daily.

Hiatal Hernia helpful video 1:49

- GERD is often associated with a hiatal hernia: sliding, rolling and mixed:
 - What is the Hiatus? it is the opening in the diaphragm that the (thoracic esophagus) passes through to continue as the (abdominal esophagus).
 - If the hiatus opening gets enlarged, a part of the stomach will be herniated through the hiatus.

Types of hiatal hernias				
Type I (Important)	Type II	Type III	Type IV	
Sliding Hiatal Hernia	Rollin	ng (paraesophageal) Hernia	ıs (10%)	
Most common (90%)	No acid reflux May be associated with GERD			
 Portion of the stomach cardia slides through the diaphragmatic hiatus. Gastroesophageal junction lay within chest cavity resulting in negative pressure in the chest. Pressure will keep LES opened → reflux of acid (Strong association with GERD) . 	Localized defect in the phrenoesophageal membrane while the gastroesophageal junction remains fixed to the preaortic fascia and the median arcuate ligament. The gastric fundus then serves as the leading point of herniation. (may have dysphagia and/or chest pain because a part of the stomach is constricted).	Have elements of both types I and II hernias. Symptoms will include both of them.	Associated with a large defect in the phrenoesophageal membrane, allowing other organs, such as colon, spleen, pancreas and small intestine to enter the hernia sac.	
Type I	Type II	Type III	Type IV	



Clinical features of hiatal hernias

- Heartburn (pyrosis) and regurgitation → caused by reflux of acid.
- Esophagitis → persistent acid reflux → ulceration, bleeding with anemia, fibrosis, strictures formation.
- Epigastric and lower chest pain especially in paraesophageal hernias → may be a medical emergency¹.
- Palpitations & hiccups → caused by mass effect of the hernia irritating the pericardium & diaphragm.

Clinical Presentations of GERD

- Patients who lack typical symptoms you may see the following complications:
 - Asthma → caused by aspiration of acid.
 - Hoarseness → caused by <u>irritation of vocal cords caused by acid reflux</u>.

"Classic" GERD	Extraesophageal Manifestations of GERD		
• substernal Heartburn	Pulmonary	ENT	Others
and/or regurgitation: - Postprandial. - Aggravated by change of position. - Prompt relief by antacid. - Can be relieved also by drinking milk.	 Asthma.² Aspiration pneumonia. Chronic bronchitis. Pulmonary fibrosis. 	Hoarseness.(can be only hoarseness without reflux, it called silent gerd) Chronic cough. Laryngitis. Pharyngitis. Globus Sensation. Dysphonia. Sinusitis. Subglottic Stenosis. Laryngeal Cancer.	Nausea Occasional vomiting Waterbrash (hypersalivation) Epigastric pain Chest Pain Dental Erosion.

Complicated GERD (RARE)

- Dysphagia: difficulty swallowing (food sticks or hangs up, mainly caused by strictures)
- Odynophagia³: retrosternal pain with swallowing (caused by ulcers in the esophagus)
- Bleeding. In ulcers
- Barrett's esophagus (columnar metaplasia with goblet cells): metaplasia without goblet cells is not BE.

Diagnostic Tests for GERD

Diagnosis is made by by (1) good history (2) endoscopy (3) sometimes a 24-hour esophageal pH study. If young + NO alarming symptoms → empirical treatment may be appropriate without doing any investigations.

Barium swallow⁴:

We give the patient a contrast to drink and we do an X-ray on supine position, then we lift the patient a little bit to see if the contrast will reflux on the esophagus.

- Endoscopy.
- Ambulatory pH manometry "most diagnostic": 24-hour-pH monitor.
- Esophageal manometry to know the motility of the esophagus usually done before surgery.
- CXR is normal unless it's complicated.

¹ owing to the obstruction and strangulation of the stomach.

² Sometimes the bronchial asthma is because of gerd. when there is reflux the acid aspirate through bronchial tree which cause bronchospasm and symptoms of asthma.

³ In general, may be due to a mechanical obstruction or intrinsic dysmotility.

⁴ Barium swallow <u>in general</u> is used: if endoscopy is unavailable, patient is not candidate, to exclude pharyngeal pouch & perforation, complementary to endoscopy (provide additional anatomical information ex, hiatal hernia).



Treatment

• Lifestyle Modifications (The most important):

- o Elevate head of bed 4-6 inches.
- Avoid eating within 2-3 hours of bedtime.
- o Lose weight if overweight.
- Stop smoking.
- Modify diet
 - Eat more frequent but smaller meals.
 - Avoid fatty/fried food, peppermint, chocolate, alcohol, carbonated beverages,coffee & tea.
- o OTC (over-the-counter) medications prn⁵ (as needed).

• Acid Suppression Therapy for GERD:

H2 Receptor Antagonists (H2RAs)	Proton Pump Inhibitors (PPIs) "more effective"
Cime tidine (Tagamet) Rani tidine (Zantac) Famo tidine (Pepcid) Niza tidine (Axid)	Ome prazole (Prilosec) Lanso prazole (Prevacid) Rabe prazole (Aciphex) Panto prazole (Protonix) Esome prazole (Nexium)

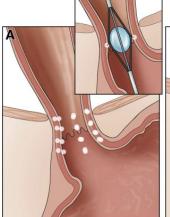
Anti-Reflux Surgery: "Indications for Surgery"

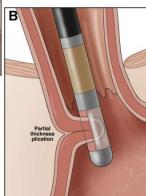
- Failed medical management; usually due to high volume acidic reflux.
- Patient's preference despite successful medical management (due to lifestyle considerations Including age, time or expense of medications, etc.)
- o Complications of GERD (e.g. Barrett's esophagus; grade III or IV esophagitis)
- Complications of <u>large hiatal hernia</u> (e.g.bleeding, dysphagia).
- **Atypical symptoms** (asthma, hoarseness, cough, chest pain,aspiration) and reflux documented on 24-hour pH monitoring.

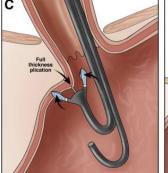
• Endoscopic GERD Therapy:

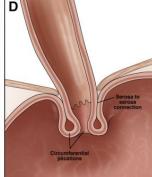
Endoscopic antireflux therapies

- **A. Stretta procedure:** Radiofrequency energy delivered to the LES.
- B. Endoscopic plication: Suture ligation of the cardia.
- **C. Enteryx:** Submucosal implantation of inert material in the region of the lower esophageal sphincter.
- D. Nissen fundoplication "most common": <u>Helpful video 4:32</u>, Gastric fundus wrapped around the lower esophageal sphincter.









