



Benign Esophageal Disease

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Objective:

Not given by the doctor

According to dr.Sami the lecture is enough..



Not mentioned by the dr

Color Index:

-Doctor's Notes **-Surgery Recall** **-Doctor's Slides** **-Important** **-Extra**

Anatomy of esophagus

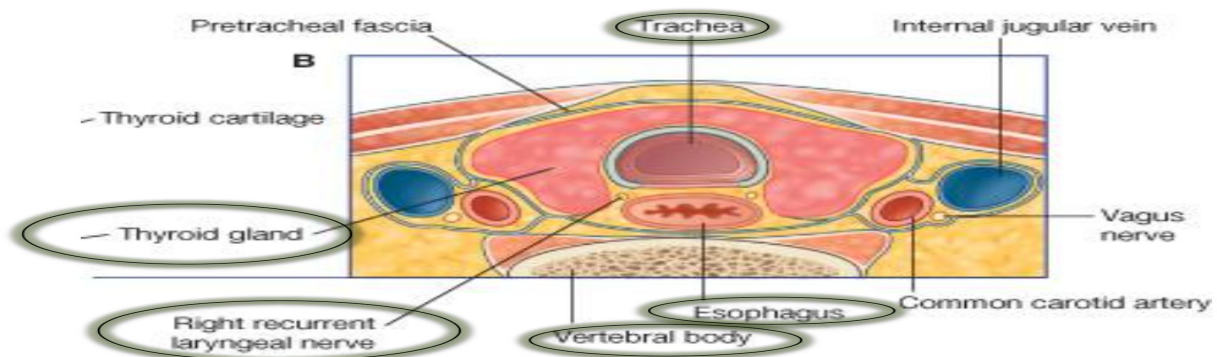
Taken from team 433 Anatomy team

Relations

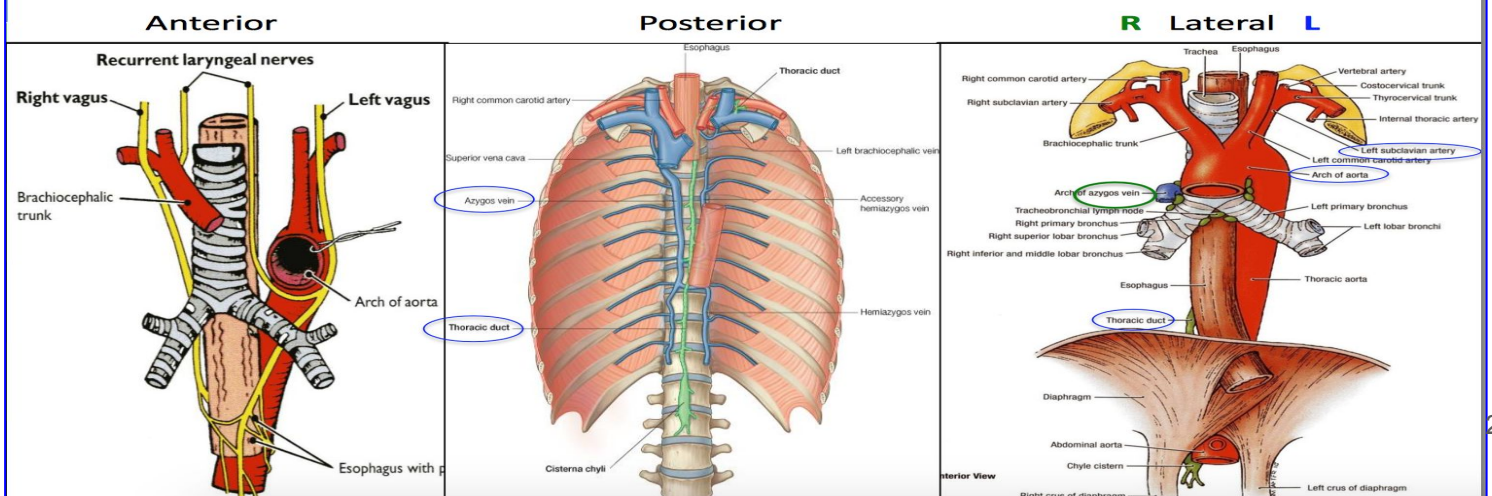


Part	Anterior	Posterior	Laterally
Cervical	Trachea and the recurrent laryngeal nerves	Vertebral column	Lobes of the Thyroid gland
Thoracic	<ol style="list-style-type: none"> ① Trachea ② Left recurrent laryngeal nerve ③ Left principal bronchus ④ Pericardium ⑤ Left atrium 	<ol style="list-style-type: none"> ① Bodies of the thoracic vertebrae ② Thoracic duct ③ Azygos vein ④ Right posterior intercostal arteries ⑤ Descending thoracic aorta (at the lower end) 	<p><u>On the Right side:</u></p> <ul style="list-style-type: none"> • Right mediastinal pleura • Terminal part of the azygos vein. <p><u>On the Left side:</u></p> <ul style="list-style-type: none"> • Left mediastinal pleura • Left subclavian artery • Aortic arch • Thoracic duct
Abdomen	Left lobe of liver	Left crus of diaphragm	_____

Cervical part of Esophagus



Thoracic part of Esophagus

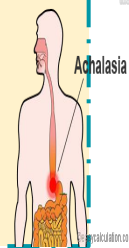


Clinical scenario

Ahmad 24 yr old gentleman came to your clinic complaining of 2 years of difficulties in swallowing (dysphagia), he said that: "I need to drink plenty of water or أقامز so the food passes"

As the name indicates it's like a كليجة blocking the food follow through the esophagus..

يقامز عشان الاكل ينزل مع الجاذبية



Achalasia

- Achalasia is an uncommon disease of esophageal motility disorder ⇒ Idiopathic disease affects the neurogenic blocks within the esophagus wall causing aperistalsis + Hypertensive Lower esophageal sphincter (LES) and failure of sphincter relaxation..

