ESOPHAGEAL DISORDERS:
- ESOPHAGEAL MOTOR DISORDERS.
- GERD
- ESOPHAGEAL TUMORS.

ESOPHAGEAL MOTOR DISORDERS

Present with chest pain, dysphagia, or both.
Dysphagia is for liquids as well as solids.
Chest pain may mimic cardiac disease (non-cardiac chest pain) & must be excluded.

Include:

1. Oropharyngeal dysphagia.
2. Esophageal dysphagia.
3. Primary esophageal motor disorders.

1. Oropharyngeal dysphagia:
Due to NM disorders of the oropharynx & the skeletal muscle of the esophagus, as stroke, Parkinson’s disease, ALS, MS, myasthenia gravis, polymyositis, myotonic dystrophy.
Characterized by difficulty in bolus transfer from mouth to esophagus, with nasal regurgitation or coughing (from aspiration) with swallowing. Commonly have signs / symptoms of underlying disorder.

Diagnosis: modified Ba swallow with videofluoroscopy.

Treatment: Nutritional support via diet, swallow maneuvers, feeding tubes, or PEG is appropriate + neuromuscular disorder

2. Esophageal dysphagia:
Due to disease of the smooth muscles of the esophagus.
Dysphagia is not accompanied by failure of bolus transfer from mouth to esophagus, nasal regurgitation, or coughing with swallowing.
Dysphagia characteristically occurs with liquids as well as solids, indicating a lack of discrimination for bolus size.

3. Primary esophageal motor disorders:
   a. Achalasia.
   b. Diffuse esophageal spasm.
   c. Nutcracker esophagus, hypertensive LES & ineffective peristalsis are manometric abnormalities may be symptomatic or not.

4. Secondary disorder: scleroderma is the most common.
Manometry is the diagnostic procedure of choice because it yields both quantitative & qualitative information about peristaltic & sphincter function.
a. ACHALASIA: “failure to relax"

The most common primary esophageal motor disorder.
Prevalence 10/100,000, etiology is unknown
Can occur at any age but usually 30-60 years.

Histopathology: degeneration of Auerbach’s plexus, but changes also occur in the vagus nerve & swallowing center.

Increase in pressure & incomplete relaxation of the LES with swallowing & complete aperistalsis in the esophagus body, all produces an obstruction at the GEJ that leads to esophageal retention. Dilation of esophagus body occurs due to the increase in intraluminal pressure & the presence of weak, aperistaltic contractions.

Symptoms are chronic. Dysphagia for liquids & solids is the primary complaint. Regurgitation is common, nocturnal cough suggests aspiration on reclining. Atypical chest pain & heartburn occur in 1/3 due to increased intraesophageal pressure & stasis-induced mucosal inflammation, respectively. Weight loss is highly variable & should raise concerns about adenocarcinoma of the esophagus or gastric cardia. Chagas’ disease, due to Trypanosoma cruzi can mimic achalasia by producing a megaesophagus.

Diagnosis:
CXR show a widened mediastinum, air-fluid level & absent gastric air bubble. Barium swallow shows a dilated esophagus, air-fluid level, delayed esophageal emptying & a smooth, tapered “bird’s beak” deformity at the LES. Manometry show characteristic incomplete relaxation of the LES with swallows & complete aperistalsis in the body.
LES pressure may or may not be elevated.

- Neither radiographic nor manometric criteria can effectively exclude achalasia secondary to cancer, so endoscopy must be performed prior to treatment.

Treatment: A muscle relaxant, as nifedipine 10 mg sublingually before meals, may be helpful. More effective treatment; either endoscopic inj of botulinum toxin into the LES (20 units per quadrant), pneumatic dilation, or surgical Heller myotomy. The benefits of botulinum toxin are relatively short lived (3-6 months) compared with pneumatic dilation, so repeated injections are necessary for the life of the patient. Pneumatic dilation under conscious sedation inflates a balloon placed across the LES to rupture its musculature; repeated treatment may be necessary for maximum benefit. A surgical Heller myotomy, direct incision of the LES, is usually done laparoscopically. Success rates with pneumatic dilation or Heller myotomy are comparable, with excellent relief of symptoms for 5 to 10 years in 85%. The major risk of pneumatic dilation is esophageal perforation (3%) & the major side effect of Heller myotomy is reflux esophagitis (925%), antireflux procedure with the Heller myotomy reduce this risk.

- Achalasia can predispose to SC cancer esophagus.
b. DIFFUSE ESOPHAGEAL SPASM:

Uncommon motor disorder presents clinically with chest pain, dysphagia, or both. It remains one of the most commonly sought (but uncommonly found) in patients with noncardiac chest pain. Symptoms/signs are intermittent, so diagnosis may be difficult. Like achalasia, associated with degeneration of the nerves in Auerbach’s plexus & in rarely evolve to achalasia.

**Diagnosis:**

**Bar swallow:** prominent, spontaneous, nonpropulsive, tertiary contractions give rise to a “corkscrew” esophagus, but this not pathognomonic as it also observed in asymptomatic elderly; *presbyesophagus.*

The manometric diagnosis requires exclusion of similar manometric abnormalities from DM, amyloid, scleroderma, idiopathic pseudo-obstruction & reflux esophagitis.

