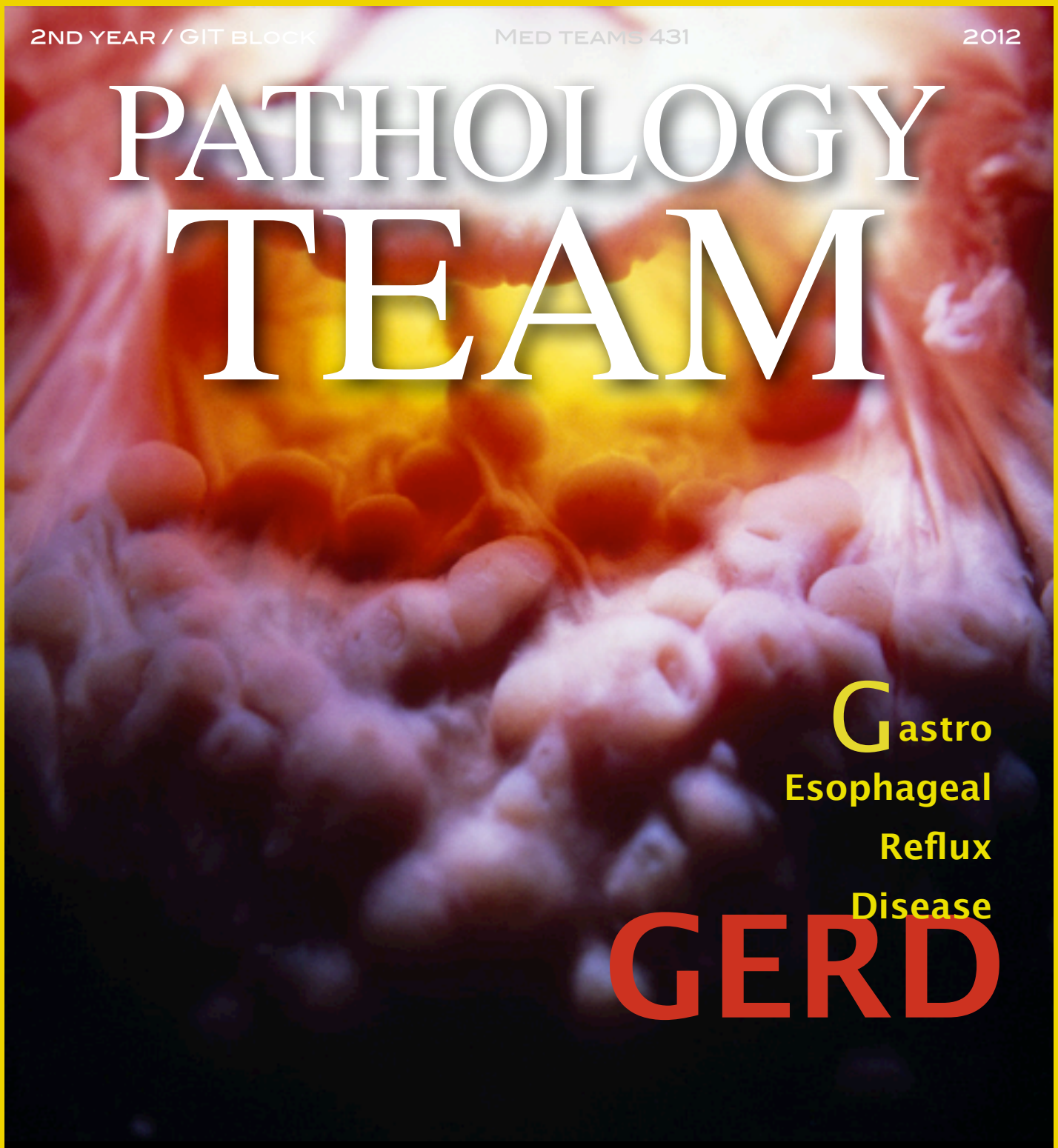
PATHOLOGY TEAM



Gastro Esophageal Reflux Disease

GERD

Done by:

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Gastrointestinal Diseases

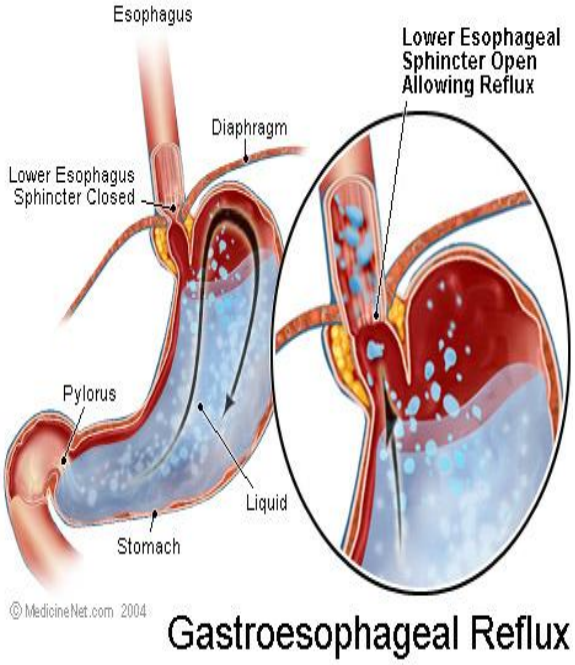
Gastro-esophageal reflux disease

- **Gastroesophageal reflux** is a normal physiologic phenomenon experienced intermittently by most people, particularly after a meal.
- **Gastroesophageal reflux disease (GERD)** occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury

Definition

American College of Gastroenterology (ACG)

- 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
- distinction between normal and GERD is blurred because some degree of reflux is physiologic is all folks
  - **Physiologic :** postprandially, short lived, asymptomatic, not during sleep
  - **Pathologic** : symptoms or mucosal injury and often with nocturnal symptoms



Gastroesophageal Reflux

Esophagitis

Esophagitis is rarely caused by agents other than reflux

Acute esophagitis may be caused by:

- infective agents:
  - Bacterial infection is very rare, but fungal infection (mainly by Candida albicans) is common
  - Viral infections of the esophagus (particularly by herpes simplex and cytomegalovirus) are seen in AIDS patient
- physical agents:
  - irradiation and by ingestion of caustic agent

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

Pathophysiology

- **Abnormal lower esophageal sphincter**
  1. Functional (frequent transient LES relaxation)
  2. Mechanical (hypotensive LES)
  3. Foods (eg, coffee, alcohol, smoking)
  4. Medications (eg, calcium channel blockers),
  5. Location : **hiatal hernia**

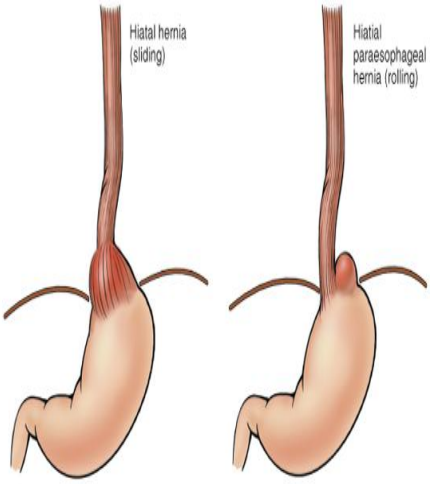
1+ 2 The most common cause of GEFD

3+4+5 decrease the pressure of the LES.

Hiatal Hernia from robbins , separation of the diaphragmatic crura and widening of the space between the muscular crura and the esophageal wall permits a dilated segment of the stomach to protrude above the diaphragm. Two anatomic patterns are recognized (Fig. 15-6): the axial, or sliding, hernia and the nonaxial, or paraesophageal, hernia. The *sliding hernia* constitutes 95% of cases; protrusion of the stomach above the diaphragm creates a bell-shaped dilation, bounded below by the diaphragmatic narrowing. In *paraesophageal hernias*, a separate portion of the stomach, usually along the greater curvature, enters the thorax through the widened foramen. The cause of this deranged anatomy, whether congenital or acquired, is unknown.

- **Increase abdominal pressure**
  1. Obesity
  2. Pregnancy
  3. Increased gastric volume
- Primary barrier to gastroesophageal reflux is the lower esophageal sphincter
- LES normally works in conjunction with the diaphragm
- If barrier disrupted, **acid goes from stomach to esophagus**