Characteristics

- It is characterized by degeneration of the myenteric neurons that innervate (LES) and esophageal body.
- the pathogenesis :autoimmune, Viral, Familial

Clinical features

- Most commonly presents in patients between the ages of 25 and 60 years
- an equal male-to-female gender distribution
- Dysphagia to solids and liquids is the most common presenting symptom, experienced by greater than 90% of patients
- Regurgitation is the second most common symptom, occurring in approximately 60% of patients
- Nocturnal regurgitation of esophageal contents can lead to nighttime cough and aspiration
- Weight loss occurs in end-stage disease
- Chest pain is reported in 20% to 60% of patients
- Heartburn is reported in a large number of patients with achalasia (30% of achalasia patients)
- may be related to direct irritation of the esophageal lining by retained food, pills, or acidic byproducts of bacterial metabolism of retained food

Diagnosis

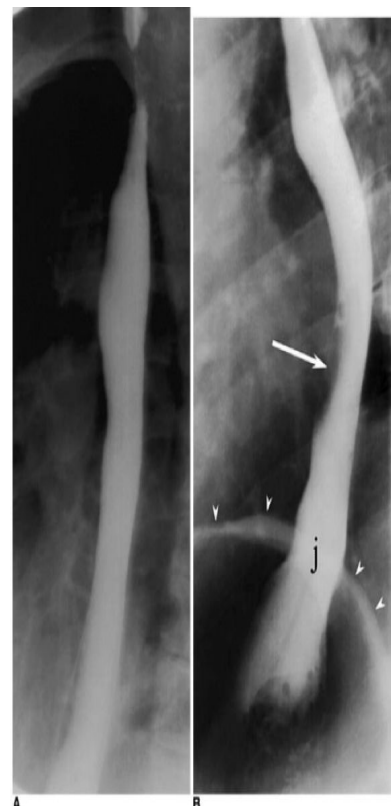
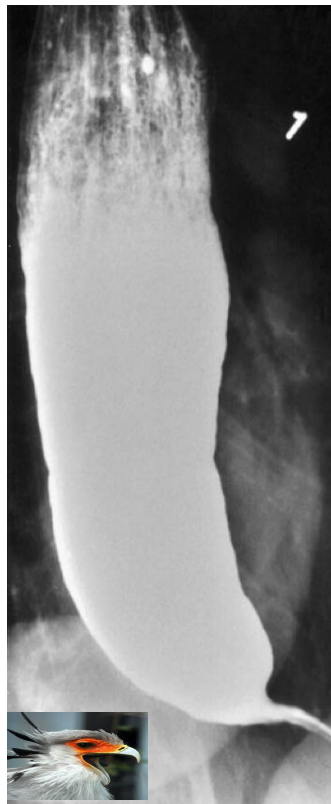
- CXR may show air-fluid level.
- Barium (**swallow**) study quite dilated, and an air-fluid (**obstruction**) level may be 2ry to retained secretions. The classic finding is a gradual tapering at the end of the esophagus, similar to a bird's beak

منقار الطائر

- Upper endoscopy is the next diagnostic test in a patient with dysphagia or suspected achalasia
- Findings can include :
 - dilated esophagus with retained food or secretions
 - normal in as many as 44% of patients with achalasia
- Difficulty traversing the (Gastroesophageal Junction)GEJ should raise suspicion for pseudoachalasia due to neoplastic infiltration of the distal esophagus

Diagnosis Cont.

- Esophageal manometry has the **highest sensitivity** for the diagnosis of achalasia :
 - aperistalsis of the distal esophageal body
 - incomplete or absent LES relaxation
 - hypertensive LES
 - Manometric variants of achalasia exist
 - The best known is vigorous achalasia
 - defined by the presence of normal to high amplitude esophageal body contractions in the presence of a nonrelaxing LES
 - Vigorous achalasia may represent an early stage of achalasia
 - Chagas' disease is a parasitic infection caused by *Trypanosoma cruzi* which can cause secondary achalasia
 - The most concerning secondary etiology is cancer, which can present as achalasia through mechanical obstruction of the GEJ
 - Additional secondary forms of achalasia exist
 - An increasingly recognized etiology is post fundoplication* achalasia caused by mechanical obstruction of the GEJ by the fundoplication or diaphragmatic crural closure
- *is a surgical procedure to treat gastroesophageal reflux disease (GERD) and hiatal hernia
- Similar cases have been described following bariatric surgery using a gastric band device which constricts the proximal stomach a few centimeters below the LES



- The primary therapeutic goal in achalasia is to:
 - Reduce the LES basal pressure**
- Treatment options include
 - medical therapy,
 - botulinum toxin injection,
 - pneumatic dilation,
 - and surgical myotomy
- Symptom relief, particularly relief of dysphagia, is accepted as the primary desired outcome

1. Medical Therapy

- | | |
|---|---|
| <ul style="list-style-type: none"> • Nitrates (<i>relaxes the smooth muscles</i>) were first recognized as an effective treatment of achalasia | <ul style="list-style-type: none"> • Calcium channel antagonists have a better side-effect profile when compared with nitrates |
| <ul style="list-style-type: none"> - their systemic vasodilatory effects and headaches limit their tolerability by patients | <ul style="list-style-type: none"> - 30% of patients report adverse side effects including peripheral edema, hypotension, and headache |

2. Botulinum Toxin

- injected into the LES targets the excitatory, acetylcholine-releasing neurons that generate LES basal muscle tone
- is easy to administer and associated with relatively few side effects
- It is apparent that, with repeated injections, the response rates reported are similar or lower to that achieved with the initial injection
- 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%
- Given the limitations of the efficacy and durability of response, botulinum toxin is generally reserved for use in patients who are not candidates for more invasive treatments

3. Pneumatic Dilation

- pneumatic dilation remains one of the most effective **first-line** therapies for achalasia
- Long-term follow-up studies reported significant symptom relapse of 50% at 10 years
- Complications of pneumatic dilation exist :
 - Gastroesophageal reflux 25-35%
 - Esophageal perforation 3 %
 - **Doesn't last long**

Treatment



4. Surgical Therapy

Treatment

- has success rates in excess of 90% with hospital stays averaging only a few days
 - **acid exposure** is a known complication of surgical intervention for achalasia
 - Even with a successful myotomy, it is expected that patients will have some degree of dysphagia as a consequence of esophageal peristaltic dysfunction
-
- **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
 - laparoscopic Heller myotomy demonstrated excellent results, with 98% of patients reporting symptomatic improvement at 5.3 years

Refractory Achalasia

- In patients with achalasia that is refractory to therapy with Heller myotomy, options are limited
- Although esophagectomy is considered in patients with marked dilation and sigmoid deformity, such patients may respond to Heller myotomy

