SURGICAL DISORDERS OF THE ESOPHAGUS

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GASTROESOPHAGEAL REFLUX DISEASE (GERD)

- Most common problem of the esophagus
- Increase esophageal exposure to gastric juice
- Caused by 3 mechanisms
  1. Incompetency of cardia
  2. Inefficient esophageal clearance
  3. Abnormality of gastric reservoir

DIAGNOSIS OF GERD

- Symptoms: heart burn, regurgitation aspiration
- Endoscopic esophagitis: rosette’s like
- Manometry: low amplitude peristalsis
  
  ↓ LES pressure

- pH study: 24 hr-pH monitoring
SURGICAL TREATMENT OF GERD

- Symptoms caused by defective LES
- Antireflux procedures
- Indication for antire
  1. Failure medical regimens; after 8-12 weeks of intensive acid suppression therapy
  2. Increase esophageal exposure: pH-study
  3. Mechanically defective LES
  4. Adequate esophageal body motor function

PRINCIPLES OF SURGICAL THERAPY

- Restore the pressure of LES 2 times resting gastric pressure
- Adequate length of LES in abdomen
- Constructed cardia relax on deglutition
- Floppy
- Fundoplication placed in the abdomen
TYPES OF ANTIREFLUX

1. Nissen’s fundoplication
   - most common antireflux procedure
   - 360 degree wrap around

2. Belsey Mark IV
   - Partial fundoplication
   - 270-280 degree : anteriorly wrap
   - Transthoracic approach

TYPES OF ANTIREFLUX (cont.)

3. Dor’s and Toupet’s
   - 180 degree (half) fundoplication
   - Dor’s – anteriorly
   - Toupet’s – posteriorly

4. Hill’s and Collis’s
   - Median arcuate ligament repair
   - Most physiologic
   - Short esophagus → Collis’s gastroplasty
COMPLICATIONS OF GERD

1. Esophagitis; objective findings, not symptom
2. Ulceration; pain on swallowing
3. Stricture; 2-3 cm above EG-junction
4. Barrett’s esophagus 7-20% of GERD
5. Adenocarcinoma 0.5% of Barrett’s

HIATAL HERNIAS

- 3 type of esophageal hiatal hernia
- Type I: sliding hernia; upward dislocation of cardia into posterior mediastinum
- Type II: paraesophageal hernia; upward dislocation of gastric fundus alongside a normally position cardia
- Type III: combined sliding – rolling; upward dislocation of both the cardia and fundus
MECHANISMS OF HIATAL HERNIA

1. Structural deterioration of phrenoesophageal membrane; explain the higher incidence in the older age
2. Persistent intra-abdominal presore cranial direction of membrane stretching
3. Defective, perhaps congenital in the esophageal hiatus anterior to the esophagus
4. Posterior fixation of the cardia to preaortic fascia and median arcuate ligament

SYMPTOMS OF HIATAL HERNIA

- Type I: reflux symptoms
- Type II: dysphagia and postprandial fullness, bleeding from ulcer of gastric mucosa, incarceration
- Type III: mixed
DIAGNOSIS OF HIATAL HERNIA

- Chest film upright; air-fluid level behind the cardiac shadow
- LGI-series; detect paraesophageal greater than sliding since type II is permanent herniation
- Fiberoptic esophagoscopy

THERAPY OF HIATAL HERNIA

- Type I: anatomical reduction and fixation of cardia and fundoplication
- Type II: anatomical reduction of fundus and repair of hiatal defect
- Fundoplication for type II depends on sphincter length and pressure of LES
ACHALASIA

- Cardiospasm is synonymous
- Classical characters: myenteric ganglion cell degeneration and chronic inflammation of esophageal smooth muscle
- Similarly with Chagas’ disease: Trypanosoma cruzi
- Both sexes are equally affected

ACHALASIA (CONT.)

- Age 25-60 years, common at 40 years
- Dysphagia, regurgitation, weight loss solid food dysphagia and dysphagia for liquid as well
- Emotional stress, cool liquid worsen dysphagia
ACHALASIA
(cont.)

- Chest pain and heart burn is described near one-half of patients
- 7% developed squamous cell carcinoma after 15-25 years

INVESTIGATION FOR ACHALASIA

- Chest film; widened mediastinum, air-fluid level, absence of gastric air bubble and pulmonary aspiration
- Barium swallowing: failure or delayed esophageal clearance, dilated esophageal body, bird’s beak or rat-tail appearance
INVESTIGATION FOR ACHALASIA
(cont.)

