GASTROINTESTINAL DISEASES

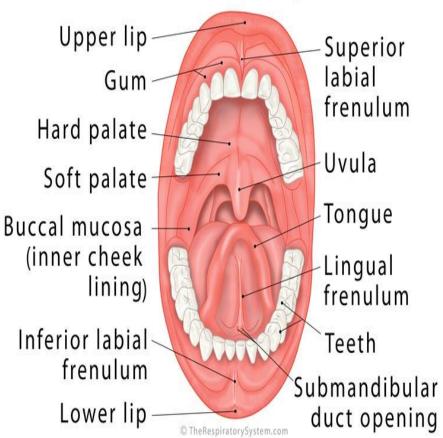
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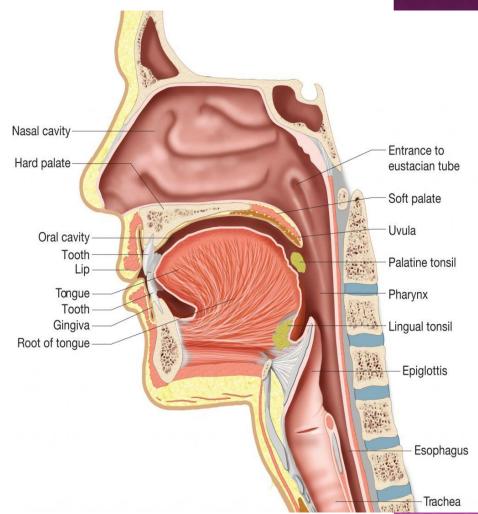
THE MOUTH & SALIVARY GLANDS.

ESOPHAGUS.

ANATOMICAL CONSIDERATIONS

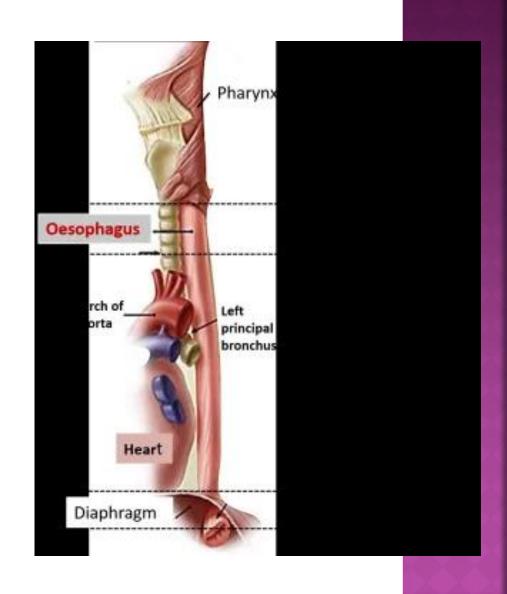
Oral Cavity





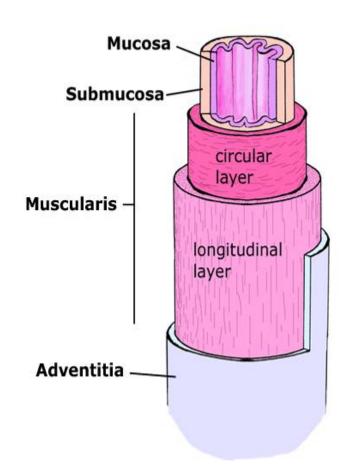
ESOPHAGUS:

- The esophagus is a muscular tube that convey food from the pharynx to the stomach
- 10 inch long
- Divided into 3 parts:
- Cervical (4 cm)
- Thoracic (20 cm)
- Abdominal (1-2cm)



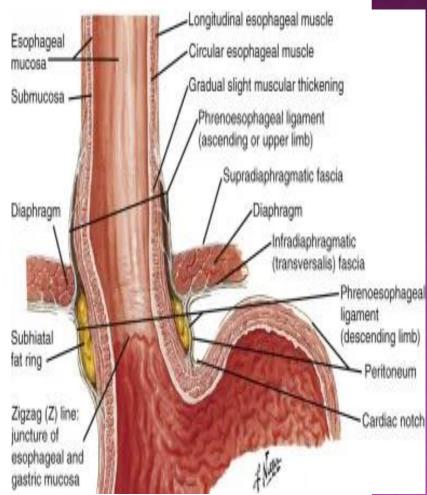
LAYERS OF THE ESOPHAGEAL WALL

- Histologically,4 concentric layers
- Mucosal layer
- 2. Sub mucosal layer
- 3. Muscular layer
- 4. Adventitial layer

