Barrett's Esophagus and Indications for Anti-reflux Procedures

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History

46 y/o. female, 109 lbs, with PMH significant for long standing acid reflux disease that failed medical management x3 years, presented for elective Nissen Fundoplication.

- PMH- HTN, Hypothyroidism, GERD, Non-dysplastic Barrett's esophagus
- PSH- Appendectomy, Rt. kidney stones lithotripsy
- Meds- Synthroid, HCTZ
- SH- No ETOH or smoking
- Allergy- PCN, IV contrast and Erythromycin
- FH- Mother (NIDDM)
Labs

- CBC- 7.9/12.2/37/215
- BMP-140/3.7/105/21/7/0.8/102
- LFTs- 6/3.4/17/16/101/0.2
- Coags- 12/21/1.1
- Urinalysis- Negative
- Pregnancy Test- Negative
Perioperative Workup

- **EGD**
  - NDBE, severe reflux esophagitis

- **24-hour esophageal PH Monitoring**
  - Demeester score 24 with increased reflux episodes over 24 hours

- **Esophageal Manometry**
  - Normal peristalsis, normal esophageal swallow transient time 8.4 sec., normal UES resistance and coordination, LES pressure 7.7 mmhg, LES total length 2.5 cm, LES intra-abdominal length 1.5 cm
s/p Laparoscopic Nissen Fundoplication
POD# 0 started on clear liquid diet
POD# 1 advanced to mechanical soft diet and discharged home.
Overview

- Anatomy
- Epidemiology
- History
- Pathophysiology
- Diagnosis
- Management
- Conclusions
Anatomy

• A muscular tube; 25 cm in length (5 cm cervical, 20 cm thoracic, 2 cm abdominal)

• 15 cm from incisor teeth is cricopharyngeal muscle, at the lower border of the cricoid cartilage (C6)

• Muscles (Inner circular, outer longitudinal)

• Esophageal Narrowing (cricopharyneal muscle, crossing of Lt. main stem & aortic arch, Hiatus of the diaphragm)
Anatomy

- Blood supply
  - Cervical sup. & inf. thyroid artery
  - Thoracic bronchial arteries from aorta
  - Abdominal branches of left gastric artery and inferior phrenic branches of splenic artery
Anatomy

• Venous Drainage

➢ Cervical *inferior thyroid vein* to innominate

➢ Thoracic *bronchial, azygus & hemiazygus to SVC*

➢ Abdominal *coronary veins to L. gastric & splenic vein*
Anatomy

• **Esophageal Sphincters**
  - **UES** - between pharynx & esophagus. It relates to cricopharyng. Muscle.
  - **LES** - 2-4 cm length, located at the EG junction, no distinct anatomical sphincter. It relaxes during swallowing / belching.
What is GERD?

Condition characterized by heartburn and regurgitation due to the loss of the HPZ.
GERD/Epidemiology

- It accounts for majority of esophageal pathologies
- **50 million Americans** have GERD at least 1/wk
- **80%** of heartburn pts have nocturnal symptoms, **65%** have both day & night
- **63%** report that it affects their ability to sleep and impacts their work the next day
- **72%** are on prescription medications
- **45%** report that current remedies do not relieve all symptoms
Symptoms of GERD

- Heart burn and regurgitation
- Belching
- Dysphagia
- Atypical chest pain
- Water brash
- Sore throat
- Throat clearing
- Hoarseness
- Cough
- Bronchospasm
- Aspiration pneumonia
- Pulmonary fibrosis

Resp. symptoms are most common in elderly (LPRD)
Risk Factors

- Prolonged gastric emptying (high fat)
- Fundic distention (overeating)
- Obesity
- Pregnancy
- Hiatal Hernia
- Medications or Food that relax LES (chocolate, caffeine, Fatty/Spicy foods, Onions, mint, ETOH, Acidic fruits)
Antireflux Mechanism

