Internal Medicine – Gastrointestinal and Pancreatic disease lecture for 4Th-year medical students'. University of Anbar College of Medicine by dr Yasin Hamad Majeed

Lecture 1*[2hrs]*

The digestive system consists of the gastrointestinal tract consisting of the mouth, pharynx, esophagus, stomach, small intestine, and large intestine plus accessory organs consisting of the salivary glands, exocrine pancreas, liver, and gallbladder.

DIEASES OF ESOPHAGUS

The esophagus is a Muscular tube 25 cm length extends from the cricoid cartilage to the cardiac orifice of the stomach Transfer the food from the mouth to the stomach with an upper and a lower sphincter**.**

Swallowing (Deglutition)

Swallowing is a complicated mechanism because the pharynx sub serves respiration as well as swallowing. The pharynx is converted for only a few seconds at a time into a tract for the propulsion of food. It is especially important that respiration not be compromised because of swallowing.

Swallowing can be divided into

1-The oral Stage. [voluntary stage]

When the food is ready for swallowing it is squeezed posteriorly into the pharynx by the pressure of the tongue upward and backward against the palate

2-Pharyngeal Stage. [involuntary Stage]

When the bolus of food enters the posterior mouth and pharynx it stimulates swallowing **Receptor** around the opening of the pharynx and impulses from these receptors to pass to the brain stem by trigeminal and glossopharyngeal nerves and the motor impulses from the swallowing center to the pharynx and upper esophagus are transmitted by the 5th, 9th, 10th, 12th cranial nerves, and superior cervical nerves.

to initiate a series of automatic pharyngeal muscle contractions as follows:

a. The soft palate is pulled upward to close the posterior nares to prevent the passage of food into the nose. to form a sagittal slit through which the food passes into the posterior pharynx. This slit performs a selective action allowing food that has been masticated sufficiently to pass with ease and any large object is usually prevented to pass into the esophagus.

c. The larynx is pulled upward by the neck muscle contraction. These actions cause the epiglottis to swing over the opening of the larynx. The upward movement of the larynx also pulls up and enlarges the opening to the esophagus. At the same time, the **U**pper **E**sophageal **S**phincter [UES] relaxes thus allowing food to enter into the upper esophagus.

3-Esophageal Stage[involuntary stage] .

Normally the esophagus has two types of peristaltic movements.

Primary peristalsis is a continuation of the peristaltic wave that begins in the pharynx and moves into the esophagus. primary peristalsis moves all food that entered the esophagus to the stomach.

Secondary peristalsis. This peristaltic waves result from the distention of the esophagus itself by the retained food. The secondary peristaltic waves are initiated by the esophageal enteric nervous system.

DYSPHAGIA

Means difficulty swallowing .Clinically it includes the inability to initiate swallowing and or the sensation that the swallowed solids or liquids stick in the esophagus.

***Odynophagia**. pain or burning sensation during swallowing.

Causes of odynophagia include *GERD *infection* chemical esophagitis due to drugs such as bisphosphonates or slow-release potassium.

***Globus sensation.** Constant or intermittent Sensation of a lump, fullness or pressure in the throat that is unrelated to meal or swallowing .Globus sensation disappear or transiently lessen during swallowing with the absence of dysphagia, odynophagia, pathological GERD, achalasia and other motility disorders.

Classification of dysphagia

1-Oropharyngeal or Transfer dysphagia. [difficulty in initiating the swallow]. Difficulty in transferring food from the mouth to the upper esophagus.

The patient presented with

*Choking or aspiration of food during swallowing .

*Abnormality of speech like dysarthria or nasal speech may be present.

Causes include

Neuromuscular disorders

1-Central nervous system conditions.

Cerebrovascular accident ,Bulbar or Pseudo bulbar palsy ,brain stem tumors, paraneuplastic disorders

2-Peripheral nervous system conditions.

Poliomyelitis, demyelinating disease, Guillain-Barre syndrome.

3-Disorders of the myoneural junction.

Myasthenia graves, Eaton Lambert syndrome.