Complications

- The primary complications of achalasia are related to the functional obstruction rendered by the nonrelaxing LES and include progressive malnutrition and aspiration.
- Uncommon but important secondary complications of achalasia include the formation of epiphrenic diverticula and esophageal cancer.
- There is an established link between achalasia and esophageal cancer, most commonly squamous cell carcinoma
- The overall prevalence of esophageal cancer in achalasia is approximately 3% with an incidence of approximately 197 cases per 100,000 persons per year

Surgery Versus Pneumatic Dilation

- Several retrospective and prospective studies have reported superior success rates for **surgery** when compared with pneumatic dilation
- a study of outcomes of 1181 patients treated with pneumatic dilation with that of 280 patients treated with Heller myotomy as initial therapy showed that the risk of subsequent therapeutic intervention at 10 years was significantly higher with dilation (64%) when compared with surgery (38%)

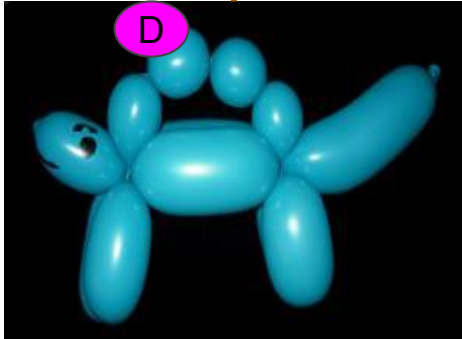
Esophageal Diverticula:

- most diverticula are a result of a **primary motor disturbance or an abnormality of the UES or LES**
- can occur in several places along the esophagus
- The three most common sites of occurrence are:

1-pharyngoesophageal (Zenker's)

2-peribronchial (mid esophageal)

3-epiphrenic

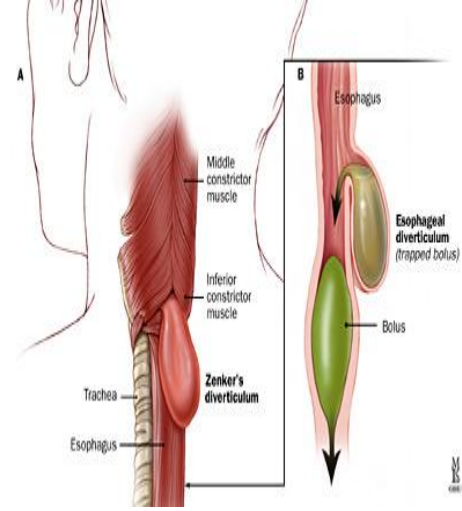
	True Diverticula	False Diverticula
Layers involvement	True diverticula involve all layers of the esophageal wall, including mucosa, submucosa, and muscularis	A false diverticulum consists of mucosa and submucosa only
Definition	Traction, or true, diverticula result from external inflammatory mediastinal lymph nodes adhering to the esophagus	Pulsion diverticula are false diverticula that occur because of elevated intraluminal pressures generated from abnormal motility disorders
		<p>Zenker's diverticulum and an epiphrenic diverticulum fall under the category of false, bulging diverticula.</p> <p>What happens is?? Normally when one area contracts in the esophagus the other will relaxes.. But, when two areas contract like "a balloon" at the same time ⇒ increase intraluminal pressure ⇒ bulging of the mucosa ⇒ diverticula Blue= twisting esophagus Pink= Diverticulum arising from the esophagus</p>

Pharyngoesophageal (Zenker's) Diverticulum:

case scenario:

70 Y/O male came to the clinic with his wife, she complains from his mouth bad smell. She also noticed that when he eats anything he rotate his neck and press on the food passes .

he have diverticulum ,common in elderly patient and it's called (zenker's diverticulum) diagnosed with barium swallow.

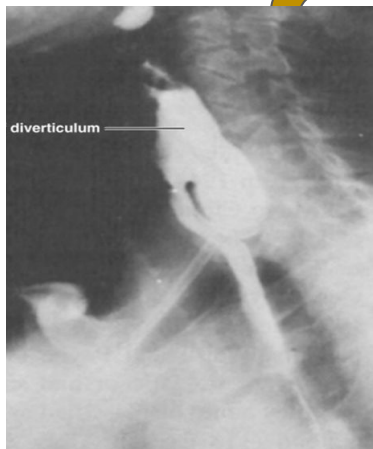


- is the most common esophageal diverticulum found today
- It usually presents in older patients in the **7th decade** of life
- found herniating into Killian's triangle, between the oblique fibers of the thyropharyngeus muscle and the horizontal fibers of the cricopharyngeus muscle

Symptoms and Diagnosis:

- Commonly, patients complain of a **sticking in the throat**.
- nagging cough, excessive salivation, and intermittent dysphagia often are signs of progressive disease
- As the sac increases in size, regurgitation of **foul-smelling**, undigested material is common
- Halitosis, voice changes, retrosternal pain, and respiratory infections are especially common in the elderly population
- The most serious **complication** from an untreated Zenker's diverticulum **is aspiration pneumonia or lung abscess**
- Diagnosis is made by barium esophagram

Neither esophageal manometry nor endoscopy is needed to make a diagnosis of Zenker's diverticulum.



Treatment

- **Surgical or endoscopic repair of a Zenker's diverticulum is the gold standard of treatment**
- Open repair involve :
 - myotomy of the proximal and distal thyropharyngeus and cricopharyngeus muscles
 - diverticulectomy or diverticulopexy are performed through an incision in the left neck
- An alternative to open surgical repair is the endoscopic Dohlman procedure
- Endoscopic division of the common wall between the esophagus and the diverticulum using a laser or stapler has also been successful

normal: linear contrast movement as long as esophagus
 abnormal:
 here sames as pouch in contractility of the esophagus (upper esophageal sphincter & cricopharyngeal muscle)=>abnormal contraction of both muscles ,what will increase the esophagus=> mucosa will bulge between muscle fibers .
 will increase in size gradually تصير زي الحوصلة
 (false diverticulum)=2 layers
 treat with diverticulectomy+myotomy (to decrease the pressure

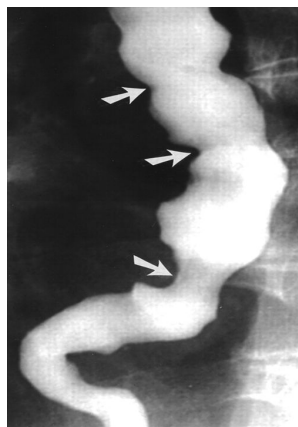


Diffuse Esophageal Spasm

- **DES is a hypermotility disorder** of the esophagus
- is seen most often **in women** and is often found in patients with multiple complaints
- The basic pathology is related to a motor abnormality of the esophageal body that is most notable in the lower two thirds of the esophagus
- **the esophageal contractions are repetitive**, simultaneous, and of high amplitude