⁵ Abbreviation meaning "when necessary" (from the Latin "pro re nata")



BARRETT'S ESOPHAGUS

Definition

A condition whereby an intestinal, columnar epithelium replaces the stratified squamous epithelium that normally lines the distal esophagus (intestinal metaplasia).

Surface epithelium of Barrett's esophagus: columnar epithelium **WITH** goblet cells! If you don't see goblet cells the it is Just METAPLASIA.

Risk Factors

- Chronic GERD:
 - Injury of squamous epithelium \rightarrow promotes repair through columnar metaplasia.
 - Metaplastic cells may be more resistant to injury from reflux and are more prone to malignancy.
 - With continued exposure to the reflux disease, metaplastic cells undergo cellular transformation to low- and high-grade dysplasia.
 - these dysplastic cells may evolve to cancer

[continuous exposure to GERD (esophagitis + hyperplasia) → Barrett's esophagus (intestinal metaplasia) → dysplasia -low or high- → adenocarcinoma (neoplasia)]



- 10% of patients with GERD develop Barrett's esophagus.
- More common in men (70%) | aged 55 to 63 years.
 - Incidence of **adenocarcinoma** of the esophagus men > women.
 - Women with BE are <u>increasing</u> in number (diminished differences in western lifestyles among men & women).
- 40-fold increase risk for developing esophageal carcinoma in patients with Barrett's esophagus.

Symptoms

- Many patients harboring intestinal metaplasia in their distal esophagus are asymptomatic.
- Most patients present with **symptoms of GERD:** Heartburn, regurgitation, acid or bitter taste in the mouth, excessive belching⁶, and indigestion.
- Recurrent respiratory infections, adult asthma, & infections of head & neck are common complication.

Diagnosis

- Diagnosis is made by: Endoscopy & pathology (histopathology)
 - **Endoscopy**: shows visible segment of columnar mucosa within the esophagus (the red spots).
 - Pathology (biopsy): identifies intestinal metaplasia.





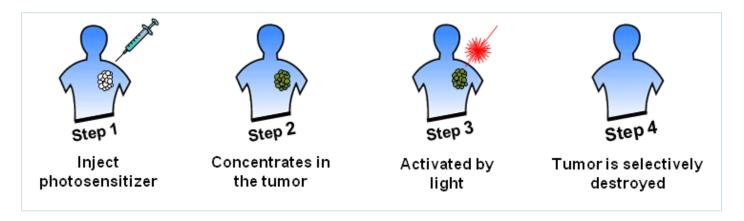
⁶ Burbing



Treatment

Barrett's esophagus

- 1. Yearly surveillance endoscopy is recommended in all patients with a diagnosis of BE.
 - Patients are placed on acid suppression medication and monitored for changes in their reflux symptoms.
- 2. Photodynamic therapy (PDT) is the most common ablative method used to treat BE.



Low-grade dysplasia

1. Surveillance endoscopy

- 6-months intervals for the first year.
- Annually after the first year if there has been no changes.
- 2. **Endoscopic mucosal resection (EMR)** is gaining favor for the treatment of Barrett's esophagus with low-grade dysplasia.

High-grade dysplasia

Esophageal resection for Barrett's esophagus is <u>recommended</u>.

Pathologic data on surgical specimens demonstrate a 40% risk for adenocarcinoma within a focus of high-grade dysplasia.

Antireflux surgery

- Benefits of antireflux surgery are controversial in patients with BE.
- Those in favour of surgery argue that medical therapy and endoscopic surveillance may treat the symptoms but fail to address the problem.
- Problem is a functional impairment of the LES that leads to chronic reflux and metaplastic transformation of the lower esophageal mucosa.
- Surgery renders the LES competent and restores the barrier to reflux.
- Studies have demonstrated regression of metaplasia to normal mucosa up to 57% of the time in patients who have undergone antireflux surgery.



ACHALASIA Helpful video 2:51

Definition

Achalasia is an uncommon disease. However, it is the most common type of esophageal motility disorders. It is characterized by partial or complete degeneration of the myenteric plexus of Auerbach that innervate LES and esophageal body.

The main feature is failure of relaxation of the lower esophageal sphincter.

Possible etiologies (It is idiopathic)

- · Autoimmune.
- Infectious: Trypanosoma cruzi "Chagas disease⁷" → causes secondary achalasia.
- Gastroesophageal junction tumors sometimes mimic achalasia (pseudoachalasia)

Necrosis so the nerve supply of the esophagus disappear.

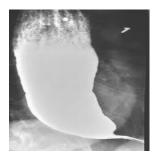
Clinical features

- Common in (young patients) between the age of (25-60 years) | males = females
- Most common presenting symptoms (in order):
- **1. Progressive dysphagia (main symptom)**: to both solids & liquids at the same time [If dysphagia occurs for solids first and then liquids → carcinoma]
- 2. Regurgitation is the second most common symptom
 - nocturnal regurgitation of esophageal contents → nighttime cough & aspiration.
- 3. Others: chest pain | Heartburn⁸ | Halitosis (Bad breath smell)| Weight loss (in end-stage disease)
- Patients prefer eating while standing up to make the gravity help them to swallow.

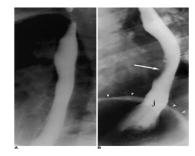
Gravity is responsible for food leaving the oesophagus rather than peristalsis in achalasia, so the patient finds it easier to eat when standing.

Diagnosis

- CXR: may show air-fluid level.
- Barium study⁹: (1) classic finding bird's beak (gradual tapering at the end of the esophagus) It also shows (2) dilated esophagus (3) air-fluid level : may be secondary to retained secretions.
- Upper endoscopy is the NEXT diagnostic test in a patient with suspected achalasia or dysphagia.
 - **Findings**: (1) Dilated esophagus with retained food or secretions. (2) Normal in ≈44% of patients.
 - Difficulty traversing the GEJ (Gastroesophageal junction) → suspicion of **pseudoachalasia** due to neoplastic infiltration of the distal esophagus.