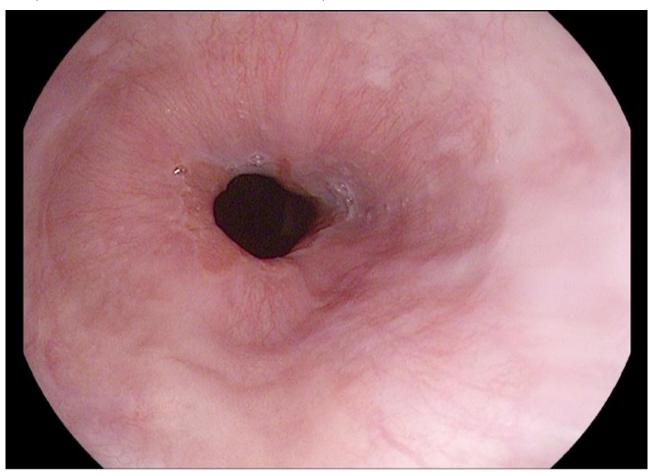


GASTROESOPHAGEAL JUNCTION. (GE) JUNCTION.

- Mucosa forms the innermost layer and is formed by a nonkeratinizing stratified squamous epithelium that is continuous with that of the pharynx.
- Mucosal epithelium changes from squamous cell epithelium to columnar cell epithelium at the gastroesophageal junction.
- This junction has been termed the <u>"Z line</u>" or <u>squamocolumnar junction</u>



(GE) JUNCTION ; (ENDOSCOPY)



DISEASES OF THE MOUTH & SALIVARY GLANDS

- Oral cavity ; mucosa
- Aphthous ulceration
- Superficial ,painful ,recurrent ,30% of population
- Unknown cause
- Management : symtomatic ttt, local ttt, may need oral steroid .
- > DD:

APHTHOUS ULCERS





Differential Diagnosis of Aphthous Ulcers

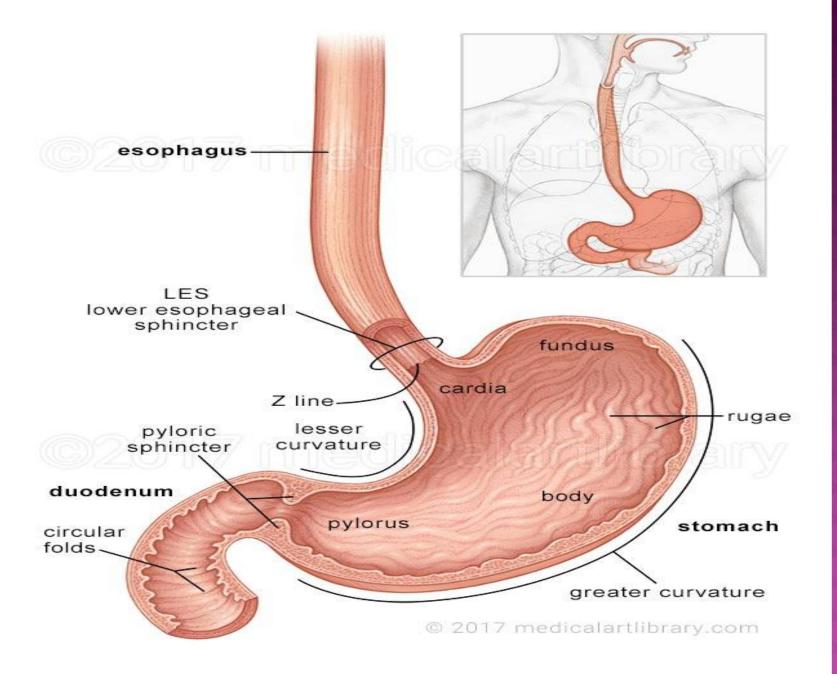
- Infection: HSV, CMV, HIV, Coxsackie virus, syphilis, histoplasmosis
- Autoimmune: Behcet's disease, SLE, Crohn's disease, bullous pemphigoid, pemphigous vulgaris, cicatricial pemphigoid, erythema multiforme.
- Neoplasms: squamous cell carcinoma.
- Medications: methotrexate, chemotherapy.
- Poor nutrition: including vitamin deficiencies.