- Endoscopy: rule out malignancy with pseudoachalasia, hiatal hernia (4%) scope can be passed through LES in typical achalasia, retension esophagitis
- Manometry
  1. Absence of distal esophageal peristalsis
  2. Incomplete or abnormal LES relaxation
  3. Elevation of LES pressure
  4. Elevation of intra-esophageal pressure
Treatment of Achalasia

1. Oral nitrates and cal-channel blockers
2. Intraspincteric injection of botulinum toxin
3. Pneumatic dilatation
4. Heller’s cardiomyotomy
DIFFUSE ESOPHAGEAL SPASM (DES)

- Dysphagia with chest pain
- High amplitude, simultaneous contraction
- Stress and cold liquid precipitate symptoms
- Ba-swallowing: pseudodiverticulum or cork-screw rosary
- Cal-blockers and nitrates
- Long esophageal myotomy
ZENKER’S DIVERTICULUM

- Mucosal outpouching occurring through triangular bare area (Killian’s triangle)
- Prominent on left side
- Premature contraction of UES
- Most common after 50 years
ZENKER’S DIVERTICULUM  
(cont.)

- Symptoms: dysphagia, GERD, aspiration
- Incidental finding on barium (no symptom)
- Squamous cell carcinoma 0.4% of diverticulum
- Treatment: surgical cricopharyngomyotomy
- Large diverticulum, excision or pexy
- Small diverticulum: left untouched

SCLERODERMA

- Collagen-vascular disease
- Most common GI involvement is esophagus
- Effect smooth muscle component
- Dysfunction of distal third and LES
- 75-85% of scleroderma has esophageal involvement
SCLERODERMA
(cont.)

- Predominant in white female
- Age 30-50 years
- Presentations: dysphagia, heart burn
- Manometry: low to absent LES pressure, weak distal esophageal peristalsis, normal upper esophageal pressure and motility

SCLERODERMA
(cont.)

- Medical treatment of GERD
- Surgical treatment: Belsey Mark IV much better than Nissen since weak peristasis
- For esophageal shortening: Collis’s augmented
ESOPHAGEAL PERFORATION

- Most devasting perforation of GI tract
- Causes: iatrogenic 60% traumatic 25% and spontaneous 15%
- Sites: 25% cervical 60% thoracic and 15% abdominal esophagus

CERVICAL ESOPHAGEAL PERFORATION

- Occur posteriorly where esophageal wall is thinnest
- Late presentation: infection down to the mediastinum
- Clinical syndrome: neck stiffness and ache, bloody regurgitation, subcutaneous emphysema
- Diagnosis: gastrograffin swallowing
- Treatment: surgery most appropriate
THORACIC ESOPHAGEAL PERFORATION

- Mortality rate three times of cervical perforation
- Delayed treatment over 24 hours mortality to 50%
- Mortality was twice if operation done after 24 hours compared to that done before 24 hours
- Mortality with underlying pathology 23% while without underlying pathology 4%

THORACIC ESOPHAGEAL PERFORATION (cont.)

- Severe retrosternal discomfort
- Fever, dysphagia
- Mediastinal emphysema by chest film
- Esophagogram: demonstrate 60% cervical perforation but 90% thoracic perforation
- Optimal period for closure was within the first 6 hours
THORACIC ESOPHAGEAL PERFORATION
(cont.)

Cameron criteria for conservative management

1. Localized to mediastinum
2. Contained between mediastinum and visceral pleura
3. Cavity drained back to esophagus
4. Minimal symptom
5. Minimal evidence of sepsis
BOERHAAVE’S SYNDROME

- Hermann Boerhaave of Netherlands 1724
- Spontaneous injury
- Bursting pressure 350-700 mmHg
- Male to female 4:1
- Mortality 20-50%
- Common at distal one third left side
BOERHAAVE’S SYNDROME
(cont.)

- Sudden excruciating chest pain
- Vomitus blood-streaked
- Abdominal rigidity and tenderness
- Pleural effusion, hydropneumothorax
- Subcutaneous neck crepitus is pathognomonic

- Plain chest upright most valuable
- Mediastinal air, widen mediastinum
  - airfluid level, pleural effusion
- Water-soluble esophagogram confirm
  - site of leakge
- Urgent thoracotomy is recommend
MALLORY WEISS SYNDROME

- Hematemesis: mucosal tear at EG junction
- Mallory and Weiss 1929
- Forceful vomiting followed by hematemesis
- Longitudinal laceration of mucosa extending deeply into submucosa
- Gastric laceration more common than esophageal: isolated esophageal uncommon

Flexible endoscopy is the investigation of choice
- Selected angiogram for active bleeding
- Barium not helpful
- Conservation treatment first
- 7% required surgical intervention
- Vertical gastrotomy (not cross EG) with deep interrupted non-absorbable suture
CORROSIVE INGESTION

- Concentrated alkali most common: sodium hydroxide → liquefaction necrosis
- Weaker alkali: washing soda, ammonia
- Concentrated acid: HCl, sulfuric, nitric → coagulation necrosis
- Esophageal tissue susceptible to alkali

