ESOPHAGEAL DISORDERS
LEARNING OBJECTIVES

• At the end of the lecture student should be able to
  – Know the quick overview of Anatomy and Physiology of Esophagus
  – Know the common esophageal disorders
  – Know the clinical picture and presentation of GERD, Reflux Esophagitis, Hiatus hernia and Barrett esophagus.

ANATOMY

• Esophagus is a muscular tube connecting the pharynx to the stomach.
• Has two muscular layers
  – Outer longitudinal layer
  – Inner circular layer
• Superior third skeletal muscles
• Middle third mixed muscles
• Inferior third are smooth muscles
• Esophagus joins the stomach just below the diaphragm after a short intra abdominal segment
• It is lined by stratified squamous epithelium
• The squamo-columnar junction lies 2cm above the gastro-esophageal junction recognized as irregular Z line endoscopically

• The esophagus is separated from pharynx by Upper Esophageal Sphincter, which is normally closed by continuous contraction of Cricopharyngeus muscle.
• At the distal end of esophagus there is Lower Esophageal Sphincter that has a high resting tone and is responsible for prevention of gastric reflux.

PHYSIOLOGY

• Its basic function is to transport swallowed food from pharynx into the stomach.
• Retrograde flow of gastric contents into the esophagus is prevented by lower esophageal sphincter.
• Entry of air into the esophagus is prevented by upper esophageal sphincter.
• Esophageal contraction are of three types.
  – Primary peristalsis
  – Secondary peristalsis
  – Tertiary contractions

**SYMPTOMS OF ESOPHAGEAL DISORDERS**
• Major esophageal symptoms are
  – Dysphagia
  – Substernal discomfort/heartburn
  – Acid regurgitation
  – Odynophagia (painful swallowing)

**COMMON ESOPHAGEAL DISORDERS**
• Motility disorders
  – GERD
  – Achalasia
  – Pharyngeal pouch
  – Diffuse esophageal spasm
  – Nutcracker esophagus
• Functional disorders
  – Functional heartburn
  – Functional chest pain of presumed esophageal origin
  – Functional dysphagia
  – Globus
• Esophagitis
  – Pill induced esophagitis
  – Infectious esophagitis
  – Reflux esophagitis
  – Eosinophilic esophagitis
• Tumors
  – Benign
    • GI stromal tumors
  – Malignent
    • Adenocarcinoma
    • Squamous
    • Small cell

**GASTROESOPHAGEAL REFLUX DISEASE**
**DEFINITION**
• Most common and costly digestive disease

• Affects approx. 30% of general population
  – 5% describes symptoms on daily basis
  – 12% at least once a week
– 25% reports symptoms once a month

– Symptoms OR mucosal damage produced by the abnormal reflux of gastric contents into the esophagus

– Often chronic and relapsing

– Either physiological or pathological

**PHYSIOLOGIC V/S PATHOLOGIC**

• Physiologic GERD
  – Postprandial
  – Short lived
  – Asymptomatic
  – No nocturnal sx
  – Pathologic GERD
  – Symptoms
  – Mucosal injury
  – Nocturnal sx

**PATHOPHYSIOLOGY**

• Primary barrier to gastroesophageal reflux is the lower esophageal sphincter
• LES normally works in conjunction with the diaphragm
• If barrier disrupted, (transient or sustained LES relaxation), acid goes from stomach to esophagus

**Clinical Manifestations**

• Most common symptoms
  – Heartburn
    • retrosternal burning discomfort
  – Regurgitation
    • effortless return of gastric contents into the pharynx without nausea, retching, or abdominal contractions
- Other symptoms include
  - Chest pain
  - Water brash
  - Globus
  - Nausea

Alarms
- **Alarm Signs/Symptoms**
  - Dysphagia
  - Early satiety
  - GI bleeding
  - Odynophagia
  - Vomiting
  - Weight loss
  - Iron deficiency anemia

Precipitating Factors for GERD
- Citrus fruits
- Chocolate
- Drinks with caffeine
- Fatty and fried foods
- Garlic and onions
- Spicy foods
- Tomato-based foods, like spaghetti sauce, chili, and pizza
- Weight gain & smoking

Diagnostic Evaluation
  - If classic symptoms of heartburn and regurgitation exist in the absence of “alarm symptoms” the diagnosis of GERD can be made clinically and treatment can be initiated
  - If PPI response inadequate despite maximal dosage then Confirm diagnosis
    - EGD
    - 24 hrs. pH monitoring

Complications
- Erosive esophagitis
- Stricture
- Barrett’s esophagus
HIATUS HERNIA

A hiatus hernia is the protrusion or herniation of the upper part of the stomach into the thorax through a tear or weakness in the diaphragm.