Bird's beak



Normal Esophagram



Nutcracker Esophagus(DES)

⁷ Causes degeneration of the myenteric plexus, leading to a motor disorder of the oesophagus.

⁸ Maybe related to direct irritation of the esophageal lining by retained food, pills, or acidic byproducts of bacterial metabolism of retained food.

⁹ Initial test if we suspect achalasia (from the history)



- Esophageal manometry [has the highest sensitivity (Most accurate) for achalasia diagnosis]:
 - Aperistalsis of the distal esophageal body.
 - o Incomplete or absent LES relaxation.
 - Hypertensive LES.
- Manometric variants of achalasia vigorous achalasia (best known)
 - Defined by the presence of normal to high amplitude esophageal body contractions in the presence of a nonrelaxing LES.
 - May represent an early stage of achalasia.

Secondary forms of achalasia (by endoscopy) to know about pseudoachalasia

- Cancer (most concerning etiology): can present as achalasia through mechanical obstruction of the GEJ.
- Chagas disease: parasitic infection caused by Trypanosoma cruzi which can cause secondary achalasia.
- **Post fundoplication achalasia:** (increasingly recognized etiology): caused by mechanical obstruction of the GEJ by the fundoplication or diaphragmatic crural closure.
- Following bariatric surgery (مثل قص المعدة : using a gastric band device which constricts the proximal stomach a few centimeters below the LES following bariatric surgery.

Treatment

- Primary therapeutic goal: to reduce LES basal pressure.
- Primary desired outcome: symptoms relief, particularly of dysphagia.
- Treatment options¹⁰: medical therapy, botulinum toxin injection, pneumatic dilation, and surgical myotomy.

• Medical Therapy:

- Inconvenient, only modestly effective, and frequently associated with side effects, it is reserved for
 patients awaiting or unable to tolerate invasive treatment modalities.
- Pharmacologic therapies attempt to decrease the LES pressure by causing smooth muscle relaxation
- Nitrates were first recognized as an effective treatment of achalasia.
 - Their systemic vasodilatory effects and headaches limit their tolerability by patients.
- o Calcium channel blocker have a better side-effect profile when compared with nitrates
 - 30% of patients report adverse side effects (peripheral edema, hypotension, and headache)

• **Botulinum Toxin Injection:** (endoscopically)

Administration: injection into the LES targets the excitatory, acetylcholine-releasing neurons.
 It requires reinjection although it is apparent that with repeated injections, the response rates reported are similar or lower to that achieved with the initial injection.

We inject Botulinum toxin which causes paralysis of LES Temporarily. Response rates at 1 month following administration average 78%, By 6 months, the clinical response rate drops to 58% and by 12 months to 49%.

Mechanism of action:

Blocks excitatory-acetylcholine-releasing neurons generated by LES basal muscle tone.

Characteristics:

Easy to administer | associated with relatively few side effects.

Indication:

Patients who are not candidates for more invasive treatments given the limitations of the efficacy and durability of response.

¹⁰ treatment of choice depends on several factors including age, surgical risk, comorbidities, severity, patient preference, and locally available expertise according to new guidelines.



• Pneumatic Dilation: "first-line"

- One of the most effective first line **therapies** for achalasia.
- o **Procedure:** placing an endoscopy that inflates a device to enlarge the lower esophagus.
- Long-term follow-up studies reported significant symptom relapse of 50% at 10 years.
- Complications of pneumatic dilation:
 - Gastroesophageal reflux (25-35%).
 - Esophageal perforation (3%) (higher perforation percentage than surgery).

• Surgical Therapy: "best treatment" (We cut the muscles of the sphincter to reduce the pressure)

- Surgical myotomy: laparoscopic Heller's myotomy (common) & open surgical myotomy (rare)
- has success rates in excess of 90% with hospital stays averaging only a few days
- Heller myotomy: muscles of the cardia (lower esophageal sphincter or LES) are cut, allowing food and liquids to pass to the stomach | high success rates | hospital stays only for few days.
- Laparoscopic Heller myotomy shows excellent results (98% symptomatic improvement at 5.3 years)
- Complications:
 - Acid exposure¹¹.
 - Dysphagia
 - As a consequence of esophageal peristaltic dysfunction <u>even</u> with a successful myotomy .
 - Delayed recurrence of postoperative dysphagia is most commonly caused by development of a recurrent high pressure zone at the LES or a peptic stricture complicating acid reflux.

Complication of Heller's myotomy:

Early: perforation

Late: reflux esophagitis & recurrent dysphagia from an inadequate myotomy.

Surgery Versus Pneumatic Dilation:

- Several studies have reported success rates of surgery more than pneumatic dilation.
- Surgery is more effective but more dangerous than pneumatic dilation.
- Pneumatic dilation: long-term follow-up studies reported significant symptom relapse of 50% at 10 years.

Refractory achalasia

Achalasia that is refractory to therapy with Heller myotomy, options are limited.

Esophagectomy

Considered if there is marked dilation, sigmoid esophagus (>10 cm dilation of distal esophagus) Such patients may respond to Heller's myotomy.

Complications of achalasia

• **Primary complications:** progressive malnutrition and aspiration. (related to the functional obstruction caused by the nonrelaxing LES)

- Secondary complications (uncommon but important):
 - Formation of epiphrenic diverticulum
 - Esophageal cancer (most commonly **squamous cell carcinoma**)

Prevalence of esophageal cancer in achalasia ≈3% I Incidence ≈197 cases per 100,000 persons per year.

¹¹ Surgery is often combined with partial fundoplication to reduce the incidence of postoperative acid reflux & GERD.



ESOPHAGEAL DIVERTICULA¹²

- •Most diverticula are a result of: (1) primary motor disturbance. | (2) abnormality of the UES or LES.
- Can occur in several places along the esophagus. The three most common sites:
 - o Pharyngoesophageal (Zenker's).
 - o Peribronchial (mid-esophageal).
 - o Epiphrenic.

