Board Review 2017

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Disclosures

- none

GERD- Gastroesophageal Reflux Disease

- Etiology (most common)
  - Transient lower esophageal relaxation
  - Weak lower esophageal sphincter
  - Hiatal hernia

Diagnostic tests

- Not necessary for most pts with GERD
- Alarm symptoms warrant further testing
  - Dysphagia, hematemesis, anemia, wt. loss
- ~50% of pts with typical GERD symptoms have normal endoscopy

A 62 yo white male presents with reflux-predominant dyspepsia and no alarm signs or symptoms

- What would you do?

a. EGD
b. Test for *H.Pylori*
c. Prescribe PPI

Answer: a EGD

- According to guidelines:
  - Over the age of 55 **or** alarm symptoms
    - Unintended weight loss
    - Dysphagia
    - GI bleeding
    - Iron def anemia
    - Abnormal physical exam (?mass in abdomen)

Diagnostic tests

- Endoscopy
  - Identifies complications of GERD
    - Ulcer, barretts, stricture, adenoca
  - UGI x-ray
    - Major usefulness: id strictures and large hiatal hernias
    - Sensitivity for GERD only 20%
  - pH monitoring
    - Indications: atypical symptoms, frequent atypical CP, refractory symptoms, Preop confirmation of GERD

PH Monitoring

- ❑Document abnormal acid exposure in EGD negative patient
- ❑After anti-reflux surgery to document abnormal reflux
- ❑Normal or equivocal EGD findings and reflux symptoms
- ❑refractory to PPI therapy

pH Monitoring

- ❑To detect reflux in chest pain patient (after 4 wk trial of PPI and cardiac evaluation)
- ❑Suspected ENT GERD (laryngitis, pharyngitis, cough) after symptoms failed to respond to 4 week trial of PPI
- ❑Document concomitant reflux in adult onset asthmatic

Impedance testing

- When combined with pH testing
  - GERD independent of pH
    - Acid and non-acid reflux
    - Can measure bolus movement

Esophageal Question

- What is the single most informative study in pts with medically refractory GERD?
  a. EGD
  b. pH monitor
  c. Barium swallow
  d. CT scan of chest

Answer: b pH monitor

- 24 or 48 hour intraesophageal pH monitor while on antisecretory meds. A similar study off medication may be considered if the diagnosis of GERD is in doubt.

Factors associated with severe esophagitis

- Low LES pressure
- Esophageal motor abnormalities
- Recumbent reflux

**Most important determinant of severe endoscopic esophagitis

- Presence of hiatal hernia also important

Treatment options

1. Lifestyle modifications--- healing rate 20-30%
2. Acid neutralization---- healing rate 20-30%
3. Acid suppression

    H2 blockers--- healing rate 50%

    PPI--- healing rate >80%
4. Prokinetics----healing rates 30-40%
5. Mechanical prevention of reflux

    laparoscopic surgery----healing rate >80%

    Endoscopic therapies---healing rate >50%

Treatment failure—optimizing PPI therapy

- Timing of Ppi therapy
  - Optimal f 30-60 minutes before a meal
  - Ppi accumulate in acid environment of actively secreting parietal cells and be converted to a reactive species via an acid-catalyzed reaction.
  - They then covalently inhibit ATPase molecules recruited to the luminal parietal cell surface

Change PPI therapy

- Gastric acid suppression can differ among PPIs
- Increase or changing of therapy may therefore improve GERD

Barretts

- Columnar epithelium replaces the stratified squamous epithelium in the esophagus.
  - Due to years of gastric reflux into esophagus
  - Associated with reflux symptoms, advancing age, male sex, and white race.
- Pt require surveillance endoscopy due to increased risk of adenocarcinoma
    - Absolute risk of cancer is ~0.005 cancer/patient annually

AGA Guidelines 2011

- Screen for Barrett's in pt with multiple risk factors
  - Male
  - >50 yrs old
  - White
  - Chronic GERD, hiatal hernia
  - Obesity (increased BMI), intra-abdominal distribution
- AGA is against screening general population with GERD

AGA cont…

- Pts with Barrett’s
  - GERD therapy to heal esophagitis
- Dysplasia should be confirmed by second expert GI pathologist
- Surveillance:
  - No dysplasia→ 3 to 5 years
  - Low-grade dysplasia→ 6 to 12 months
  - High-grade dysplasia in the absence of eradication therapy→ 3 months

Esophageal motility

- 31-year-old female presents with 6 months of dysphagia and recurrent chest pain. She has difficulty swallowing after every meal. Dysphagia has progressed to solids and liquids. She had a previous fundoplication. EGD showed it intact. Manometry showed no peristalsis, high LES pressure and no relaxation with wet swallows. The most likely diagnosis?

a. Failed reflux surgery
b. Nutcracker esophagus
c. Nonspecific motility disorder
d. achalasia

Answer d achalasia

- Findings classic.
- EGD did not show malignancy (pseudoachalasia)
- May have been misdiagnosed before fundoplication