- Lower esophageal sphincter (LES)
- Crural diaphragm (pinchcock action of the diaphragm)
- Stomach (the reservoir)
- Esophageal peristalsis
Pathophysiology of antireflux barrier (HPZ)

- Resting LES pressure $10-25$ mm $< 6$ mm
- Overall sphincter length $3-4$ cm $< 2$ cm
- Intrabdominal sphincter length $2-3$ cm $< 1$ cm (most common cause)
GERD Diagnosis

• Clinically- empiric treatment

• **Endoscopy** (men>50, Caucasian, GERD>5ys, nocturnal GERD, hiatal hernia, elevated BMI, tobacco use)

• Barium Esophagram

• Manometry

• PH testing
Complication

- **Mucosal** esophagitis, stricture or bleeding
- **Extra-esophageal** laryngitis, pneumonia, asthma, and pulmonary fibrosis.
- **Metaplastic and neoplastic** Barrett's esophagus and adenocarcinoma
Treatment

- **Life style and dietary modifications**, meals 2 hours before bed time, no exercise after meal, healthy body weight, bed head elevation, no smoking

- **Medications** H2 blockers, PPI, Reglan. 80 % effective. (Discontinuation of therapy results in symptomatic relapse within 6 months in approximately 90% of patients with esophagitis)

- **Anti-reflux surgery**
Norman Barrett

• Born in South Australia 1903
• He moved to England at the age of 10
• Educated at Eton College and Trinity College, Cambridge. His lifelong nickname, Pasty, while at Eton.
• Spent most of career at St. Thomas Hospital in London
• Travelled to the United States on a Rockefeller Travelling Fellowship from 1935 to 1936 at the Mayo Clinic (decided to focus on thoracic surgery)
Norman Barrett

• In 1946- wrote a paper on spontaneous rupture of the esophagus (Boerhaave syndrome)
• In 1947- performed the first successful repair of a ruptured esophagus
• In 1950- defined the esophagus as lined by squamous epithelium and columnar-lined distal esophagus was a tubular portion of stomach
Norman Barrett

- 1953- Allison and Johnstone argued that this columnar epithelium lined structure was esophagus and not stomach, and called ulcers in this structure as **Barrett's ulcers**.
- 1957- Barrett finally agreed that was esophagus and not stomach.
- The columnar epithelium surrounding the chronic Barrett's ulcers has subsequently become known as **Barrett's esophagus**.
Barrett's esophagus

Acquired condition characterized by replacement of stratified squamous epithelium by metaplastic columnar intestinal epithelium with goblet cells, in response to injury associated with GERD.
NDBE is associated with 0.5% annual incidence of HGD or esophageal adenocarcinoma.

The prevalence of BE in the general population has been reported to be 1–25%.

It carries a 30- to 50-fold increased risk of developing esophageal adenocarcinoma.

5% of esophageal adenocarcinoma cases undergoing resection occur in patients with known Barrett’s esophagus.
BE/Epidemiology

• HGD 6-10% progression to cancer per year
• LGD 1.7% progression to HGD or cancer per year
  ➢ Curves WL, AM J Gastro 2010;105(7):1523-30
  ➢ Shaheen et al. NFJM 2009; 360(22): 2277-88

• Five-year survival with esophageal adenocarcinoma remains a dismal 13–16%
Predisposing Factors

- Defective LES
- Bile reflux
- Hiatal hernia larger than 4 cm
- Esophageal motility disorder
• GERD symptoms not present in 60% BE pts.
• Most BE undetected
  ➢ Endoscopy 22.6/100.000
  ➢ Autopsy 376/100.00
  ➢ Kaiser study, only 23/589
    EAC had known BE>6mon.