Symptoms and Diagnosis

- The clinical presentation of DES is typically that of **chest pain** and **dysphagia**
- These symptoms may be related to eating or exertion and may mimic angina
- Patients will complain of a squeezing pressure in the chest that may radiate to the jaw, arms, and upper back
- The symptoms are often pronounced during times of heightened emotional stress
- Regurgitation of esophageal contents and saliva is common, but acid reflux is not
- acid reflux can aggravate the symptoms, as can cold liquids
- **irritable bowel syndrome and pyloric spasm, may accompany DES, whereas other gastrointestinal problems, such as gallstones, peptic ulcer disease, and pancreatitis, all trigger DES**
- The diagnosis of DES is made by an **esophagram and manometric studies**



Corkscrew appearance

Treatment

- the mainstay of treatment for DES is nonsurgical, and **pharmacologic or endoscopic intervention is preferred**
- Surgery is reserved for patients with recurrent incapacitating episodes of dysphagia and chest pain who do not respond to medical treatment

GASTROESOPHAGEAL REFLUX DISEASE

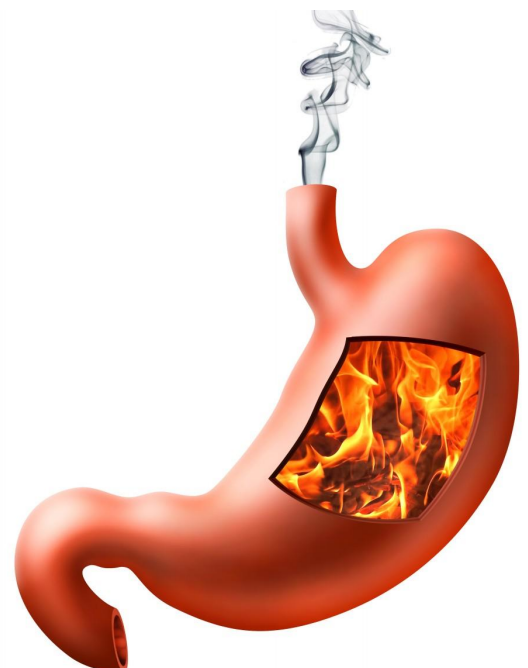
- GERD is often associated with a hiatal hernia
- the most common is the type I hernia, also called a *sliding hiatal hernia*
- Type II and III hiatal hernias are often referred to as *paraesophageal hernias* and they may be associated with GERD
- Type IV when there is other organ herniated into the chest (Spleen ,Colon)

Definition

- *Symptoms OR mucosal damage produced by the abnormal reflux of gastric contents into the esophagus
- *Often chronic and relapsing
- *May see complications of GERD in patients who lack typical symptoms

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



GASTROESOPHAGEAL REFLUX DISEASE

<p>Physiology</p>	<ul style="list-style-type: none"> • LES has the primary role of preventing reflux of the gastric contents into the esophagus • GERD may occur when the pressure of the high-pressure zone in the distal esophagus is too low to prevent gastric contents from entering the esophagus 		
<p>Clinical Presentations</p>	<p>1-Classic GERD</p>	<p>2-Extraesophageal Manifestations</p>	<p>3-Complicated Symptoms</p>
	<ul style="list-style-type: none"> • Substernal burning and or regurgitation • Postprandial • Aggravated by change of position • Prompt relief by antacid 	<p>*Pulmonary</p> <ul style="list-style-type: none"> • Asthma • Aspiration pneumonia • Chronic bronchitis • Pulmonary fibrosis <p>*ENT</p> <ul style="list-style-type: none"> • Hoarseness • Laryngitis • Pharyngitis • Chronic cough • Globus sensation • Dysphonia • Sinusitis • Subglottic stenosis • Laryngeal cancer <p>*Other</p> <ul style="list-style-type: none"> • Chest pain • Dental erosion 	<ul style="list-style-type: none"> • Dysphagia • Difficulty swallowing: food sticks or hangs up • Odynophagia • Retrosternal pain with swallowing • Bleeding
<p>Diagnosis</p>	<ul style="list-style-type: none"> • Barium swallow • Endoscopy • Ambulatory pH monitoring • Esophageal manometry 		

GASTROESOPHAGEAL REFLUX DISEASE

Treatment

1-Lifestyle Modifications

- Elevate head of bed 4-6 inches
- Avoid eating within 2-3 hours of bedtime
- Lose weight if overweight
- Stop smoking
- Modint but smaller meals
- Avoid fatty/fried food, peppermint, chocolate, alcohol, carbonated beverages, coffee and tea
- OTC medications prn

2-Acid Suppression Therapy

H2-Receptor Antagonists (H2RAs)

Cimetidine (Tagamet®)
Ranitidine (Zantac®)
Famotidine (Pepcid®)
Nizatidine (Axid®)

Proton Pump Inhibitors (PPIs)

Omeprazole (Prilosec®)
Lansoprazole (Prevacid®)
Rabeprazole (Aciphex®)
Pantoprazole (Protonix®)
Esomeprazole (Nexium®)

3-Anti-Reflux Surgery

- Indication for Surgery :
 - have failed medical management
 - opt for surgery despite successful medical management (due to life style considerations including age, time or expense of medications, etc)
 - have complications of GERD (e.g. Barrett's esophagus; grade III or IV esophagitis)
 - have medical complications attributable to a large hiatal hernia. (e.g. bleeding, dysphagia)
 - have "atypical" symptoms (asthma, hoarseness, cough, chest pain, aspiration) and reflux documented on 24 hour pH monitoring

4-Endoscopic GERD Therapy

- Endoscopic antireflux therapies
- Radiofrequency energy delivered to the LES
- Stretta procedure
- Suture ligation of the cardia
- Endoscopic plication
- Submucosal implantation of inert material in the region of the lower esophageal sphincter
- Enteryx