4-Muscular disorders .

dermatomyositis, Muscular dystrophy, collagen vascular disease, amyloidosis.

5-Others- Zenker's diverticulum**,** Aphthous ulcer**,** Candidiasis.

2-Oesophageal dysphagia .Means difficulty in the passage of food through the esophagus to the stomach. Any disorder, structural, neuromuscular involving the body of the esophagus, LES, or the gastroesophageal junction may result in dysphagia.

Causes of esophageal dysphagia include

1-Structural

initially, dysphagia is noted with solid food when the lumen narrows with enlarging lesion passage of liquids also becomes impaired.

A-Malignant Tumors –squamous carcinoma, adenocarcinoma of the esophagus.

B-Strictures

*Peptic strictures most esophageal strictures are in the distal or mid esophagus, peptic strictures usually benign but those associated with Barrett's may be malignant.

*Post corrosive esophageal strictures.

*****Drugs .may lodge in the esophagus and cause local inflammation, ulceration, and stricture.

*****Foreign bodies**.[**coins or button batteries] may be swallowed and cause obstruction or injury of the esophagus.

C-Webs and Ring

Web-like constrictions of the esophagus are usually congenital or inflammatory. can cause intermittent dysphagia to solids.

1-Plummer-Vinson syndrome**.**

Is the combination of dysphagia caused by post cricoid web [thin circumferential mucosal shelves that protrude into the esophageal lumen] and iron-deficiency anemia usually in middle-aged women . this post cricoid web can be complicated by squamous cell carcinoma of the esophagus.

2-Schatzki ring**.**

Is a thin web-like constriction located at the squamo-columnar junction [gastro-esophageal junction] causes episodic dysphagia for solids when the lumen diameter is <13 mm. Symptomatic webs and mucosal lower esophageal rings are usually treated by dilatation.

D-Eosinophilic Esophagitis –an inflammatory condition of the esophagus characterized by the presence of multiple concentric rings throughout the esophagus.

E-Extrinsic Compression include

Mediastinal tumors, vascular lesion, cervical osteoarthritis, esophageal diverticula.

2- Gastro Esophageal Reflux Disease[GERD].

3-Neuromuscular or motility disorders-results in dysphagia for both solids and liquids without evidence of structural abnormalities.in these disorders peristalsis is either absent, week, too strong and sustained or uncoordinated.

A-Primary motor disorders include- Achalasia and diffuse esophageal spasm,

B-secondary motor disorders,

Evaluation of the patient with dysphagia

History

Type of food causing dysphagia.*

*Dysphagia for solids indicates mechanical obstruction.

*Dysphagia for solids and liquids from the onset indicate esophageal dysmotility like [Achalasia and diffuse esophageal spasm]

*Patients with scleroderma have dysphagia to solids that are unrelated to posture and liquids while recumbent.

The duration and course of dysphagia.

*Transient dysphagia may be due to an inflammatory process.

*Progressive dysphagia indicates an organic cause ex [carcinoma or peptic stricture].

*Episodic Dysphagia to solids lasting several years indicates a benign disease like Schatzki ring[a lower esophageal ring].

*Progressive short history < 3m indicates malignancy.

The site of dysphagia described by the patient correlates poorly with the true site of obstruction.

Associated Symptoms provide important diagnostic clues.

*Nasal regurgitation and tracheobronchial aspiration with swallowing indicate pharyngeal paralysis or tracheo-esophageal fistula.

*Tracheo-bronchial aspiration unrelated to swallowing may be secondary to Achalasia, Zenker's diverticulum, or gastroesophageal reflux.

*Severe weight loss is highly suggestive of carcinoma.

*Hoarseness following dysphagia may suggest the involvement of the recurrent laryngeal nerve by extension of esophageal carcinoma. Sometimes hoarseness may be due to laryngitis secondary to gastroesophageal reflux.

* Unilateral wheezing with dysphagia indicates a meditational mass involving the esophagus and a large bronchus.

*Chest pain with dysphagia occurs in esophageal dysmotility.