True diverticulum	False diverticulum
ALL LAYERS of esophageal wall (mucosa - submucosa - muscularis)	Mucosa and submucosa <u>ONLY</u>
True diverticulum (Traction) Results from: External inflammatory mediastinal lymph nodes adhering to the esophagus. The best example is: Meckel's Diverticulum	Pulsion (pressure) diverticula: Caused by elevated intraluminal pressures; generated from abnormal motility disorders. It includes: Zenker's & epiphrenic diverticulum

Pharyngoesophageal (Zenker's) Diverticulum

- Most common | usually in older male patients | 7th decade of life.
- Found herniating into **Killian's triangle** (between the oblique fibers of the thyropharyngeus muscle and the horizontal fibers of the cricopharyngeus muscle). Incoordination of swallowing and failure of relaxation of the cricopharyngeus muscle cause the herniation.

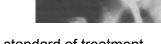
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Symptoms & Diagnosis

- Common: patients complain of a sticking (gurgling) in the throat.
- Especially common in elderly: Halitosis 13 | voice changes | retrosternal pain | respiratory infections.
- Signs of progressive disease: nagging cough | hypersalivation | intermittent dysphagia.
- As pouch increase in size regurgitation of foul-smelling & undigested material is common.
- The most serious complication from an untreated Zenker's diverticulum is aspiration pneumonia or lung abscess.
- * A lump in the neck might be seen (pharyngeal pouch) | might be asymptomatic.
- Diagnosis is made by barium esophagram

(which demonstrates pouch + uncoordinated swallowing).

Esophageal manometry and endoscopy are **NOT** needed to make a diagnosis. Endoscopy rather <u>confirms</u> the diagnosis. It must be performed with <u>care to avoid accidental perforation of the pouch.</u>



Treatment: Surgical or endoscopic repair of a Zenker's diverticulum is the gold standard of treatment.

- Open surgical repair: (myotomy + resection of pouch) "gold standard of treatment"
 - Myotomy of proximal & distal thyropharyngeus and cricopharyngeus muscles.
 (We do the myotomy to prevent the recurrence).
 - Pouch resection: **diverticulectomy**¹⁴/**diverticulopexy**¹⁵ through an incision in the left neck.
- Endoscopic repair is an alternative to open surgical repair: endoscopic Dohlman procedure (also known endoscopic Zenker's diverticulotomy 16): by dividing the common wall between the esophagus & diverticulum using a laser or stapler.

¹² Also known as (pouches): protrusions of mucosa through a weak area in the muscle wall.

¹³ Bad breath.

¹⁴ **Diverticulectomy**: surgical excision (resection) of a diverticulum.

¹⁵ Diverticulopexy: without resection. Usually by securing the tip to a nearby structure so diverticulum no longer fills.

¹⁶ **Diverticulotomy:** surgical incision into diverticulum.



DIFFUSE ESOPHAGEAL SPASM **O**

Dr. Sami did not explain this during the lecture but it is mentioned in the slides & objectives.

- Seen most often in: women | patients with multiple complaints | middle-aged to elderly.
- Basic pathology:
 - Motor abnormality (hypermotility) of the esophageal body.
 - Most notable in the lower ¾ of the esophagus.
 - Esophageal contractions are repetitive, irregular, simultaneous, and of high amplitude.

Symptoms & Diagnosis

- Clinical presentation (typically): non-cardiac retrosternal chest pain | intermittent dysphagia. May also complain of a squeezing pressure of the chest that may radiate to the jaw, arms and upper back.
- Exacerbating factors: eating or exertion (may mimic angina) | times of heightened emotional stress.
- Associated symptoms:
 - **Common:** regurgitation of esophageal contents and saliva.
 - **Uncommon:** acid reflux (can <u>aggravate</u> the symptoms, as can cold liquids)
- Triggering factors: gallstones | peptic ulcer disease | pancreatitis.

Diffuse esophageal spasm can be precipitated by GERD and this should be excluded by 24-hour pH studies.

- Comorbidities: Irritable bowel syndrome | pyloric spasm.
- Diagnosis: made by esophagram and manometric studies.

manometry is required to make the diagnosis

Treatment

- Mainstay of treatment for DES is **nonsurgical**. Pharmacologic or endoscopic intervention are <u>preferred</u>. Medical treatment includes: calcium channel blockers, sublingual GTN and proton pump inhibitors.
- Surgery is reserved for patients with:
 - Recurrent incapacitating episodes of dysphagia and chest pain.
 - NO response to medical treatment.

Surgical treatment involves a long myotomy but the results are unpredictable and most patients will be treated medically.

"Corkscrew sign"







CAUSTIC INJURY

Dr. Sami did not explain this during the lecture but it is mentioned in the slides & objectives.

- •Children → usually accidental ingestion | small quantities.
- **Teenagers and adults** → deliberate ingestion during suicide attempts | larger quantities.
- · Alkali ingestion is more common and much more devastating than acid ingestion because of its lack of immediate symptoms and almost always lead to significant destruction of the esophagus.

Three Phases of Tissue Injury From Alkali Ingestion					
Phase	Tissue injury	Onset	Duration	Inflammatory response	Symptoms
4	Aguto nogrania	1 4	1 4 dove	Coagulation of intracellular proteins.	*Oral and substernal pain
1	Acute necrosis.	1-4 days	1-4 days	Inflammation.	*Hypersalivation *Odynophagia & dysphagia *Vomiting & Hematemesis
2	Illogration 9 granulation	2.5	2.40	Tissue sloughing.	
	Ulceration & granulation	3-5 days	3-12 days	Granulation of ulcerated tissue bed.	Symptoms may disappear
3	Cigatrization 9 coording	3	1-6	Adhesion formation.	Dysphagia reappears; as
	Cicatrization & scarring	weeks	months	Scarring	fibrosis and scarring begin to narrow the esophagus.

Symptoms & Diagnosis

Other symptoms	Physical examination	Investigations
 Symptoms of respiratory distress: hoarseness, stridor, and dyspnea → suggest upper airway edema and are usually worse with acid ingestion. Pain: chest & back → may indicate perforation of mediastinal esophagus, abdominal → may indicate abdominal visceral perforation. 	 Evaluation of: mouth, airway, chest, and abdomen. Careful inspection of: lips, palate, pharynx, and larynx. Abdomen is examined for signs of perforation. 	 Early endoscopy is recommended 12 to 24 hours after ingestion to identify the grade of the burn. First degree: Mucosal hyperemia, edema Second degree: Limited hemorrhage, exudates, ulcerations, pseudomembrane formation. Third degree: Mucosal sloughing, deep ulcerations, massive hemorrhage, complete luminal obstruction, charring, perforation Questionable chest and abdominal exams → Serial chest and abdominal radiographs are indicated

Treatment (The best cure for this condition is an ounce of prevention)

- Management of the acute phase is aimed at limiting and identifying the extent of the injury.
- It begins with **neutralization** of the ingested substance:
 - 1. **Alkalis** (including lye) are neutralized with half-strength vinegar or citrus juice.
 - 2. **Acids** are neutralized with milk, egg whites, or antacids.