CARCINOMA OF THE ESOPHAGUS

Epidemiology

- Esophageal cancer is the fastest growing cancer in the western countries
- Squamous cell carcinoma still accounts for most esophageal cancers diagnosed
- However, in the US, esophageal adenocarcinoma is noted in up to 70% of patients presenting with esophageal cancer
- Squamous cell carcinomas arise from the squamous mucosa that is native to the esophagus and is found in the upper and middle third of the esophagus 70% of the time

Risk Factors

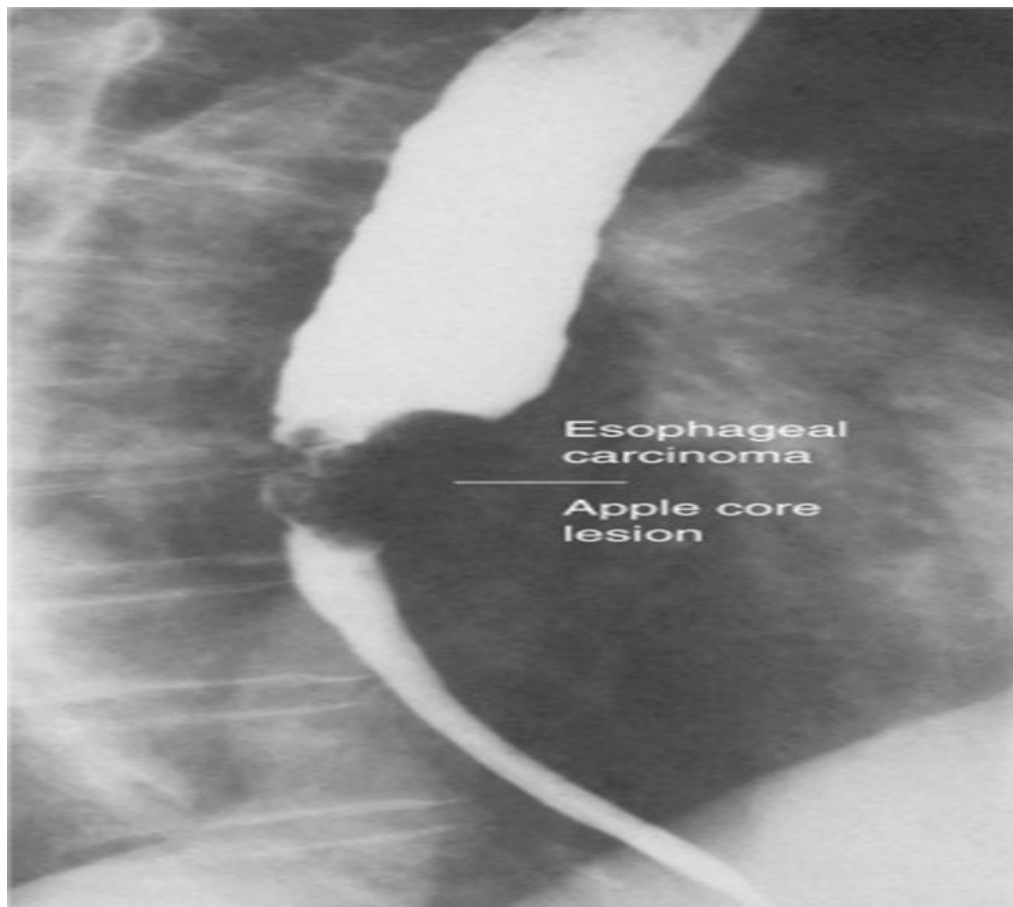
- Smoking and alcohol both increase the risk for foregut cancers by 5-fold. Combined
 - Food additives, including nitrosamines found in pickled and smoked foods, long-term ingestion of hot liquids
 - The 5-year survival rate varies but can be as good as 70% with polypoid lesions and as poor as 15% with advanced tumors.
 - esophageal adenocarcinoma now accounts for nearly 70% of all esophageal carcinomas diagnosed in Western countries
-
- caustic ingestion, achalasia, bulimia, tylosis (an inherited autosomal dominant trait), Plummer-Vinson syndrome, external-beam radiation, and esophageal diverticula all have known associations with squamous cell cancer.
 - There are a number of factors that are responsible for this shift in cell type:
 - Increasing incidence of GERD
 - Western diet
 - Increased use of acid-suppression medications
 - Intake of caffeine, fats, and acidic and spicy foods all lead to decreased tone in the LES and an increase in reflux
 - As an adaptive measure, the squamous-lined distal esophagus changes to become lined with metaplastic columnar epithelium (Barrett's esophagus)
 - Progressive changes from metaplastic (Barrett's esophagus) to dysplastic cells may lead to the development of esophageal adenocarcinoma

CARCINOMA OF THE ESOPHAGUS

- There are 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.

Symptoms

- Early-stage cancers may be asymptomatic or mimic symptoms of GERD
- Most patients with esophageal cancer present with 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
- Choking, coughing, and aspiration from a tracheoesophageal fistula, as well as hoarseness and vocal cord paralysis from direct invasion into the recurrent laryngeal nerve, are ominous signs of advanced disease
- Systemic metastases to liver, bone, and lung can present with jaundice, excessive pain, and respiratory symptoms.



1-Esophagram

- A barium esophagram is recommended for any patient presenting with dysphagia
- is able to differentiate intraluminal from intramural 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 of an apple-core lesion in patients with esophageal cancer is recognized easily (see the picture in the previous slide).
- Although the esophagram will not be specific for cancer, it is a good first test to perform in patients presenting with dysphagia and a suspicion of esophageal cancer

2-Endoscopy

- The diagnosis of esophageal cancer is made best from an endoscopic biopsy
- any patient undergoing surgery for esophageal cancer must have an endoscopy performed by the operating surgeon before entering the operating room for a definitive resection

3-CT

- 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

4-Positron Emission Tomography

- PET scan evaluates the primary mass, regional lymph nodes, and distant disease
- Its sensitivity and specificity slightly exceed those of CT; however, they remain low for definitive staging