*A prolonged history of heartburn and reflux preceding dysphagia indicates peptic stricture.

*A history of prolonged nasogastric intubations, ingestion of caustic agents or pills without water, previous radiation therapy, or associated mucocutaneous diseases may provide the cause of esophageal stricture.

* Presence of Odynophagia indicate [candida, herpes, pill-induced Esophagitis or GERD]

Physical Examination

Physical examination is important in dysphagia.

*inspection of the mouth and pharynx looking for lesions that may interfere with the passage of food because of pain or obstruction

*The neck should be examined for goiter, spinal abnormality, and supraclavicular lymph nodes for carcinoma of the stomach.

*Changes in the skin and extremities may suggest a diagnosis of scleroderma and other collagen-vascular diseases or mucocutaneous diseases.

***Signs of bulbar or pseudo bulbar palsy including dysarthria, dysphonia, ptosis, tongue atrophy, and hyperactive jaw jerk

*Pulmonary complications of acute aspiration pneumonia or chronic aspiration may be present.

Investigations.

* Endoscopy is the investigation of choice because it allows biopsy and dilatation of suspicious strictures. If no abnormality is found, then

*barium swallow with video-fluoroscopic swallowing assessment is indicated to detect motility disorders.

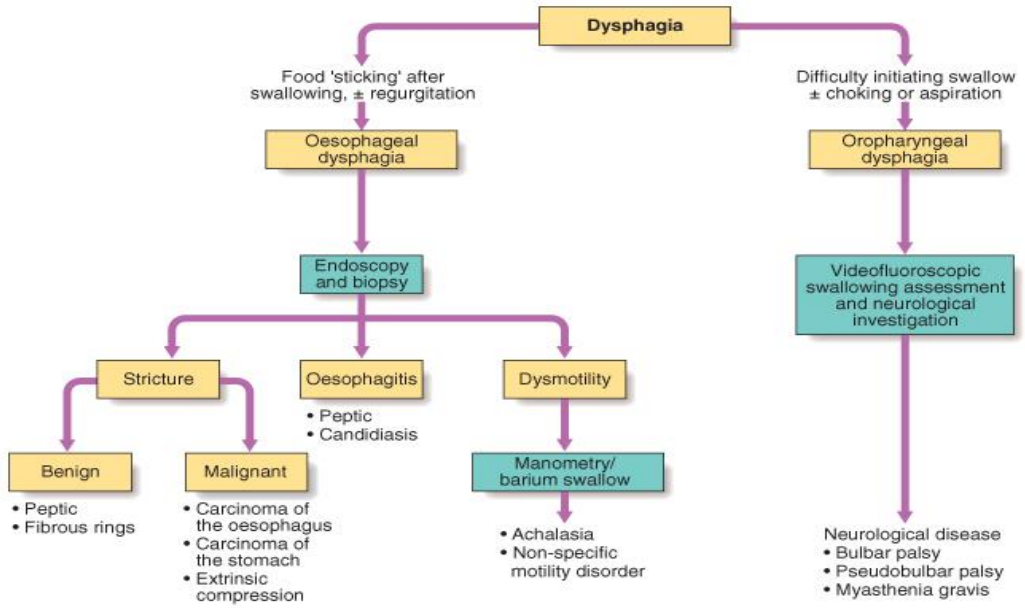
*Esophageal manometry.

Chest X-ray Looking for pulmonary consolidation or hilar tumor, absent of gastric bubble on CXR indicate achalasia.

*Blood test. complete blood count

***Pulmonary function test in a patient with scleroderma**.

Diagnostic approach to dysphagia



Gastro Esophageal Reflux Disease[GERD]

In healthy patients, the Lower Esophageal Sphincter creates a valve that prevents gastric content from traveling back into the esophagus.
Gastroesophageal reflux occurs when gastric content escape into the esophagus this may or may not produce symptoms.
GERD is any symptomatic condition or histopathological change resulting from exposure of Esophageal mucosa to gastric content for a prolonged period. GERD is one of the most prevalent gastrointestinal disorders affecting between 10- 30% of the general population.