- Emetics and sodium bicarbonate need to be avoided because they can increase the chance of perforation.			
First-Degree Burn	Second-Degree Burns	Third-Degree Burns	
 48 hours of observation. Oral nutrition can be resumed when a patient can painlessly swallow saliva. A repeat endoscopy and barium esophagram are done in follow-up at intervals of 1, 2, and 8 months. Acid suppression. 	 Aggressive resuscitation. The patient is monitored in the ICU. NPO with IV fluids IV antibiotics Fiberoptic intubation (if needed) Acid suppression. PPIs are started. 	Fiberoptic intubation (if needed) Inhaled steroids.	



ESOPHAGEAL PERFORATION¹⁷

- •Perforation of the esophagus is a surgical emergency. (very serious. It may lead to sepsis and death).
- \leq 24-hrs early detection and surgical repair \rightarrow 80%-90% survival. | \geq 24-hrs \rightarrow survival less than 50%.

Etiology: [common sites of perforation are at sites of anatomical narrowing (constrictions)]

Intraluminal	Outside the wall	Spontaneous:
Foreign body ingestion (14%)	Trauma (10%) (by penetrating injuries such as knife	Forceful vomiting (Boerhaave's syndrome) (15%)
latrogenic; most esophageal perforations occur after endoscopic ¹⁹ instrumentation for a diagnostic endoscopy (rare) or therapeutic procedure, such as dilatation (commoner).	wounds to the neck but are rare)	A tear to the esophageal mucosa only following vomiting is known as a Mallory–Weiss tear and tends to cause haematemesis and pain. ¹⁸

Symptoms and Diagnosis [depend on the site and size of the perforation]

Perforation that may be present			
Cervical perforations	Thoracic perforations	Abdominal perforations	
Neckache, stiffness due to contamination of the prevertebral space. Local tenderness, and surgical emphysema	Retrosternal chest pain (lateralizing to the side of perforation) and dysphagia (odynophagia) * If pleural space is involved → pneumothorax or pleural effusion → shock, SOB and cyanosis. * Mediastinitis and septic shock	Epigastric pain that radiates to the back if the perforation is posterior. Peritonitis and a rigid abdomen.	

If we suspect perforation we need to investigate immediately or we lose the patient.

- **Consistently associated with**: neck, substernal, or epigastric pain.
- Other associated symptoms: vomiting, hematemesis, or dysphagia.
- **History of**: trauma | advanced esophageal cancer | violent retching (ex.Boerhaave's syndrome) | swallowing a foreign body | recent instrumentation → must raise the question of esophageal perforation.
- With increased mediastinal and pleural contamination → hemodynamic instability.
- On examination:
 - o Tachypnea, tachycardia, and a low-grade fever but have no signs of perforation.
 - Subcutaneous air in the neck or chest, shallow decreased breath sounds, or a tender abdomen.
- Laboratory values of significance:

 WBCs count &

 salivary amylase in blood or pleural fluid.

¹⁷ One of the most important DDx for anyone who collapses with chest pain, shortness of breath or vomiting.

¹⁸ Boerhaave syndrome: transmural perforation of the esophagus | Mallory-Weiss syndrome: nontransmural esophageal tear that is also associated with vomiting

¹⁹ Any patient complains of chest pain, fever or vomiting after endoscopy think of perforation.



Imaging

•diagnosis of an esophageal perforation may be made radiographically.

- CXR (appropriate next step): may demonstrates hydropneumothorax or pneumomediastinum.
 Normally the mediastinum should be clear of air (except in the trachea), so if you see air means there is perforation.
- Contrast esophagram with water soluble contrast (ex. Gastrografin), if you suspect a perforation in the abdominal part of the esophagus.
- Barium swallow is contraindicated if you suspect abdominal esophageal perforation (because it is very toxic to the abdominal peritoneum and it can cause peritonitis).
 - Most perforations (found above GEJ on left lateral wall of esophagus) \rightarrow results in 10% false-negative rate if patient is not in lateral decubitus position.
- Chest CT shows mediastinal air and fluid at the site of perforation.
- Surgical endoscopy:
- Has to be performed if (1) esophagram is negative (2) operative intervention is planned.
- Endoscopy suggests mucosal injury if:
- (1) blood, mucosal hematoma, or a flap is seen. (2) esophagus is difficult to insufflate.



Treatment

If you suspect a perforation, the most important next step (Before CXR) is to make sure that the patient stays NPO (Nothing by mouth), Because he will be in a risk of Mediastinitis and sepsis.

After confirming the perforation, the patient can be either managed medically (conservative) or Surgically:

- if the patient is stable we keep him NPO + iv fluid + antibiotic and keep the patient in the ICU, after a week we check again and see if it closed or not. Most of the patient if the perforation is small it will close.
- if the patient unstable / septic / large perforation / didn't close : we go for surgery.
- If perforation is suspected → appropriate resuscitation with placement of large-bore peripheral IV catheters, a urinary catheter, and a secured airway (before patient is sent for diagnostic testing)
 Note that perforation can progress rapidly to hemodynamic instability and shock if patient is not stabilized.
- 2. IV fluids and broad-spectrum antibiotics are started immediately, and the patient is monitored in an ICU.
- 3. The patient is kept NPO, and nutritional access needs are assessed (total parenteral nutrition TPN)



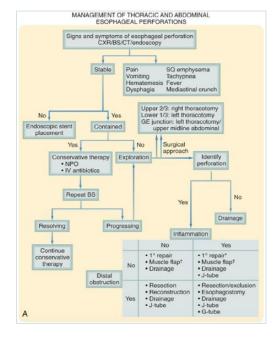
Cont. Treatment:

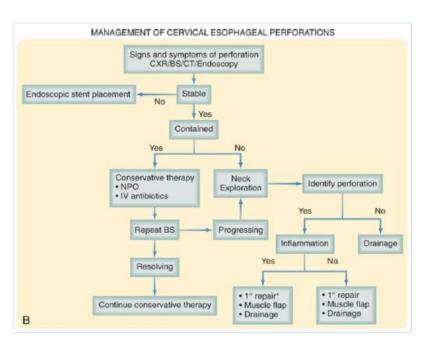
4. Further management:

- Dependent on several variables: stability of the patient, extent of contamination, degree of inflammation, underlying esophageal disease, and location of perforation.
- Surgery is not indicated for every patient. The most critical variable that determines the surgical management of an esophageal perforation is the **degree of inflammation surrounding the perforation**. (small perforation treated conservatively | big perforation treated with surgical intervention)
 - Patient presents within 24 hours of perforation & minimal inflammation → primary surgical repair.
 - Perforation progresses with time (inflammation & friable tissues) → patient may NOT be amenable to primary repair.
 - The final variable to consider in the surgical management of esophageal perforations is the location of the perforation.
- Let's say that you did an Upper GI endoscopy for a patient, and after 6 hours he complains of chest pain a have a fever, You think of perforation. What is the most important symptom that can give you a good clue about the perforation?
 - Odynophagia (pain with swallowing), You have to ask the patient about it.

<u>Summary:</u> if you suspect perforation **(1)** Resuscitation & stabilization **(2)** Diagnostic testing **(3)** IV fluids, IV Abx, NPO (TPN) in ICU **(4)** Further management varies: **a)** Surgical repair with large perforations & minimal inflammation <24 hrs **b)** Conservative management is enough with small perforations & progressive inflammation >24 hrs.

You Don't need this







LEIOMYOMA 🛇

Dr. Sami did not explain this during the lecture but it is mentioned in the slides & objectives.

- Leiomyomas constitute 60% of all benign esophageal tumors.
- Men > women | 4th and 5th decades.
- They are found in the distal two thirds of the esophagus more than 80% of the time
- They are usually solitary and remain intramural, causing symptoms as they enlarge.
- Recently, they have been classified as a gastrointestinal stromal tumor (GIST):
 - Most common mesenchymal tumors of the gastrointestinal tract and can be benign or malignant.
 - Nearly all GIST tumors occur from mutations of the c-KIT oncogene, which codes for the expression of c-KIT (CD117).
- All leiomyomas are slow-growing benign tumors with malignant transformation being rare (progressively symptomatic with time if it becomes malignant)

Symptoms and Diagnosis

- · Many leiomyomas are asymptomatic.
- Dysphagia, bleeding and pain are the most common symptoms (can result from even the smallest tumors).
- A chest radiograph is NOT usually helpful to diagnose leiomyomas, but on barium esophagram, a leiomyoma has a characteristic appearance.
- During endoscopy, extrinsic compression is seen, and the overlying mucosa is noted to be intact
- Diagnosis also can be made by an endoscopic ultrasound (EUS), which will demonstrate a hypoechoic mass in the submucosa or muscularis propria.



Treatment

- Leiomyomas are slow-growing tumors with rare malignant potential that will continue to grow and become progressively symptomatic with time.
- Although observation is acceptable in patients with small (<2 cm) asymptomatic tumors or other significant comorbid conditions, in <u>most patients</u>, **surgical resection is advocated**.
- Surgical enucleation of the tumor remains the standard of care and is performed through a thoracotomy or with video or robotic assistance.
- The mortality rate is less than 2%, and success in relieving dysphagia approaches 100%.



CARCINOMA OF THE ESOPHAGUS Helpful Video 4:53

- Esophageal cancer is the fastest growing cancer in western countries.
- It is of TWO common types:

Squamous cell carcinoma	Adenocarcinoma
Accounts for most esophageal cancers diagnosed.	In the US and Western countries, esophageal adenocarcinoma is noted in up to 70%. More common in white males
 Arise from the squamous mucosa that is native to the esophagus and is found in upper and middle third of esophagus 70% of the time. 	There are a number of factors that are responsible for this shift in cell type: Increasing incidence of GERD. Western diet.
Caustic ingestion, achalasia, bulimia, tylosis (an inherited autosomal dominant trait), Plummer-Vinson syndrome (also called Paterson—Brown—Kelly syndrome or sideropenic dysphagia, is a rare disease characterized by difficulty in swallowing, iron deficiency anemia, glossitis, cheilosis and esophageal webs), external-beam radiation, and esophageal diverticula all have known associations with squamous cell cancer.	 ○ Increased use of acid-suppression medications. • (1) Adaptive measure: squamous-lined distal esophagus → metaplastic columnar epithelium (Barrett's esophagus) (2) Progressive changes from Barrett's esophagus → dysplastic cells → development of esophageal adenocarcinoma.

Risk factors:

The most important risk factors for adenocarcinoma of the oesophagus are reflux and obesity.

- Smoking and alcohol both increase the risk for foregut cancers by 5-fold combined.
- Food additives: nitrosamines (found in pickled), smoked foods and long-term ingestion of hot liquids.
- Intake of caffeine, fats, acidic and spicy foods \rightarrow decreased tone in LES and \rightarrow increase in reflux. Others: Leukoplakia, achalasia, consumption of salted fish.
- The 5-year survival rate varies: 70% with polypoid lesions (good), 15% with advanced tumors (poor).

Symptoms

- Early-stage cancers may be asymptomatic or mimic symptoms of GERD.
- Most patients with esophageal cancer present with progressive dysphagia and weight loss.
- Because of the distensibility of the esophagus, a mass can obstruct two thirds of the lumen before symptoms of dysphagia are noted.
- · Signs of advanced disease:
 - Ominous signs of advanced disease: choking, coughing, aspiration from a tracheoesophageal fistula, and hoarseness (vocal cord paralysis from direct invasion into the recurrent laryngeal nerve)
 - Systemic metastases: to liver (hepatomegaly/ascites/abdominal mass/jaundice), bone (excessive pain), and lung (respiratory symptoms). + lymph nodes (supraclavicular lymph node in particular²⁰)

Other general features of malignancy: anorexia, anaemia and lassitude (lethargy)

Diagnosis

- There is a plethora of modalities available to diagnose and stage esophageal cancer.
- Radiologic tests, endoscopic procedures, and minimally invasive surgical techniques all add value to a solid **staging** workup in a patient with esophageal cancer.