- Tongue: glossitis (B12,folate, iron ↓), black hairy
 ,geographic.
- Gums: acute .chronic gingivitis, swelling
- <u>Teeth</u>:caries, abscess, artificial, erosions(GERD).
- Salivary glands:
- Excessive salivations (ptyalism)
- Dry mouth (xerostomia)
- Sialoadenitis (mumps; viral) bacterial .
- Salivary duct obstruction by calculus .(X- ray).
- Tumors: (parotid gland -(B or M)- 7 th cr N.

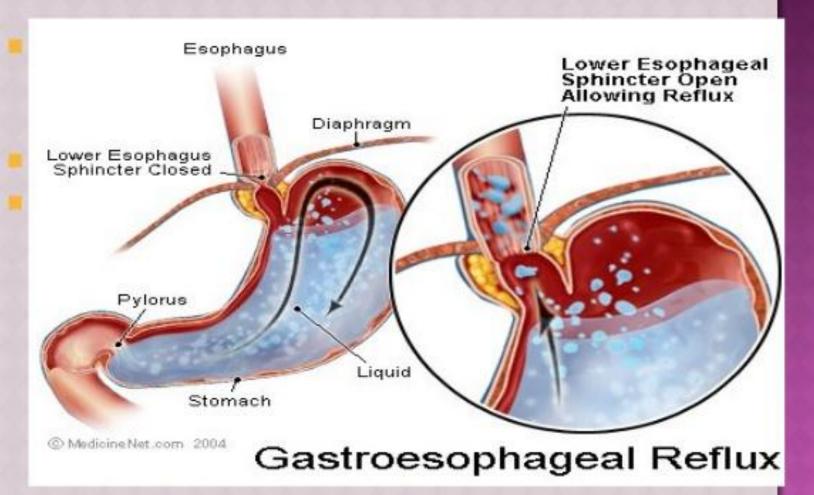
GASTROESOPHAGEAL REFLUX DISEASE (GERD)

DEFINITION:

 GERD is a chronic disorder resulting from the retrograde flow of gastro duodenal contents into the esophagus or adjacent organs, producing a various spectrum of symptoms with or without tissue damage (; NERD).



DEFINITION



PHYSIOLOGIC VS PATHOLOGIC

- Physiologic GERD
 - Postprandial
 - Short lived
 - Asymptomatic
 - No nocturnal sx

- Pathologic GERD
 - Symptoms
 - Mucosal injury
 - Nocturnal sx

EPIDEMIOLOGY

- GERD occurs in all ages but, most common in those older than 40 years of age.
- About 10-20% of people in western countries suffer from GERD symptoms on a weekly basis
- About 7% have symptoms daily.
- Except for NERD and pregnancy, no much difference in incidence between men and women.
- But for Barrett's esophagus, prevalence is more in males particularly white adult males.

PATHOPHYSIOLOGY

1) DECREASED LOWER ESOPHAGEAL SPHINCTER PRESSURE

- Primary barrier to gastro esophageal reflux is the lower esophageal sphincter
- LES normally works in conjunction with the diaphragm
- If barrier disrupted, acid goes from stomach to esophagus

May be due to

- Spontaneous transient LES relaxations
- Transient increase in intra abdominal pressure
- An atonic LES

FACTORS AFFECTING LES TONE

- Drugs that reduce LES tone include calcium channel antagonists (e.g., nifedipine, verapamil, diltiazem), nitrates, anticholinergic agents(e.g.,tricyclic antidepressants, antihistamines), and oral contraceptives and estrogen.
- Foods that reduce LES tone include chocolate, fatty foods, onions, peppermint, and garlic
- Smoking(nicotine) reduces LES tone.

PATHOPHYSIOLOGY (CONTD.)

2)DISRUPTION OF ANATOMICAL BARRIERS

- Associated with hiatal hernia
- The size of hiatal hernia is proportional to the frequency of LES relaxations
- Hypotensive LES pressures and large hiatal hernia- more chance of GERD following abrupt increase in intra abdominal pressure

3) ESOPHAGEAL CLEARANCE

- The GI acid produced spent too much time in contact with the esophageal mucosa
- Normally swallowing contributes to esophageal clearance by increasing salivary flow
- Saliva decreases with increasing age, so more often seen with elderly.

PATHOPHYSIOLOGY (CONTD.)