Pathophysiology

Occasional episodes of GERD are common in health. Reflux is followed by Esophageal peristaltic waves which clear the esophagus, alkaline saliva neutralizes residual acid.
Antireflux barriers include 1-Esophageal peristalsis. 2-Intrinsic tone of the LES.
3-The crural diaphragm. *The anatomic location of the gastro esophageal junction and tissue resistance.

Factors that can contribute to GERD:

1-Abnormalities of the Lower Esophageal sphincter [LES].
In health, the LES is tonically contracted to relax only during swallowing some patients have reduced LES tone permitting reflux when the intra-abdominal pressure rises. In others basal sphincter tone is normal but reflux occurs in response to frequent episodes of inappropriate sphincter relaxation.
Causes of reduced LES tone include *Scleroderma.* Myopathy associated with chronic intestinal pseudo-obstruction, * Pregnancy* smoking* Anticholinergic drugs* Smooth-muscle relaxants [B-adrenergic agents, aminophylline, nitrates, calcium channel blockers*Surgical destruction of the LES.
2-Delayed Esophageal clearance

3-Hiatus hernia -Herniation of part of the stomach into the thoracic cavity through the

esophageal hiatus in the diaphragm –[hiatus hernia disrupts the integrity of the sphincter mechanism and esophageal clearance leading to increased esophageal acid exposure.

4-Gastric contents

*Gastric volume is increased (after meals, in pyloric obstruction, in gastric stasis, during acid hyper secretion states [Zollinger-Ellison syndrome], Hypercalcemia, which can increase gastrin production leading to increased acidity*Gastric content near the gastroesophageal junction (in recumbency, bending down and hiatus hernia).
5-defective gastric emptying.
6-Increased intra-abdominal pressure[(obesity, pregnancy, Ascites, and tight clothes).
7-Dietary and environmental factors *Smoking *high fat* chocolate *alcohol * coffee.

Clinical Features

*Heartburn. is produced by the contact of refluxed material with the inflamed or sensitized esophageal mucosa
*Angina-like or atypical chest pain occurs in some patients probably due to reflux induced esophageal spasm. This pain or burning worse on bending, stooping or lying down rarely radiates to the arms Worse with hot drinks or alcohol, Relieved by antacids
*Regurgitation of sour material in the mouth are the characteristic symptoms of GERD often provoked by bending, straining or lying down
*Excess salivation [water brash] from the salivary gland in response to GER.
*Night choking due to refluxed fluid that irritates the Larynx.
*Odynophagia occurs in severe esophagitis.
*Dysphagia may be due to a narrowing or stricture. Sometime due to inflammation and edema that may resolve with medical therapy.
Rapidly progressive dysphagia and weight loss in a patient with a prolonged history of GERD may indicate the development of adenocarcinoma in Barrett's esophagus.
*Bleeding occurs due to mucosal erosions or Barrett's ulcer.

Extra-esophageal symptoms of GERD

*Severe reflux may reach the pharynx and mouth and result in an extra esophageal manifestation of GERD which includes. Dental erosion, Laryngitis, and Recurrent pulmonary aspiration causing [aspiration pneumonia, pulmonary fibrosis or chronic asthma].

Differential diagnosis of GERD

Eosinophilic esophagitis-
Medication-induced esophagitis
Esophageal motor disorders