Cameron, et al. Gastro 1990;99: 918
Relative Change in the Incidence of Esophageal Adenocarcinoma and Other Cancers (1975-2001)

- Esophageal adenocarcinoma
- Lung cancer
- Melanoma
- Breast cancer
- Prostate cancer
- Colorectal cancer

Pohl H, Welch H G JNCI J Nat'l Cancer Institute 2005;97:142-146
BE Classification

1. BE without dysplasia
2. Indefinite for dysplasia
3. Low-grade dysplasia (LGD)
4. High-grade dysphagia (HGD)
   - Short-segment BE (<3 cm)
   - Long-segment BE (>3 cm)
Endoscopic Tri-Modal Imaging (ETMI) With Optical Magnification

• This technology incorporates high resolution while light endoscopy (HRE), Auto Fluorescence Imaging (AFI) and Narrow Band Imaging (NBI) in one endoscope.
Treatment

- Antisecretory therapy (PPI)
- Ablation (Thermal/ Photodynamic/Mechanical)
- Surgery
- Chemoprevention
Ablation

- **Mechanical** (Endoscopic mucosal resection)
- **Thermal** (MPEC, APC, laser, cryotherapy, radiofrequency \textit{best technique})
- **Photodynamic** (5-ALA, porfimer sodium, hematoporphyrin derivative)
ACG Guidelines

• 4 quadrant biopsies Q 1-2 cm while on PPI therapy

• Q 1 yr x 2 if negative for dysplasia, then Q 3 yr

• Any grade of dysplasia warrants confirmation by expert pathologist

<table>
<thead>
<tr>
<th>Pathological Diagnosis</th>
<th>Downgraded Diagnosis by expert pathologist</th>
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<tbody>
<tr>
<td>Indefinite for dysplasia</td>
<td>Downgraded 73%</td>
</tr>
<tr>
<td>Low-grade dysplasia</td>
<td>Downgraded 90%</td>
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<tr>
<td>High-grade dysphagia</td>
<td>Downgraded 48%</td>
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Management of Low-Grade Dysplasia: ACG Guidelines

• Confirm diagnosis by expert pathologist
• **Endoscopic ablation** as an alternative to surveillance (should be a therapeutic option)
• Repeat EGD within **6 months**
• **Q 1 yr x 2** if negative for dysplasia, then **Q 3 yr**
Management of High-Grade Dysplasia: ACG Guidelines

• Confirm diagnosis by expert GI pathologist
• HGD is a threshold for intervention
• **Endoscopic ablation** (first line therapy)
• Esophagectomy for multifocal HGD
• Repeat EGD within **3 months**
• Management options based on patient (age, comorbidity and preference)
• **BE with adenoCA involving submucosa or deeper** -> esophagectomy/ exclude antireflux Sx

• **HGBE** -> **Endoscopic therapy** alone (PDT, EMR, RFA). Esophagectomy in case of endoscopic/ delay ARS till eradication. therapy failure

• **LGBE** -> ARS before/ after **Endoscopic therapy** or Surveillance endoscopy with immediate ARS

• **NDBE** -> ARS may be performed, with or without **endoscopic therapy** (FH, long seg., age<60, prior dysplasia)
SAGES Guidelines

After successful complete eradication of BE, surveillance should continue according to their baseline Barrett’s histology grade until further evidence.

Antireflux surgery does not change recommended surveillance guidelines.
Indications of Anti-reflux Surgery

- Patient who have Failed medical treatment
- Despite successful medical treatment (patient choice, life quality considerations, lifelong need for medication intake, expense of medications, noncompliance with PPI, Young patients)
- Have complications of GERD (Esophageal stricture, Barrett’s esophagus)
- Respiratory complications secondary to GERD
Preoperative Workup

- EGD: estimate risk of progressive disease & GERD diagnosis (Strictures, large hiatal hernia, High-grade dysplasia, mass in the esophageal, gastric, or duodenal lumen will change management)

- 24-hour intraesophageal pH study: gold standard for diagnosis of GERD. Without abnormal pH study, surgery is unlikely to benefit. Gives a composite score (Johnson-DeMeester score) highly sensitive and specific (>96%) for diagnosing GERD

- Esophageal manometry: Evaluate esophageal body function. Rules out esophageal motility disorders, (achalasia or aperistalsis) should change management.