5-Endoscopic Ultrasound

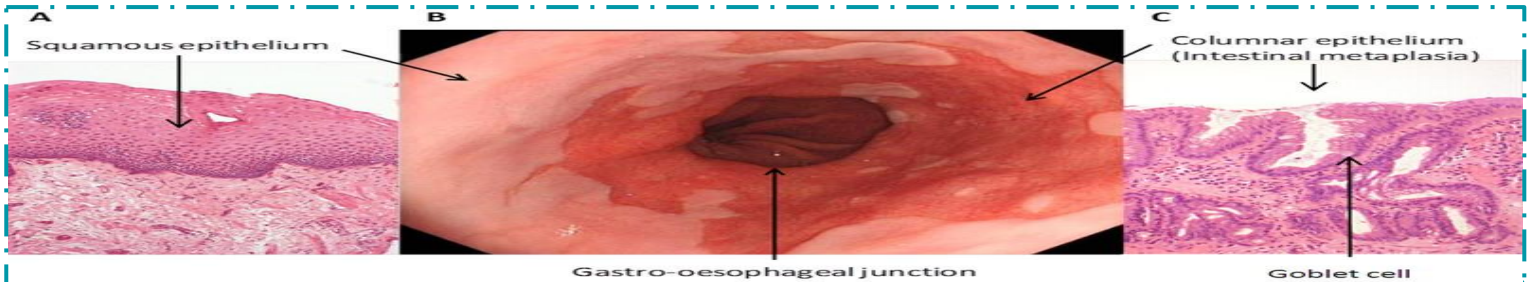
- EUS is the most critical component of esophageal cancer staging.
- The information obtained from EUS will help guide both medical and surgical therapy
- biopsy samples can be obtained of the mass and lymph nodes in the paratracheal, subcarinal, paraesophageal, celiac region

- Chemotherapy
- Radiation therapy
- Chemoradiotherapy
- Surgical resection

Barrett's Esophagus

Epidemiology:

- 70% of patients are men aged 55 to 63 years
- Men have a 15-fold increased incidence over women of adenocarcinoma of the esophagus, but women with Barrett's esophagus are increasing in number as the differences in the Western lifestyle between men and women diminish



In this case when we took biopsy we found metaplasia (from squamous to columnar) + goblet cell ⇒ it's Barrett's
Note: Metaplasia without goblet cell isn't Barrett's ⇒ we call it Intestinal metaplasia

Definition & Pathology

- Barrett's esophagus is a condition whereby an intestinal, columnar epithelium **"with goblet cell"** replaces the stratified squamous epithelium that normally lines the distal esophagus.

the patient may have metaplasia with no association with cancer (e.g. columnar metaplasia "without goblet cells"). this is not Barrett's esophagus).

- Chronic gastroesophageal reflux is the factor that both injures the squamous epithelium and promotes repair through columnar **metaplasia**
- Although these metaplastic cells may be more resistant to injury from reflux, they also are more prone to malignancy
- Ten percent of patients with GERD develop Barrett's esophagus
- the 40-fold increase in risk for developing esophageal carcinoma in patients with Barrett's esophagus
- 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

Symptoms and Diagnosis



- 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 belching, and indigestion are some of the common symptoms associated with GERD
- Recurrent respiratory infections, adult asthma, and infections in the head and neck also are common complaints.
- The diagnosis of BE is made by endoscopy and pathology
- The presence of any endoscopically visible segment of columnar mucosa within the esophagus that on pathology identifies intestinal metaplasia defines BE

Treatment

PPI (help to relieve the symptoms not solve it)+ Advice to(decrease weight and not to eat then sleep directory)⇒ then follow the patient with endoscopy.

- Yearly surveillance endoscopy is recommended in all patients with a diagnosis of Barrett's esophagus
- For patients with low-grade dysplasia, surveillance endoscopy is performed at 6-month intervals for the first year and then yearly thereafter if there has been no change
- Patients undergoing surveillance are placed on acid suppression medication and monitored for changes in their reflux symptoms.
- Controversy surrounds the benefits of antireflux surgery in patients with Barrett's esophagus
- Those in favour of surgery argue that medical therapy and endoscopic surveillance may treat the symptoms but fail to address the problem
- The problem is the 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
- (surgery indications:
 - refuse the medical therapy
 - refractory to medical treatment
 - There is complication
- Studies have demonstrated regression of metaplasia to normal mucosa up to 57% of the time in patients who have undergone antireflux surgery
- Photodynamic therapy (PDT) is the most common ablative method used to treat BE
- Endoscopic mucosal resection (EMR) is gaining favor for the treatment of Barrett's esophagus with low-grade dysplasia.
- Esophageal resection for Barrett's esophagus is recommended only for patients in whom high-grade dysplasia is found
- Pathologic data on surgical specimens demonstrate a 40% risk for adenocarcinoma within a focus of high-grade dysplasia

Caustic Injury (the Dr didn't even mention this part!)



- the best cure for this condition is an ounce of prevention
- In **children**, ingestion of caustic materials is **accidental** and tends to be in **small quantities**
- In **teenagers and adults**, however, ingestion usually is **deliberate during suicide attempts**, and much **larger quantities** of caustic liquids are consumed
- **Alkali ingestion is more common** than acid ingestion **because of its lack of immediate symptoms**
- alkali ingestion are much more devastating and almost always lead to significant destruction of the esophagus

Table 41-3 -- Three Phases of Tissue Injury From Alkali Ingestion

PHASE	TISSUE INJURY	ONSET	DURATION	INFLAMMATORY RESPONSE
1	Acute necrosis	1-4 days	1-4 days	Coagulation of intracellular proteins Inflammation
2	Ulceration and granulation	3-5 days	3-12 days	Tissue sloughing Granulation of ulcerated tissue bed
3	Cicatrization and scarring	3 weeks	1-6 months	Adhesion formation Scarring

Esophageal Caustic injury phases

phase one

patients may complain of oral and substernal pain, hypersalivation, odynophagia and dysphagia, hematemesis, and vomiting

phase two

these symptoms may disappear only to see dysphagia reappear

phase three

fibrosis and scarring begin to narrow the esophagus

Table 41-4 -- Endoscopic Grading and Treatment of Corrosive Esophageal and Gastric Injuries

DEGREE OF BURN	ENDOSCOPIC EVALUATION	TREATMENT
First degree	Mucosal hyperemia	48-hr observation
	Edema	Acid suppression
Second degree	Limited hemorrhage	Aggressive IV resuscitation
	Exudates	IV antibiotics
	Ulceration	Acid suppression
	Pseudomembrane formation	
Third degree	Mucosal sloughing	Inhaled steroids
	Deep ulcerations	Fiberoptic intubation (if needed)
	Massive hemorrhage	
	Complete luminal obstruction	
	Charring	
	Perforation	