Investigations include

Endoscopy
Esophageal biopsy
Barium swallow
Ambulatory impedance and pH monitoring.
Esophageal manometry
***Endoscopy** is the gold slandered for diagnosing GERD and its complications.
The Los Angeles Classification of Esophagitis
LA grade A: one or several erosions limited to the mucosal fold(s) and no larger than 5 mm in extent.
LA grade B: one or several erosions limited to the mucosal fold(s) and larger than 5 mm in extent
LA grade C: erosion(s) extending over mucosal folds, but are not circumferential.
LA grade D: confluent erosions extending over more than three-quarters of the circumference.
***Barium study**
Low sensitivity but increased in the presence of erosion, ulceration, and stricture.
***Ambulatory 24 h esophageal PH monitoring** is indicated when despite endoscopy the diagnosis is unclear. This involves tethering a slim catheter with a terminal pH-sensitive probe above the gastro-Esophageal junction. The luminal pH is recorded while the patient undergoes normal activities and episodes of pain are noted and related to PH.
PH of less than 4 for more than 6-7% of the study time is diagnostic of reflux disease.
***Esophageal manometry** commonly used to evaluate patients with GERD who are candidates for surgery to excluded specific dysmotility syndromes.
***Esophageal impedance** [nonacid reflux].
***Capsule endoscopy**

Treatment includes.

1-General measures include *Weight reduction*Elevation of the bed head in those who experience nocturnal symptoms. *Elimination of factors that increases abdominal pressure.*Stop smoking and avoid [fatty foods, coffee, chocolate, alcohol, orange juice, ingesting large quantities of fluids with meals and medications such as (anticholinergic drugs, calcium channel blockers, NSAID, nitrates, and other smooth-muscle relaxants)]. * antacid medications.

If general measures alone are insufficient drug treatment is indicated which include.

2-Medications

1-Antisecretery drugs

***Proton Pump Inhibitors**[PPI]. include omeprazole, esomeprazole, pantoprazole, Lansoprazole, and rabeprazole) are the most effective in reducing gastric acid secretion. PPI should be given 30 minutes before breakfast or dinner for 2w -2m. Chronic use of PPIs may affect the absorption of calcium, vitamin B12, magnesium and iron.

***H2 receptor blockers** (such as ranitidine, famotidine and cimetidine) can reduce the gastric secretion of acid. are more effective in controlling nocturnal than meal stimulated Acid secretion.

2-Transient Lower Esophageal Sphincter Relaxation Inhibitors

***Baclofen** is a GABAB receptor agonist [dose 10mg given four times daily].

3-Antireflux Surgery.

Indicated for patient who*Fail to respond to medical therapy.*Are unwilling to take long- term PPI.*And those with regurgitation.

Laparoscopic fundoplication is the surgery of choice [In which the gastric fundus is wrapped around the 0esophagus].

Patients' candidates for fundoplication are those in whom motility studies show persistently inadequate LES pressure but normal peristaltic contractions in the esophageal body.

Complications of GERD include

1-Hemorrhage, Ulcers, and Perforation

2-Esophagitis develops when mucosal defenses are unable to counteract the damage done by acid, pepsin and bile.

3-Barrett's Esophagus[BE]. in which the normal squamous epithelium of the distal esophagus is replaced by columnar epithelium in patients with long-standing GERD. Metaplastic columnar epithelium develops during the healing of erosive esophagitis with continued acid reflux because columnar epithelium is more resistant to acid-pepsin damage than squamous epithelium. BE is a premalignant condition affected patients who have increased risk of esophageal adenocarcinoma. Diagnosis requires multiple biopsies to maximize the chance of detecting intestinal metaplasia and or dysplasia.

BE classifications

a-Long segment BE [more than 3 cm of columnar lined esophagus].

b-Short segment [less than 3 cm of columnar lined esophagus.

surveillance is advisable for patients with BE to detect early changes of dysplasia. Rapidly progressive dysphagia and weight loss may indicate the development of adenocarcinoma in Barrett's esophagus.

Treatment of Barrett's include

1-Endoscopica therapy.

-Endoscopic mucosal resection EMR

-Photodynamic therapy

-Radio-phrequency ablation

-Cryotherapy

2-Surgery –Esophagectomy

4-Anaemia

Long-standing Esophagitis may cause chronic blood loss which leads to Iron deficiency anemia.

5-Benign Esophageal stricture. Causing dysphagia which is worse for solid than for liquids

Esophagitis

1-Infectious Esophagitis

Causes include

***Viral Esophagitis** *Herpes simplex virus [HSV]* Varicella-zoster virus[VZV].* Cytomegalovirus [CMV] infections. * HIV.