* At level of diaphragmatic hiatus—main deterrant to reflux



Clinical Manisfestations

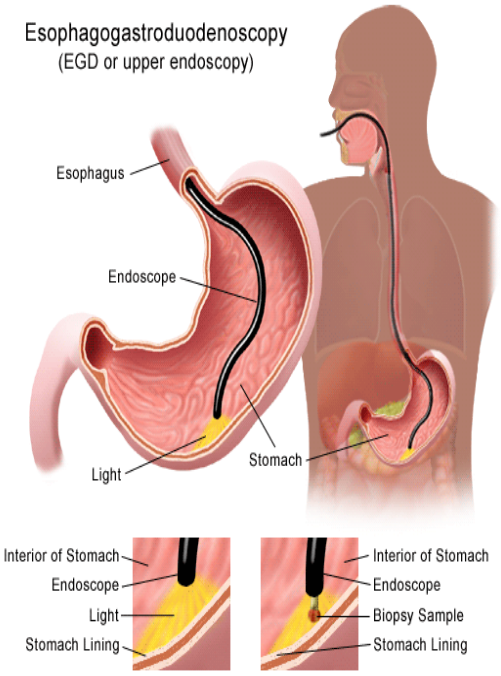
- **Most common symptoms**
  - Heartburn : retrosternal burning discomfort
  - Regurgitation : effortless return of gastric contents into the pharynx without nausea, retching, or abdominal contractions
- **Atypical symptoms**
  - coughing, chest pain, and wheezing

- gerd related chest pain may mimic angina—squeezing/burning, substernal, radiates to back, neck, jaw, arms. Minutes to hours. After meals, awakens patient from sleep, exacerbated by emotional stress
- water brash—hypersalivation—heartburn and regurg of sour fluid or tasteless saliva into mouth
- globus—lump in throat irrespective of swallowing
- odynophagia—esophageal ulcer
- nausea—infrequent
- hrt burn 70-85%//regurg 60%//dysphagi 15-20%//angina 33%//asthma 15-20%

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

* heartburn +/- regurgitation high specificity, low sensitivity

Esophagogastrodudenoscopy



Endoscopy (with biopsy if needed)

- In patients with alarm signs/symptoms
- Those who fail a medication trial
- Those who require long-term tx

- If trial of med did not work or if alarm symptoms or long term 5yrs need egd 1a evidence—dysphagia/early satiety/gi bleed/odynophagia/vomiting/wt loss/anemia
- --50-70% of patient's with gerd will have a neg egd.

Ph

- 24-hour pH monitoring
- Accepted standard for establishing or excluding presence of GERD for those patients who do not have mucosal changes
- Trans-nasal catheter or a wireless capsule shaped device

- Transnasal catheter or a wireless capsule shaped device affixed to distal esophagus
- cather positioned 5cm above manometrically defined upper limit of les
- capsul attached 6cm proximal to endoscopically defined squamocolumnar jxn
- if mucosal changes—have dx and do not need 24hph.

Complications

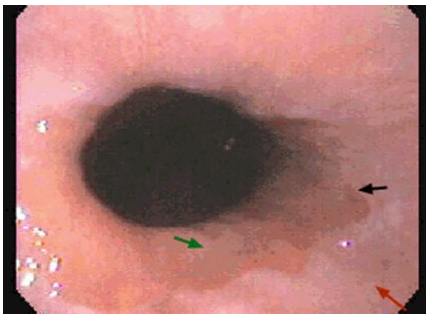
- Erosive esophagitis
- Stricture
- Barrett's esophagus

* dysphagia, odynophagia, early satiety, gi bleed, anemia, vomit, wt loss

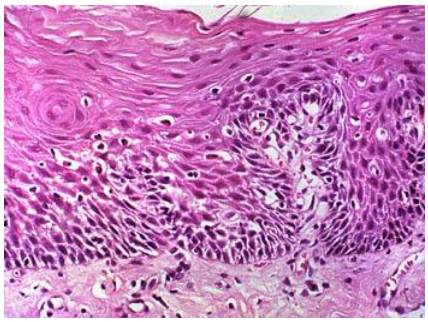
Erosive esophagitis

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

- black arrow squamo-columnar jxn—Z-line
- Z-line has undulating smooth contours
- green arrow—gastric columnar epithelium above round black sphincter
- red arow—pink white esophageal squamous epithelium
- ulcerations in 2-7%



Morphology



- Eosinophils and neutrophils
- basal zone hyperplasia,
- Elongation of lamina propria papillae

Morphology from Roobbins

The anatomic changes depend on the causative agent and on the duration and severity of the exposure. Mild esophagitis may appear macroscopically as simple hyperemia, with virtually no histologic abnormality. In contrast, the mucosa in severe esophagitis shows confluent epithelial erosions or total ulceration into the submucosa. Three histologic features are characteristic of uncomplicated **reflux esophagitis** (Fig. 15-9), although only one or two may be present: (1) eosinophils, with or without neutrophils, in the epithelial layer; (2) basal zone hyperplasia; and (3) elongation of lamina propria papillae. Intraepithelial neutrophils are markers of more severe injury.