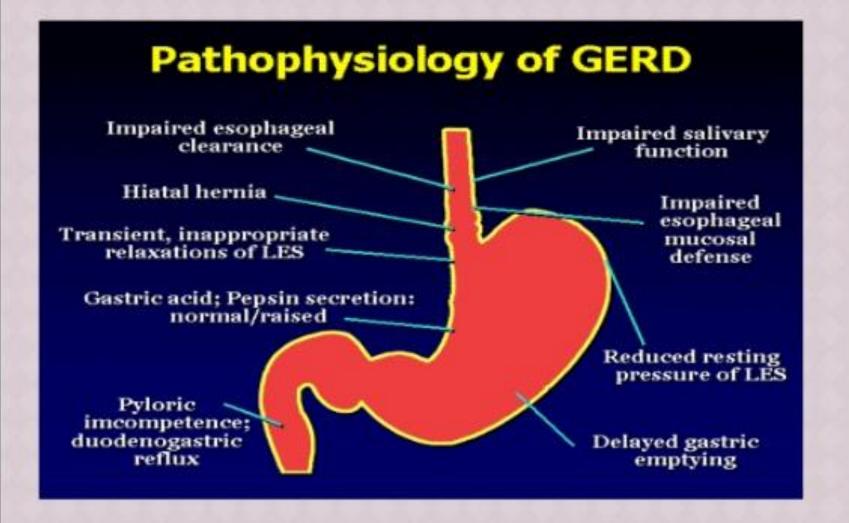
4)MUCOSAL RESISTANCE

- The mucus secreated by the mucus secreting glands involves in the protection of esophagus
- The bicarbonate s moving from the blood to the lumen can neutralize acidic refluxate in the esophagus. On repeated exposure to the refluxate or due to some defect in normal mucosal defenses hydrogen ions diffuse into the mucosa, leading to cellular acidification and necrosis leading to esophagitis.

5)DELAYED GASTRIC EMPTYING

- An increase in gastric volume may increase both the frequency of reflux and the amount of gastric fluid available to be refluxed
- Physiologic Postprandial Gastro esophageal reflux occurs

PATHOPHYSIOLOGY (CONTID.)



- Erosive esophagitis
 - Responsible for 40-60% of GERD symptoms
 - Severity of symptoms often fail to match severity of erosive esophagitis

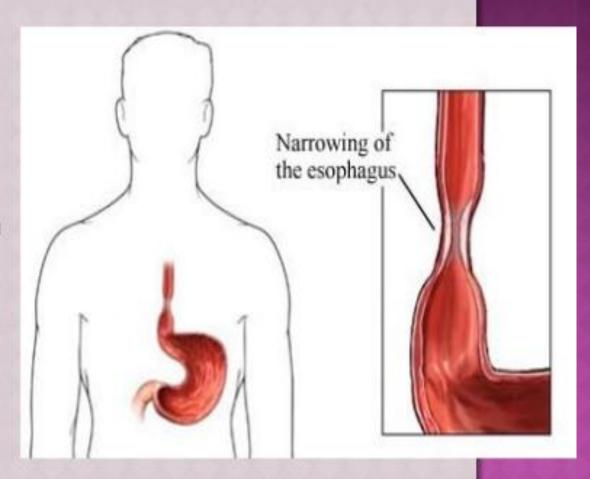




Esophagitis

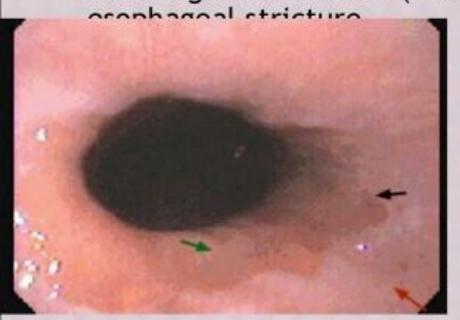
- Causes :
- GERD
- Infections (candidal >> low immunity)
- Corrosives (strong acid ,alkali)
- Drugs (NSAIDS, bisphosphonates, potassium supplements)
- Eosinophilic esophagitis. {children, eosinophilic infilteration of mucosa, topical steroid}.

- Esophageal stricture
 - Result of healing of erosive esophagitis
 - May need dilation
 - Common in the distal esophagus and are generally 1 to 2 cm in length.