Esophageal Carcinoma Apple core lesion

²⁰ Esophageal cancer metastasize to right supraclavicular lymph node | Gastric cancer metastasize to left supraclavicular (Virchow's node)



Esophagram

NOTE: It is mentioned in the slides and by Dr. Sami that esophagram is the initial investigation of **dysphagia**. HOWEVER we have asked two expert GI consultants who specialise in endoscopy, searched books & articles they all say after taking good full history endoscopy is the next step "generally speaking" unless you suspect other ddx (ex. achalasia) then you add esophagram on top of your list of investigations.

- Is able to differentiate intra<u>luminal</u> from intra<u>mural</u> lesions and to discriminate between **intrinsic** (from a mass protruding into the lumen) and **extrinsic** (from compression of a structures outside the esophagus) compression.
- The classic finding: apple-core lesion in patients with esophageal cancer.
- Esophagram is **not specific** for cancer but it is a good first test to perform in patients presenting with dysphagia and a suspicion of esophageal cancer.

★ Endoscopy "first-line & most accurate investigation for dysphagia"

- The diagnosis of esophageal cancer is made best from an endoscopic biopsy.
- Any patient undergoing surgery for esophageal cancer <u>must</u> have an endoscopy performed by the
 operating surgeon before entering the operating room for a definitive resection.
 Even if the diagnosis is made initially by barium swallow, it must always be confirmed by endoscopy
 and biopsy.

• Computed Tomography: "to stage malignancy"

 CT scan of the chest and abdomen is important to assess the length of the tumor, thickness of the esophagus and stomach, regional lymph node status and Distant disease to the liver and lungs.

• Positron Emission Tomography:

- PET scan evaluates the primary mass, regional lymph nodes, and distant disease.
- Sensitivity and specificity > CT; however, they remain **low** for definitive staging.
- Endoscopic Ultrasound: Mostly used for staging the 'T' and 'N' component of TNM staging.
 - Most critical component of esophageal cancer staging.
 - The information obtained from EUS will help Guide both medical & surgical therapy.
 - Obtain biopsy samples of masses & lymph nodes in paratracheal, subcarinal, paraesophageal, celiac region.

Treatment (depends on the staging)

- Chemotherapy.
- · Radiation therapy.
- Chemoradiotherapy.
- · Surgical resection.

We have to do staging for the tumor, and we have Three options:

- 1-Either the tumor is Localized with no metastasis: The treatment is surgical resection.
- 2-If there is lymph node involvement: the treatment will be chemoradiotherapy/Radiotherapy (Neoadjuvant) followed by surgical resection.
- 3-If there is distant metastasis treatment is chemoradiotherapy (NO SURGERY)



EXTRA CASES

EXTRA CASES:

A 42-year-old woman has heartburn after meals and a sour taste in her mouth. For the past 4 to 6 months she has had symptoms several times per week. Symptoms are worse when she lies down or bends over. Antacids help somewhat. The patient has no dysphagia, vomiting, abdominal pain, exertional symptoms, melena, or weight loss. GERD

54-year-old woman with reflux disease and columnar epithelium on distal esophageal biopsy. Barrett's esophagus

54-year-old woman with dysphagia (to both liquid and solids) and intermittent chest pain; food "comes up" after falling asleep at night; manometry reveals high LES pressures that do not decrease with swallowing; absent esophageal peristalsis.

Achalasia

A 45-year-old man with minimal past medical history began to have difficulty swallowing with the sensation of food getting stuck in his throat. Symptoms were evident with solid food, and he would often unpredictably regurgitate undigested food. This was particularly difficult in social and professional situations. He took antireflux medication for heartburn and often felt these pills got stuck in his throat. He also noted increasing episodes of halitosis/bad breath and frequently woke in the middle of the night with a coughing fit.

Zenker's Diverticulum

A 19-year-old college student presents to the emergency department with excruciating retrosternal chest pain that radiates to the back. She describes the pain as sharp and worsens with swallowing. She reports that her symptoms began after vomiting 1 hour ago. Medical history is significant for anorexia nervosa. Her temperature is 100.4°F (38°C), blood pressure is 135/90 mmHg, pulse is 105/min, and respirations are 20/min. On physical exam, there is crepitus upon chest palpation. A barium esophagram is performed and shows leakage of water-soluble contrast from the lower thoracic esophagus.

Boerhaave Syndrome.

A patient had an endoscopy, then after 6 hours he experienced fever, chest pain, dysphagia, upper GI bleeding and odynophagia. What's the diagnosis? Endoscopy was complicated by perforation.

Q1: What investigations you would do?

- Chest x-ray → pneumomediastinum (air within the mediastinum).
- Barium swallow → extravasation.

Q2: What is your management?

- 1. NPO
- 2. IV fluids (D5 0.5NS)
- 3. Abx
- Surgery
- If a patient is on IV fluids and can have nothing per mouth, after 2-3 days you should give him total parenteral nutrition (TPN) because it has fat, glucose and proteins that are important for the patient nutrition. A patient can rely on TPN only up to one year.

Mnemonic for Plummer-vinson syndrome (EXTRA):

The plummer "vinson" **DIGS** with his d**O**g for an **IRON** pipe.

Dysphagia | Iron deficiency anemia | Glossitis | Squamous cell carcinoma | Oesophageal web

IRON: Treatment with iron supplements | endoscopic dilatation & biopsy to rule out cancer.