CAUSES

- Possible causes or contributing factors are
  - Obesity
  - Frequent coughing
  - Straining with constipation
  - Frequent bending over or heavy lifting
  - Heridity
  - Smoking
  - Stress

CLINICAL PRESENTATION

- Mostly asymptomatic
- Some patients experience
  - Heartburn
  - Regurgitation

TYPES OF HIATUS HERNIA

Hiatal hernias are classified according to the position of the esophagogastric junction and the existence of a true hernia sac.

- Two major types
  - Sliding hiatus hernia
  - Para-oesophageal/rolling hernia

- Sliding hiatus hernia:
The gastro-esophageal junction slides through the hiatus and lies above the diaphragm.

- Para-oesophageal hernia:
A small part of the fundus of the stomach rolls up through the hiatus along side the esophagus.
Diagnosis
• Typical symptoms
• Suspicious CXR
• Chest C.T.
• Upper GI Series

REFLUX ESOPHAGITIS
• Definition:
  Inflammation of the lower esophagus produced by persistent episodes of reflux of gastric contents into esophagus.
Pathophysiology/causes
  – Transient LES relaxations (tLESRs)
    • No pharyngeal contraction
    • Last longer than swallow induced LES relaxation
  – Hypotensive LES
    • Gastric distention, caffeine, smoking, chocolate
  – Anatomic disruption of the GE junction (i.e. hiatal hernia)

Symptoms
• Heartburn
• Regurgitation which worsen on bending over
• Upper GI Endoscopy is diagnostic

Treatment
• Elevating the head of the bed
• Avoiding strong stimulants of acid secretions (e.g., coffee, alcohol)
• Avoiding certain drugs (e.g., anticholinergics), specific foods (fats, chocolate), and smoking, all of which lower esophageal sphincter competence
• Giving an antacid to neutralize gastric acidity and possibly increase lower esophageal sphincter competence
• Use of cholinergic agonists to increase sphincter pressure
• Use of H2 agonists & PPIs to reduce stomach acidity
BARRETT ESOPHAGUS

- **Definition:**

  Replacement of the squamous epithelium of the distal esophagus by metaplastic specialized columnar epithelium, resembling intestine, containing goblet cells.

**Overview**

- Complication of gastroesophageal reflux disease.
- Premalignant lesion for adenocarcinoma of the esophagus, which is a deadly form of cancer.
- The incidence of esophageal adenocarcinoma has increased 6-fold over past 30 years.

**Histology of Barrett’s Esophagus**

**Pathogenesis of Barrett’s Esophagus**

**Risk factors for development of Barrett’s esophagus**

- Male gender 3 times > female gender
- White race >> Blacks & Asians
- Abdominal adiposity (obesity)
- Genetic factors suspected in some patients/families
- Chronic reflux symptoms for > 5-10 years
- Age >40-50 years; mean age at diagnosis = 55 yrs
Development of esophageal adenocarcinoma from Barrett’s esophagus

**ADENOCARCINOMA**

- Occurs in lower oesophagus
- Often associated with Barrett’s oesophagus (progresses through dysplasia to cancer)

**Potential ways of reducing the cancer risk associated with Barrett’s esophagus**

- Aggressive anti-reflux medical therapy or surgical fundoplication.
- Screen individuals with chronic GERD for BE.
- In patients known to have BE, perform surveillance to take biopsies to look for dysplasia.

**Management of high-grade dysplasia in Barrett’s esophagus**


- Options:
  1. Esophagectomy
     - High mortality, except in high-volume centers
  2. Endoscopic treatments (e.g. photodynamic therapy, endoscopic mucosal resection, other)
     - Residual intestinal metaplasia can form beneath the new squamous epithelium
  3. Intensive endoscopic surveillance (until Bx reveals adenocarcinoma)

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