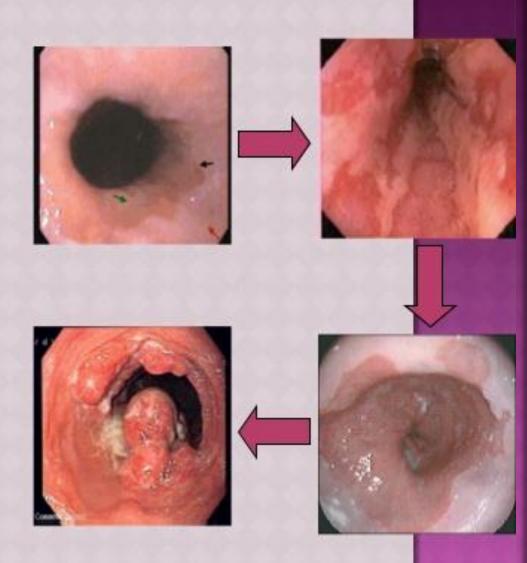
Barrett's Esophagus

- Columnar metaplasia of the esophagus, i.e replacement of the squamous epithelial lining of the esophagus by specialized columnar- type epithelium
- Associated with the development of adenocarcinoma
- Have a greater chance (30%) of developing





- Barrett's Esophagus
 - Acid damages lining of esophagus and causes chronic esophagitis
 - Damaged area heals in a metaplastic process and abnormal columnar cells replace squamous cells
 - This specialized intestinal metaplasia can progress to dysplasia and adenocarcinoma



CLINICAL MANISFESTATIONS

3 CLASSES OF SYMPTOMS TYPICAL SYMPTOMS

May be aggravated by activities that worsen gastroesophageal reflux such as recumbent position, bending over, or eating a meal high in

- Heartburn—retrosternal burning discomfort
- Regurgitation—effortless return of gastric contents into the pharynx without nausea, retching, or abdominal contractions
- Water brash (hyper salivation)
- Belching



CLINICAL MANISFESTATIONS

ATYPICAL SYMPTOMS

In some cases, these extra esophageal symptoms may be the only symptoms present, making it more difficult to recognize GERD as the cause, especially when endoscopic studies are normal.

- Nonallergic asthma
- Hoarseness
- Pharyngitis
- Chest pain
- Dental erosions

Alarming signs / symptoms

- Dysphagia
- Weight loss
- > Vomiting
- > Anorexia
- > Hematemesis or melena
- >Anemia .

May indicate complications of GERD; investigations including endoscopy must be done.

CLINICAL EVALUATION

If classic /typical symptoms like heart burn and regurgitation exist without alarming symptoms; the diagnosis of GERD can be made clinically and treatment could be initiated.

INVESTIGATIONS:

- Clinical diagnosis
- ➤ Alarm signs :

(Dysphagia – wt loss- vomiting – anorexia –hematemesis, melena)

- Endoscopy
- Intraluminal monitoring (24h PH / impedance monitoring, manometry).

MANAGEMENT

Non pharmacological

Pharmacological

Surgical

NON PHARMACOLOGICAL

Life style modification

- Weight reduction.
- Stop smoking.
- √ Stop caffeine ,alcohol.
- Dietary management.
- √ Stop drugs (NSAIDS, CCB etc) .

TREATMENT

- Goals of therapy
 - Alleviate or eliminate the patients symptoms.
 - Decrease the frequency or recurrence and duration of gastro esophageal reflux.
 - Promote healing of the injured mucosa.
 - Prevent the development of complications.

PHARMACOLOGICAL

- Anti acids:
- Prokinetic agents (adjuvant ttt): Metoclopromide, Domperidone, Itoprideetc.
- H2 receptor antagonist: (? Ranitidine (150 mg twice daily), Famotidine (20-40 mg twice daily), Nizatidine (150 mg twice daily)
- Proton pump inhibitors (PPI):

Omeprazole (20-40 mg), Pantoprazole (20-40mg), Rabeprazole (20 mg), Lansoprazole (15-30mg), Esomeprazole (20-40 mg), Dexalansoprazole (30-60 mg).

Surgical (antireflux surgery)

Endoluminal gastroplication (endoscopic)

Laparoscopic fundoplication (Nissen

fundoplication) – hiatus hernia.

Treatment of Barrett's esophagus (RF ablation)

TREATMENT

- Antireflux surgery
 - Failed medical management
 - Patient preference
 - GERD complications
 - Medical complications attributable to a large hiatal hernia
 - Atypical symptoms with reflux documented on 24-hour pH monitoring

POST SURGERY

- Dysphagia 10%
- Bloating , gas distension ,early satiety
- Persistent symptoms.
- Anti reflux medications .