Summary

GASTROESOPHAGEAL REFLUX DISEASE	BARRETT'S ESOPHAGUS	ACHALASIA
Definition: It is often a chronic and relapsing condition characterized by symptoms OR mucosal damage produced by abnormal reflux of gastric contents.	Definition: A condition whereby an intestinal, columnar epithelium WITH goblet cells! replaces the stratified squamous epithelium that normally lines the distal esophagus (intestinal metaplasia).	Definition: It is characterized by partial or complete degeneration of the myenteric plexus The main feature is failure of relaxation of the lower esophageal sphincter.
hiatal hernia: GERD is often associated with a hiatal hernia: -Type I (Important) Sliding Hiatal Hernia (Strong association with GERD) -Type II Rolling hernia No acid reflux -Type III & IV Rolling hernia May be associated with GERD	 symptoms: asymptoms of GERD: Heartburn, regurgitation, acid or bitter taste in the mouth, excessive belching, and indigestion. Recurrent respiratory infections 	Clinical features: 1. Progressive dysphagia (main symptom). 2. Regurgitation. 3. Others: chest pain Heartburn Halitosis Weight loss (in end-stage disease).
Clinical Presentations of GERD: substernal Heartburn and/or regurgitation: Postprandial. Aggravated by change of position.	Diagnosis: • Diagnosis is made by: Endoscopy & pathology (histopathology).	Diagnosis: CXR: may show air-fluid level. Barium study: (1) classic finding bird's beak (2) dilated esophagus (3) air-fluid level Upper endoscopy Difficulty traversing the GEJ → suspicion of pseudoachalasia due to neoplastic infiltration of the distal esophagus. Esophageal manometry (Most accurate) Manometric variants of achalasia
Diagnostic Tests for GERD: Barium swallow Endoscopy. Ambulatory pH manometry "most diagnostic": 24-hour-pH monitor. Esophageal manometry Treatment: Lifestyle Modifications (The most important) Acid Suppression Therapy for GERD. Anti-Reflux Surgery: Nissen fundoplication "most common"	Treatment: • Barrett's esophagus 1-Yearly surveillance endoscopy is -recommended in all patients with a diagnosis of BE. 2-Photodynamic therapy (PDT) is the most common ablative method used to treat BE.	Treatment: • Primary therapeutic goal: to reduce LES basal pressure. • Primary desired outcome: symptoms relief, particularly of dysphagia. • Treatment options: medical therapy, botulinum toxin injection, pneumatic dilation "first-line", and surgical myotomy.



ESOPHAGEAL DIVERTICULA	ESOPHAGEAL PERFORATION	CARCINOMA OF THE ESOPHAGUS
 A result of: (1) primary motor disturbance. (2) abnormality of the UES or LES. The three most common sites: Pharyngoesophageal (Zenker's). Peribronchial (mid-esophageal). Epiphrenic. 	Perforation of the esophagus is a surgical emergency.	It is of TWO common types: Squamous cell carcinoma: Caustic ingestion, achalasia, bulimia, tylosis Plummer-Vinson syndrome external-beam radiation, and esophageal diverticula all have known associations with squamous cell cancer. Adenocarcinoma: (1) Adaptive measure: squamous-lined distal esophagus → metaplastic columnar epithelium (Barrett's esophagus) (2) Progressive changes from Barrett's esophagus → dysplastic cells → development of esophageal adenocarcinoma.
True diverticulum: ALL LAYERS Results from: External inflammatory mediastinal lymph nodes adhering to the esophagus. False diverticulum: Mucosa and submucosa ONLY, Caused by elevated intraluminal pressures It includes: Zenker's & epiphrenic diverticulum	Symptoms Cervical perforations: Neckache, stiffness. Thoracic perforations: Retrosternal chest pain. Abdominal perforations: Epigastric pain.	Symptoms • Early-stage cancers may be asymptomatic or mimic symptoms of GERD. • Signs of advanced disease: -Ominous signs of advanced disease: choking, coughing, aspiration from a tracheoesophageal fistula, and hoarseness -Systemic metastases: to liver (jaundice), bone (excessive pain), and lung (respiratory symptoms).
Diagnosis: • Diagnosis is made by barium esophagram.	Diagnosis: Imaging: -CXR (appropriate next step): may demonstrates hydropneumothorax or pneumomediastinum. -Contrast esophagram with water soluble contrast (ex. Gastrografin)	Diagnosis: • Esophagram The classic finding: apple-core lesion in patients with esophageal cancer. ★ Endoscopy "first-line & most accurate investigation for dysphagia"
Treatment:	Treatment: 1- IV fluids and broad-spectrum antibiotics. 2- The patient is kept NPO	Treatment: • Chemotherapy. • Radiation therapy. • Chemoradiotherapy. • Surgical resection.



Questions

- 1-Most common site for squamous cell carcinoma of the esophagus is?
- A) Upper 1/3
- B) Middle 1/3
- C) Lower 1/3
- D) A and B
- 2- 70 year old male newly diagnosed with pharyngoesophageal(zenker's) diverticulum, which one of the following most likely the presenting symptoms?
- A)Abdominal pain
- B)Haematemesis
- C)Halitosis
- D)Vomiting
- 3- A 50 year old male presented to you in the clinic with history of heartburn and hoarseness for the last two years aggravated by food & lying down associated with cough and bitter taste. He is obese and a Smoker.
- (3-A) What is the diagnosis?
- A) Esophageal spasm
- B) GERD
- C) Esophageal perforation
- D) Adenocarcinoma
- (3-B) What most common use in endoscopic therapy for this disease?
- A) Endoscopic mucosal resection
- B) Esophageal resection
- C) Enteryx
- D) Nissen fundoplication
- (3-C) 8 months later, you did endoscopy for the patient, 6 hour post endoscopy, the patient start to complain of chest pain and fever, What is the the first thing that comes to your mind?
- A) Esophageal perforation
- B) Squamous cell carcinoma
- C) Esophageal diverticula.
- D) Achalasia.
- 4-What is the highest sensitive method to diagnosis of achalasia?
- A) CT.
- B) Barium study .
- C) Endoscopy.
- D) Esophageal manometry.



5- 70 years old male was diagnosed with Barrett's esophagus 12 years ago, he presented to your clinic complaining of Dysphagia and Weight loss What is the most likely diagnosis?

- A) Achalasia
- B) GERD
- C) Adenocarcinoma
- D) Esophageal perforation.

6-The Most accurate investigation for GERD is:

- A) Ambulatory pH monitoring
- B) Barium swallow
- C) Endoscopy
- D) Clinical picture

7-What is the most common ablative method used to treat Barrett's esophagus?

- A) Photodynamic therapy
- B) Surgery
- C) Medical
- D) Pneumatic dilation

Answers:										
1: D	2: C	(3-A): B	(3-B): D	(3-C): A	4: D	5: C	6: A	7: A		