HSV esophagitis –usually self-limited and resolve in 1 w to 10 days if persistence or recurrence of the disease may be a sign of AIDS

Presentation- fever, odynophagia and substernal chest pain that increase with eating

Diagnosis

Endoscopy revealed, vesicles of a different size or punched out ulcers or diffuse ulcerations, biopsy, and brushings of the affected areas should be obtained

Barium swallow- double-contrast may show ulcers

Treatment-

Supportive measures. sever odynophagia needs iv fluid, a local anesthetic agent like viscous lidocaine .

Acyclovir, iv or oral depend on disease severity

***Bacterial esophagitis.** caused by Lactobacillus and b-hemolytic streptococci occur in immunocompromised patients. In patients with AIDS, infection with Cryptosporidium or Pneumocystis carini. Mycobacterium tuberculosis infection may cause deep ulcerations of the distal esophagus.

***Fungal Esophagitis.** Candida albicans is the most common, the whole esophagus may be affected

Predisposing factor

Immunosuppressive conditions like HIV, neutropenia, malignancy, organ transplant,chemotherapy,,DM,malnutrition,,alcoholism, renal failure

Diagnosis.

Clinical presentation

Dysphagia, odynophagia, oral thrush

Diagnostic studies

*Endoscopy

Small raised white plaque, ulceration. Brushing the lesion followed by cytology or biopsy

*Barium studies may help in the diagnosis.

Treatment

*Underlying predisposing factors should be identified

*Oral fluconazole or a topical antifungal

2-Pill-induced Esophagitis

causes include

*Antibiotics [doxycycline, tetracycline, oxytetracycline, minocycline, penicillin, and clindamycin].

*Nonsteroidal anti-inflammatory agents such as aspirin, indomethacin, and ibuprofen

*Potassium chloride, ferrous sulfate or succinate, quinidine, theophylline, ascorbic acid

Pathogenesis

The mechanism of injury may be due to prolonged contact of the caustic content of medications with esophageal mucosa predisposed by the presence of anatomic or motility disorders or when the pills ingested without enough water or assuming a recumbent position or sleeping immediately after pill ingestion.

Presentation

Acute onset of chest pain commonly accompanied by severe odynophagia

Endoscopic finding range from small kissing ulcers to severe diffuse esophagitis

Complications

Perforation, fistula, hemorrhage, and stricture formation.

Treatment

Topical viscous lidocain solution, PPI

Pill esophagitis can be prevented by taking pills in an upright position with suitable amounts of fluids.

3-Radiation esophagitis is caused by radiation treatment for thoracic cancers.
Dysphagia and odynophagia may last several weeks to several months after therapy.
4-Corrosive Esophagitis .caused by the ingestion of caustic agents, such as strong alkali [lye-sodium or potassium, bleaches[Clorox], ammonia or acid[hydrochloric acid, sulfuric, battery acid, formic acid, disinfectants, Severity of tissue injury after caustic ingestion depends on the nature, concentration, quantity and the duration of tissue contact.
Alkalis produce injury by liquefaction necrosis which allows deep penetration into tissues resulting in full-thickness tissue burns. Caustic acids produce coagulation necrosis forming a firm productive eschar that may limit deep tissue injury except in cases where there are anatomic or motility disorders.
Severe corrosive injury may lead to esophageal perforation, bleeding. Respiratory distress is secondary to soft tissue swelling of the epiglottis, larynx, vocal cords, or lung injury due to aspiration.
Presentation
-oropharyngeal pain, dysphagia, odynophagia, hoarseness, stridor, chest or back pain suggest esophageal perforation or mediastinitis, and epigastric pain may suggest gastric perforation and peritonitis
Management
*IV line
*Nothing by mouth
*Pain control
*Antibiotic if perforation or mediastinitis is present.
*Upright x-ray films of the chest and abdomen looking for signs of aspiration or perforation.
*if perforation suspected soluble contrast may be used to localize perforation.
*Surgery.
Vomiting should be avoided and endoscopy should not be carried immediately because of the risk of perforation. Healing is usually associated with stricture formation. Caustic strictures are usually long and rigid and generally require periodic dilatation.