Symptoms and signs

- Symptoms of respiratory distress, such as hoarseness, stridor, and dyspnea, suggest upper airway edema and are usually worse with acid ingestion
- Pain in the back and chest may indicate a perforation of the mediastinal esophagus, whereas abdominal pain may indicate abdominal visceral perforation

Diagnosis

- Diagnosis is initiated with a physical exam specifically evaluating the mouth, airway, chest, and abdomen
- Careful inspection of the lips, palate, pharynx, and larynx is done
- The abdomen is examined for signs of perforation
- Early endoscopy is recommended 12 to 24 hours after ingestion to identify the grade of the burn
- Serial chest and abdominal radiographs are indicated to follow patients with questionable chest and abdominal exams

Treatment

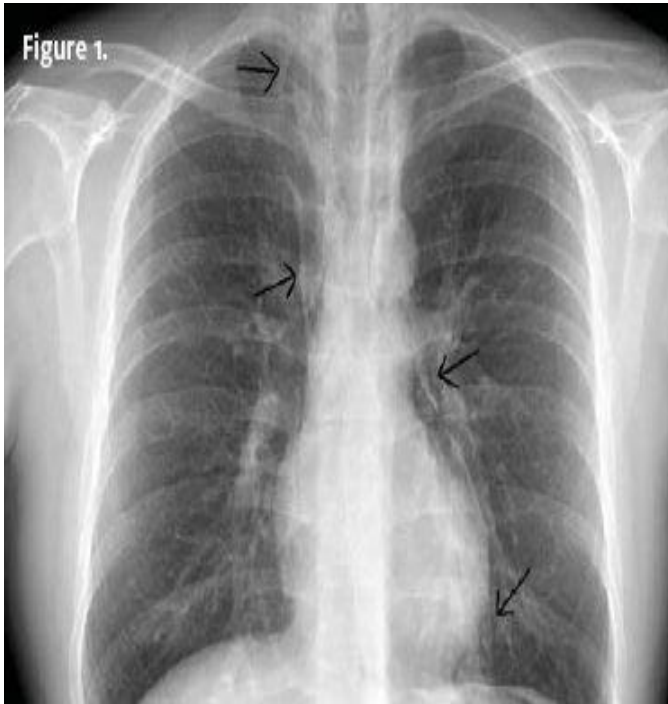
- Management of the acute phase is aimed at limiting and identifying the extent of the injury
- It begins with neutralization of the ingested substance
- **Alkalis** (including lye) are **neutralized with half-strength vinegar or citrus juice**
- **Acids** are **neutralized with milk, egg whites, or antacids**
- **Emetics and sodium bicarbonate** need to be avoided because they can **increase the chance of perforation**
- First-Degree Burn :
 - I. 48 hours of observation is indicated
 - II. Oral nutrition can be resumed when a patient can painlessly swallow saliva
 - III. A repeat endoscopy and barium esophagram are done in follow-up at intervals of 1, 2, and 8 months
- Second- and Third-Degree Burns :
 - I. Resuscitation is aggressively pursued
 - II. The patient is monitored in the intensive care unit
 - III. kept (NPO) with IV fluids. IV antibiotics and a proton pump inhibitor are started
 - IV. Fiberoptic intubation may be needed and must be available

Esophageal Perforation:

	<ul style="list-style-type: none">• Perforation of the esophagus is a surgical emergency• Early detection and surgical repair within the first 24 hours results in 80% to 90% survival• after 24 hours, survival decreases to less than 50%
Causes	<ul style="list-style-type: none">• Most esophageal perforations occur after endoscopic instrumentation for a diagnostic or therapeutic procedure,• Perforation from forceful vomiting (Boerhaave's syndrome), foreign body ingestion, or trauma accounts for 15%, 14%, and 10% of cases, respectively.
Symptoms	<ul style="list-style-type: none">• neck, substernal, or epigastric pain are consistently associated with esophageal perforation• Vomiting, hematemesis, or dysphagia also may accompany them• history of trauma, advanced esophageal cancer, violent retching as seen in Boerhaave's syndrome, swallowing of a foreign body, or recent instrumentation must raise the question of esophageal perforation.• Cervical perforations may present with neck ache and stiffness due to contamination of the prevertebral space• Thoracic perforations present with shortness of breath and retrosternal chest pain lateralizing to the side of perforation.• Abdominal perforations present with epigastric pain that radiates to the back if the perforation is posterior.• On examination , patient may present with tachypnea, tachycardia, and a low-grade fever but have no other overt signs of perforation.• With increased mediastinal and pleural contamination, patients progress toward hemodynamic instability
Diagnosis	<ul style="list-style-type: none">• On exam, subcutaneous air in the neck or chest, shallow decreased breath sounds, or a tender abdomen are all suggestive of perforation• Laboratory values of significance are an elevated white blood cell count and an elevated salivary amylase in the blood or pleural fluid.• Diagnosis of an esophageal perforation may be made radiographically (Barium Swallow)• A chest roentgenogram may demonstrate a hydropneumothorax• A contrast esophagram is done using barium for a suspected thoracic perforation and Gastrografin for an abdominal perforation.• Most perforations are found above the GEJ on the left lateral wall of the esophagus which results in a 10% false-negative rate in the contrast esophagram if the patient is not placed in the lateral decubitus position• Chest CT shows mediastinal air and fluid at the site of perforation• A surgical endoscopy needs to be performed if the esophagram is negative or if operative intervention is planned.• Mucosal injury is suggested if blood, mucosal hematoma, or a flap is seen or if the esophagus is difficult to insufflate.

Esophageal Perforation:

- Patient with GERD usually presents post-endoscopy with chest pain, dysphagia and odynophagia.
- The complication we fear the most from GERD
- Ask the patient for **chest pain, dysphagia, odynophagia (the esophagus is torn)**



Chest X-ray is very important in perforation; you will find AIR (black spaces) around esophagus:
pneumo-mediastinum
VERY DIAGNOSTIC OF PERFORATION!



ON BARIUM SWALLOW, CONTRAST WILL EXTRAVASATE (LEAK OUT)
1ST THING TO DO IS PUT PATIENT ON NPO (food extravasation in the mediastinum!)

2ND STEP: IV FLUIDS (as the patient is NPO)

3RD STEP: ANTIBIOTIC (fear of GI flora spelling in the sterile mediastinum)

Treatment:

Small perforation: conservative NPO (nothing per oral) with maintenance fluid (D5/HALF SALINE) THEN SWITCH TO TOTAL PARENTERAL NUTRITION (TPN)
if the perforation is large:, we intervene surgically!!