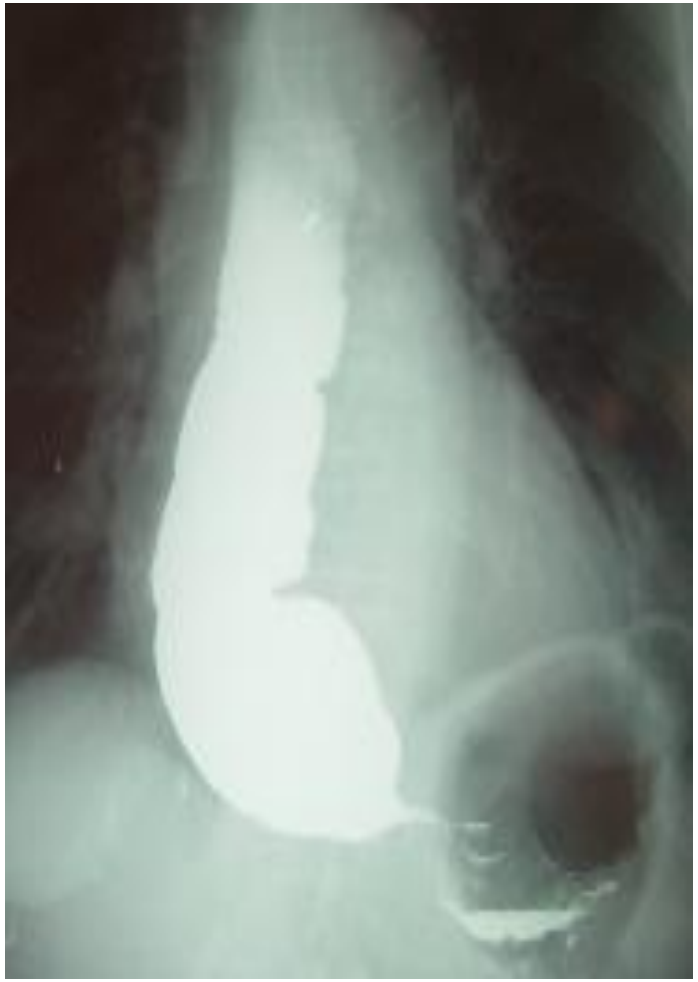
Achalasia

- Rare disease
- Loss of ganglion cells with the myenteric plexus
- Cause?
  - Increasing evidence suggests
    - Autoimmune process
      - Attributable to latent infection with herpes simplex 1 combined with genetic susceptibility

Differential diagnosis

- DES (diffuse esophageal spasm)
- Chagas Disease
  - Protozoan *Trypanosoma cruzi*

- Classic x-ray: bird-beak



Diffuse esophageal spasm

- Rare
- 3-10% of noncardiac CP or unexplained dysphagia
- Some pts improve spontaneously
- Simultaneous waves
- Tx: diltiazem
  - Treat GERD if also present

Eosinophilic esophagitis

- Typical presentation:
  - Young
  - Recurrent food impactions
  - Minimal to no GERD symptoms

EoE

- History suggests eosinophilic esophagitis
- Manometry unlikely to yield a cause
- Needs EGD with esophageal biopsies

Eosinophilic Esophagitis

- Esophageal symptoms
- **Presence of 15-20 or more eosinophils/high-power field**
- Exclusion of GERD
  - PPI trial

Eosinophilic Esophagitis

- Treatment
  - Elimination diet (6 most common food allergens)
    - Wheat, eggs, milk, soy may be sufficient (peanut, seafood other 2)
  - Acid suppression
  - Topical glucocorticoids—most pt respond with decrease eosinophil count

Oropharyngeal vs. Esophageal Dysphagia

- Difficulty initiating a swallow associated with coughing, choking, or nasal regurgitation

- Sensation of food getting stuck in the esophagus (seconds after initiating a swallow)

Oropharyngeal Dysphagia

- Abnormalities that affect the striated muscles of the mouth, pharynx and upper esophageal sphincter.

Oropharyngeal dysphagia: etiologies

- Iatrogenic
  - Meds
  - Post-surgical
- Infectious
  - Mucositis (CMV,herpes)
  - Lymes dz
- Metabolic
  - Thyrotoxicosis
  - amyloidosis
- Neurological
  - CVA
  - Parkinson’s
  - MS
  - tumor
- Structural
  - Cricopharyngeal bar
  - Zenkers diverticulum
  - Osteophytes
- Myopathic
  - Dermatomyositis
  - sarcoidosis

Testing for Oropharyngeal dysphagia

- Barium radiography
- Videofluoroscopy (usually 1st test of choice)
- Upper endoscopy
- Fiberoptic nasopharyngeal laryngoscopy
- Esophageal manometry

Goal of Therapy for Oropharyngeal dysphagia

- Improve food transfer
- Prevent aspiration

- **Risk if not treated**: life-threatening aspiration

Esophageal case presentation

- 16 yo presents with sudden onset of odynophagia after waking in the am. She is fairly healthy with hx of exercise induced asthma and acne. Her only meds are albuterol prn and doxycycline. What is the likely diagnosis?

a. Schatki ring
b. Hiatal hernia
c. Pill esophagitis
d. Infectious esophagitis

Answer: c

- Most common meds that can cause pill esophagitis:
  - Alendronate
  - Aspirin
  - Doxycycline
  - Indomethacin
  - Iron
  - Potassium
  - Quinidine

Infectious esophagitis

- May present as odynophagia and/or dysphagia
- herpes simplex virus (HSV)
- cytomegalovirus (CMV) and *Candida* species
    - HSV candida

Herpes (HSV)

- observed in patients who are immunocompromised
- occasionally be seen in patients who are immunocompetent.
- The vast majority of infections are related to HSV type 1
- may result from reactivation of HSV with spread of virus to the esophageal mucosa by way of the vagus nerve or by direct extension of oral–pharyngeal infection into the esophagus

Herpes esophagitis:treatment

- Anti-viral drugs:
  - acyclovir (Zovirax)
  - famciclovir (Famvir)
  - valacyclovir (Valtrex)