Esophageal stricture

- Result of healing of erosive esophagitis
- May need dilation
- 4-20% of patients

Barrett's Esophagus

- Intestinal metaplasia of the esophagus
- Associated with the development of adenocarcinoma
- 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
- Many patients with Barrett's are asymptomatic

- 10-15% of patients
- RFs : male, smoker, age, obese
- Adenoca with barretts 0.5%/yr--------without barretts 0.07%/yr

Treatment

- H 2 receptor Blockers
- Proton pump inhibitors

* Antireflux surgery

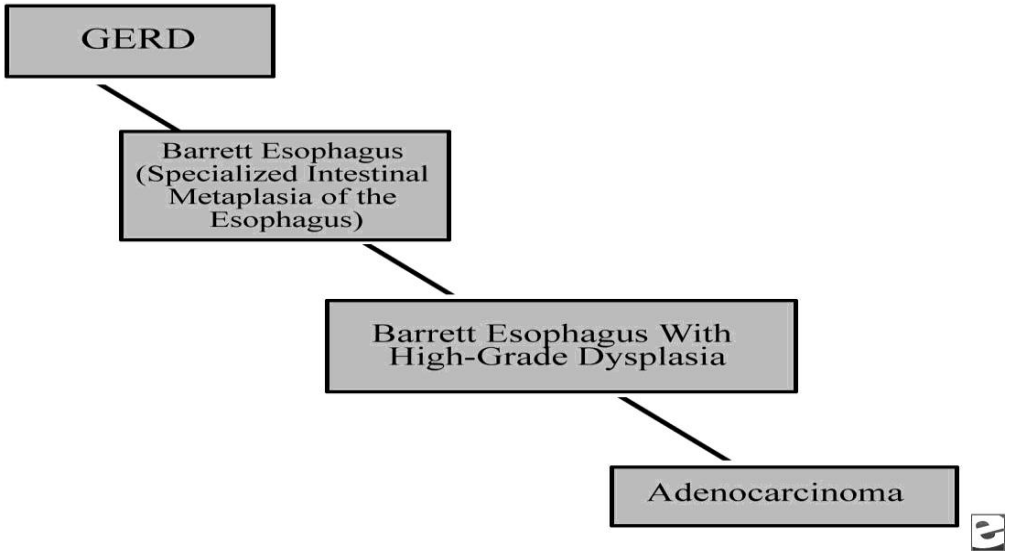
Esophageal carcinoma

- The most common malignant tumors of the esophagus are squamous carcinomas and adenocarcinomas
- The prognosis for both types of carcinoma is poor

- **Squamous carcinomas** are most common in the middle and lower esophagus. They mostly develop in men who are heavy alcohol drinkers or heavy smokers, and may be preceded by epithelial dysplastic change.
- **Adeno carcinoma** associated with Barrett's Esophagus.

Pathogenesis of GERD

1. impaired lower esophageal sphincter
2. hypersecretion of acid
3. decreased acid clearance resulting from impaired peristalsis or abnormal saliva production
4. delayed gastric emptying or duodenogastric reflux of bile salts and pancreatic enzymes.



Summary from Robbins :

Diseases of the Esophagus

Hiatal hernia: protrusion of segment of the stomach above the diaphragm; occasionally results in reflux and esophagitis.

Lacerations (Mallory-Weiss syndrome): longitudinal tears at the esophago-gastric junction caused by severe retching and vomiting; may cause upper GI bleeding.

Varices: tortuous dilated veins at the distal esophagus and proximal stomach; caused by increased portal pressure (most often due to cirrhosis), leading to increased pressure in the esophageal venous plexus; may cause severe bleeding.

Esophagitis: Inflammation of the esophageal mucosa most often caused by reflux of gastric contents; inflammatory infiltrate often contains abundant eosinophils.

Barrett esophagus: replacement of stratified squamous epithelium of distal esophagus by metaplastic columnar epithelium containing goblet cells; associated with gastroesophageal reflux in ~15% of cases; main harmful consequence is the development of dysplasia and 30- to 100-fold increased risk for adenocarcinoma.

Esophagealcarcinoma:

Squamous cell carcinomas arise from dysplastic epithelium, associated with esophagitis, smoking; may be locally invasive. Adenocarcinomas arise usually in Barrett esophagus, now more frequent in the US.