Esophageal Motility Disorders
Primary motor disorders'
Achalasia
Pathophysiology a disorder of esophageal smooth muscle function with three diagnostic features
1-Absence of primary and secondary peristalsis in the smooth muscle of the esophagus.
2-The resting LES pressure is usually high.
3-Lower Esophageal sphincter does not relax completely with swallow.
Achalasia may be Primary[idiopathic achalasia] or secondary achalasia caused by [gastric carcinoma infiltrate the lower esophagus, lymphoma, Chagas disease, eosinophlic esophagitis, and neurodegenerative disorders].
*loss of ganglion cells within the myenteric [auerbachs] plexus, degeneration of the vagus nerve, and degeneration of the dorsal motor nucleus of the vagus.
*postganglionic denervation of the esophageal smooth muscle affects inhibitory ganglion [VIP and nitric oxide] first then excitatory ganglion neurons[cholinergic] or both.
Clinical features.
Achalasia can occur at any age and both sexes.
*Progressive dysphagia and weight loss over months to years. Dysphagia occurs early with both liquid and solid and is initially intermittent it is worse for solid and is eased by drinking liquids, standing and moving around after eating .dysphagia is worsened by emotional stress and hurried to eat.
*Severe chest pain due to Esophageal spasm [vigorous achalasia-overlap between diffuse esophageal spasm and achalasia]
*Regurgitation of non-bilios no- acid
*Pulmonary aspiration occur because of retention of large volumes of saliva and ingested food in the esophagus.
* halitosis

Achalasia predisposes to squamous carcinoma of the esophagus.

Differential diagnosis

*diffuse esophageal spasm,chags disease,

Pseudo achalasia may be

Malignant [achalasia like features due to esophageal infiltration by malignant occurs usually above 50 y abrupt onset of symptom, with significant wt loss],pseudoachalasi may occur as Para neoplastic syndrome without direct tumor involvement of the oesophagogastric junction.

Nonmalignant- esophageal infiltration by amyloidosis, eosinophlic gastroenteritis, and Sarcoidosis.

Diagnosis

1-Chest x-ray shows*Absence of the gastric air bubble.* Retained food in the esophagus causing an air-fluid level in the mediastinum in the upright position.

2-Barium swallows shows

*Esophageal dilation and in advanced cases the esophagus may become sigmoid like appearance. The terminal part of the esophagus shows a persistent beak like narrowing representing the non-relaxing LES.

3-Fluoroscopy shows peristalsis is lost in the lower two-thirds of the esophagus.

4-Manometry shows high-pressure non- relaxing LES with poor contractility of the esophageal body.

5-Endoscopy, **CT, MRI, EUS** help exclude the secondary causes of achalasia, particularly gastric carcinoma.

Treatment.

1-Medical

*Reducing LES pressure by nitrates and calcium channel blockers so that gravity promotes esophageal emptying .side effects of medical therapy include [headache and postural hypotension].

2-Endoscopical treatment for achalasia include

1-Disruption of the LES by Forceful pneumatic dilatation [Rigiflex] using a 30-35or 40 mm diameter.Complications are perforation and bleeding.

2-intrasphincteric injection of botulin toxin_which acts by blocking acetylcholine release from nerves in the sphincter.

3-per oral endoscopic myotomy[POEM]

3-Surgical

*Heller' extra-mucosal myotomy of the LES. In which the circular muscle layer is incised. Laparoscopic myotomy is the procedure of choice.

Both balloon dilatation and myotomy may be complicated by gastroesophageal reflux and this can lead to severe esophagitis because Esophageal clearance is poor in these patients. For these reasons, hellers myotomy is accompanied by a partial fundoplication [anti- reflux operation]. Acid suppressing drugs therapy using a PPI following endoscopical or surgical intervention should be used to prevent esophagitis.

***Esophagectomy** indicated in patients after failure of previous less invasive treatment.