**Therapy** is principally supportive & empirical.

**Reassurance,** a trial of smooth muscle relaxants (e.g., isosorbide 10 mg, nifedipine 10 mg, or dicyclomine 20 mg before meals) or a trial of an antidepressant

In some instances, relaxation exercises, biofeedback & psychological counseling are helpful adjuncts to drug therapy.

c. NUTCRACKER ESOPHAGUS.

A relatively common manometric pattern found in patients with noncardiac chest pain. Consists of normal peristalsis but with contractions of very high amplitude (average > 180 mm Hg). Reduction in contraction amplitude by CCBs has no consistent effect on chest pain & chest pain can be relieved by medications (e.g., trazodone) having no effect on amplitude.

Patients with chest pain & nutcracker esophagus commonly exhibit symptoms/signs of depression or anxiety.

**Treatment:** TADs or anxiolytics may relieve the chest pain.

^SCLERODERMA:

The esophagus is the GI organ most often affected in scleroderma, resulting in a characteristic manometric pattern of low LES pressure & weak aperistaltic contractions in the smooth muscle part of the esophageal body.

UES & skeletal muscle–lined upper eso contractions are normal. Results in dysphagia, regurgitation, heartburn.

When dysphagia for solids is prominent, it likely reflects a peptic stricture or adenocarcinoma in Barrett’s esophagus.

Patients should be treated prophylactically for GERD.
Gastric functional motility disorders:

- Non-ulcer dyspepsia (NUD).
- Gastroparesis.
- Functional vomiting.

**NON-ULCER DYSEPSIA:**
Chronic dyspepsia (pain or upper abdominal discomfort) in the absence of organic disease. Other commonly reported symptoms include early satiety, fullness, bloating and nausea. 'Ulcer-like' & 'dysmotility-type' subgroups are reported, but there is great overlap between these & also with irritable bowel syndrome.

*Aetiology:* probably covers a spectrum of mucosal, motility & psychiatric disorders.

**Clinical features:**
Usually young (< 40 years), women are affected twice.

Abdominal pain is associated with a variable combination of other 'dyspeptic' symptoms, the most common being nausea & bloating after meals.

Morning symptoms are characteristic & pain or nausea may occur on waking.

Direct enquiry may elicit symptoms suggestive of IBS.

Peptic ulcer disease must be considered, whilst in older subjects intra-abdominal malignancy is a prime concern.

There are no diagnostic signs, apart perhaps from inappropriate tenderness on abdominal palpation.

Symptoms disproportionate to clinical well-being & no weight loss.

Patients often appear anxious.

A drug history should be taken and the possibility of a depressive illness should be considered.

Pregnancy should be ruled out in young women before radiological studies are undertaken.

Alcohol misuse should be suspected when early morning nausea & retching are prominent.

*Investigations:*
The history will often suggest the diagnosis but in older subjects an endoscopy is necessary to exclude mucosal disease.

While an ultrasound scan may detect gallstones, these are rarely responsible for dyspeptic symptoms.
**Management:**

The most important elements are explanation/reassurance. Possible psychological factors should be explored & the concept of psychological influences on gut function should be explained.

Idiosyncratic & restrictive diets are of little benefit, but fat restriction may help.

**Drug treatment** is not especially successful but merits trial.

Antacids are sometimes helpful.

Prokinetic drugs such as metoclopramide (10 mg 8-hourly) or domperidone (10-20 mg 8-hourly) may be given before meals if nausea, vomiting or bloating is prominent.

Metoclopramide may induce extrapyramidal side-effects, including tardive dyskinesia in young subjects.

H2RBs may be tried if night pain or heartburn is troublesome.

Low-dose amitriptyline is sometimes of value.

The role of *H. pylori* eradication remains controversial, although 10% may benefit & a minority (5-15%) avoid ulcer development in the next 3 years.

Eradication also removes a major risk factor for gastric cancer but at the cost of a small risk of side-effects & worsening symptoms of underlying GERD.

Symptoms which can be associated with an identifiable cause of stress resolve with appropriate counselling.

Some have chronic psychological disorders causing persistent or recurrent symptoms & need behavioural or psychotherapy

**GASTROPARESIS:**

Defective gastric emptying without mechanical obstruction

**Causes:**

- Primary: inherited or acquired disorders of the gastric pacemaker
- Secondary: disorders of autonomic nerves (sp diabetic neuropathy) or gastroduodenal musculature (e.g. systemic sclerosis / amyloidosis), postoperative & idiopathic.

Early satiety & recurrent vomiting are the major symptoms; abdominal fullness & a succussion splash may be present on exam.

**Diagnosis:** documentation of delayed stomach emptying by radioactive labeled test meal & excluding mechanical obstruction.

**Treatment:** metoclopramide / domperidone / IV erythromycin.

Treating underlying causes as DM.

In severe cases long-term jejunostomy feeding or total parenteral nutrition is required.

Surgical insertion of a gastric pacing device has been successful in some cases but remains experimental.