- Barium swallow: Assess for esophageal shortening. Hiatal hernia (80%), Paraesophageal hernia, Stricture or obstructing lesion
<table>
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<tr>
<th>Component</th>
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<tr>
<td>Percent total time pH &lt; 4</td>
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<tr>
<td>Percent Upright time pH &lt; 4</td>
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<tr>
<td>Percent Supine time pH &lt; 4</td>
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<tr>
<td>Number of reflux episodes</td>
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<tr>
<td>Number of reflux episodes ≥ 5 min</td>
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<tr>
<td>Longest reflux episode (minutes)</td>
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Goal of surgery

Relief of symptoms and cessation of ongoing epithelial damage related to reflux

Restore normal structure/HPZ while preserving patient’s ability to swallow, and to belch

- Restore pressure (>12 mmHg)
- Restore length (at least 3 cm)
- Place adequate length in abdomen (2 cm)
Principles of Surgical Therapy

• Laparoscopic fundoplication - most common antireflux operation

• Intraoperative endoscopic evaluation of the created valve is valuable to confirm the hallmarks of a successful fundoplication

• A deep groove on the surface of the fundoplication indicates that the repair is too tight

• If adequate esophageal length cannot be achieved secondary to shortening of the esophagus, wedge gastroplasty as a lengthening procedure
Steps of Antireflux Procedure

- **Gastrohepatic ligament dissection** - 25% have left hepatic artery coming from left gastric artery in the GH ligament.

- **Complete Crural dissection with identification both vagi** (injury may cause the failure of sphincter relaxation and delayed gastric emptying).

- **Circumferential mediastinal esophageal mobilization** resulting in 3 cm of tension-free intra-abdominal esophageal length.

- **Mobilization of the gastric fundus** by dividing the short gastric and posterior gastric vessels.

- **Posterior crural closure** is performed to enable easy passage of the esophageal dilator.

- **Gastric fundus used to create the fundoplication** around the distal esophagus (should be no longer than 3 cm and created over a 60F bougie).
“nipple” valve in a complete fundoplication versus a “flap” valve in a partial fundoplication.
SYMPTOMATIC GERD

Endoscopy - barium swallow - esophageal manometry - (pH monitoring)

- Hypotonic - normotonic LES
- Normal esophageal length
- Normal esophageal motility
  → Nissen fundoplication

- Hypotonic LES
- Normal esophageal length
- Poor esophageal motility
  → Partial fundoplication

- Hypotonic LES
- Short esophagus

  → Collis gastroplasty
  → Partial fundoplication
  → Nissen fundoplication
  → Thoracic approach (Belsey - Nissen)

B. Dallemagne
Complications

- Gastric and Esophageal perforation
- Pneumothorax
- Dysphagia - postop. edema or tight fundoplication (too much resistance for the esophageal pump.)
- Symptoms recurrence
- Inability to vomit and belch
- Increased flatulence
The Effect of Antireflux Surgery on Esophageal Carcinogenesis in Patients With Barrett Esophagus
A Systematic Review

Eugene Y. Chang, MD,* Cynthia D. Morris, PhD, MPH,† Ann K. Seltman, MD,* Robert W. O’Rourke, MD,* Benjamin K. Chan, MS,† John G. Hunter, MD,* and Blair A. Jobe, MD*‡
Conclusion

• Successful antireflux surgery is based on abnormal 24-hr pH score, typical GERD symptoms, and symptomatic improvement in response to acid suppression therapy.

• Laparoscopic antireflux surgery is effective at restoring the mechanical barrier to reflux with significant improvements in the LES pressure and acid reflux exposure.
Conclusion

• Antireflux surgery does not change recommended surveillance and endoscopic therapy guidelines for patients with BE

• The available evidence is inconclusive about the resolution or improvement of Barrett’s after antireflux surgery
References