Esophageal Perforation

Treatment

- Patients with an esophageal perforation can progress rapidly to hemodynamic instability and shock
 - perforation is suspected, appropriate resuscitation measures with the placement of large-bore peripheral IV catheters, a urinary catheter, and a secured airway are undertaken before the patient is sent for diagnostic testing
 - IV fluids and broad-spectrum antibiotics are started immediately, and the patient is monitored in an ICU
 - The patient is kept NPO, and nutritional access needs are assessed
 - Surgery is not indicated for every patient with a perforation of the esophagus
 - management is dependent on several variables: stability of the patient, extent of contamination, degree of inflammation, underlying esophageal disease, and location of perforation
-
- The most critical variable that determines the surgical management of an esophageal perforation is the **degree of inflammation** surrounding the perforation.
 - When patients present within 24 hours of perforation, inflammation is generally minimal, and primary surgical repair is recommended
 - With time, inflammation progresses, and tissues become friable and 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

Case scenario:

patient with GERD, you request Endoscopy for him, 6 hours later, the patient complains of sudden severe dysphagia حتى لما يبلع ريقه, odynophagia and chest pain,

to make sure that it's Esophageal Perforation: we do Barium Swallow, we see

Extravasation of the Barium and Pneumo-mediastinum on CXR

Dx:

Esophageal Perforation

Leiomyoma



Definition/ Epidemiology

- Leiomyomas constitute 60% of all benign esophageal tumors
- They are found in men slightly more often than women and tend to present in the 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)
- GIST tumors are the most common mesenchymal tumors of the gastrointestinal tract and can be benign or malignant
- All leiomyomas are benign with malignant transformation being rare.

Genetics

- Nearly all GIST tumors occur from mutations of the c-KIT oncogene, which codes for the expression of c-KIT (CD117).

Symptoms and Diagnosis

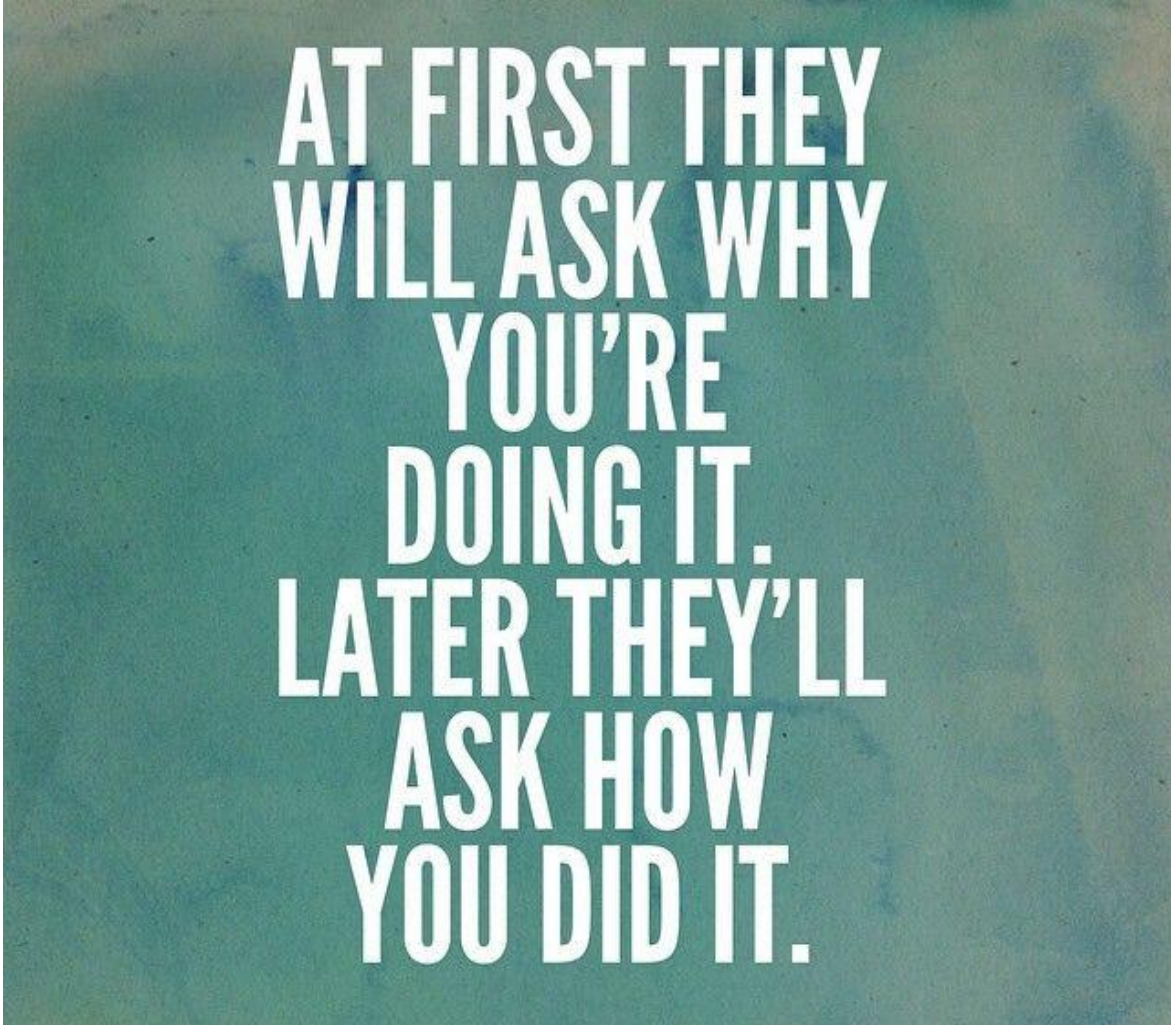
- Many leiomyomas are asymptomatic
- Dysphagia and pain are the most common symptoms and can result from even the smallest tumors
- A chest radiograph is not usually helpful to diagnose a leiomyoma, but on barium esophagram, a leiomyoma has a characteristic appearance.
- During endoscopy, extrinsic compression 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



Leiomyoma

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 most patients, surgical resection is advocated
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- 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%



AT FIRST THEY
WILL ASK WHY
YOU'RE
DOING IT.
LATER THEY'LL
ASK HOW
YOU DID IT.