COMPLICATION OF CORROSIVE BURN

1. Acute perforation of the esophagus, stomach or intestine
2. Subsequence fibrous stricture of the esophagus, stomach (rare)
3. Destruction of larynx by overspill
DIAGNOSIS OF CORROSME INJURY

- Acute phase; early endoscopy
- Subacute; radiological examination
- Radiologic examination in acute phase is indicated for suspected perforation

EMERGENCY TREATMENT

- No emergency treatment can influence the extent or depth of injury
- Not to neutralize, irrigation or dilution
- NPO with IV-fluid
- Steroids and antibiotic still controversial
- Chest film to exclude perforation
INDICATION FOR EMERGENCY

1. Signs of peritonitis: peritoneal irritation
2. Free air under diaphragm
3. Interstitial air in the wall of stomach or jejunum
4. Alkali aspiration from the stomach
5. Radiographic confirmation of perforation

LEIOMYOMA OF ESOPHAGUS

- Most common benign tumor
- Arise from muscularis mucosae
- 90% at middle-lower third
- Intramural encapsulated tumor
- Relatively avascular: blood supply from submucosa
- Differentiation between leiomyosarcoma and
- leiomyoma difficult
LEIOMYOMA OF ESOPHAGUS
(cont.)

- Age 20-59% years
- Dysphagia (48%) pain or discomfort (48%)
  weight loss (20%)
- Radiography, smooth filling defect
- 50% incidental finding
- CT, aortogram may be helpful

Indication for surgical excision (LEIO)

1. Relief of dysphagia or pain
2. Progressive increase in size
3. Confirmation of pathologic diagnosis
4. To facilitate the performance of other procedure;
   antireflux, myotomy and diverticulopexy
5. Enucleation is accepted
CANCER OF ESOPHAGUS

- 4 types: squamous cell CA, adeno CA, carcinoma sarcoma, and sarcoma
- Squamous most common
- Adenocarcinoma increasing in frequency
- Carcinosarcoma and sarcoma 1%

CANCER OF ESOPHAGUS (cont.)

- 3-8/100,000
- High risk area 20-30 times
- Caspian littoral in Iran
- Honan province of China
- Transkei of South Africa
Etiology of ESO.cancer

- Nitrosamines, hot tea pipe tar
- Smoking, alcohol
- Achalasia, lye-strictures, plummer-Vinson syndrome, tylosis
- For adenocarcinoma: reflux is suspected

Presenting Complaints

- Dysphagia is most common
- Approximately 75-90% involvement of esophageal circumference causes symptoms
- Almost always greater than 2 cm
- Weight loss, adynophagia, regurgitation and aspiration
DIAGNOSIS OF ESOPHAGEAL CANCER

- Mass screening in high risk area
- Radiology lesion < 1 cm might be missed
- Endoscopy popular in Japan
- Cytologic screening
- EUS, CAT-scan, CXR, bone scan, liver scan
### TABLE 12-2. GEOGRAPHIC DISTRIBUTION:
MORTALITY PER 100,000

<table>
<thead>
<tr>
<th>Locality</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iran: Gonbad</td>
<td>114.6</td>
<td>130.8</td>
</tr>
<tr>
<td>Gilan</td>
<td>20.1</td>
<td>6.2</td>
</tr>
<tr>
<td>South Africa: Transkei</td>
<td>70.4</td>
<td>33.3</td>
</tr>
<tr>
<td>China: Linskien</td>
<td>85.8</td>
<td>54.0</td>
</tr>
<tr>
<td>Rhodesia</td>
<td>75.6</td>
<td>36.1</td>
</tr>
<tr>
<td>USSR: Turkmenia</td>
<td>51.1</td>
<td>33.2</td>
</tr>
<tr>
<td>Lithuania</td>
<td>2.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Switzerland</td>
<td>20.0</td>
<td>3.8</td>
</tr>
<tr>
<td>France</td>
<td>15.5</td>
<td>2.3</td>
</tr>
<tr>
<td>India: Bombay</td>
<td>14.4</td>
<td>11.0</td>
</tr>
</tbody>
</table>

### Localities outside the U.S.

<table>
<thead>
<tr>
<th>Locality</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puerto Rico</td>
<td>12.3</td>
<td>4.9</td>
</tr>
<tr>
<td>Japan</td>
<td>8.5</td>
<td>3.6</td>
</tr>
<tr>
<td>England</td>
<td>7.0</td>
<td>3.6</td>
</tr>
<tr>
<td>Germany</td>
<td>5.8</td>
<td>2.7</td>
</tr>
<tr>
<td>Denmark</td>
<td>5.7</td>
<td>3.3</td>
</tr>
<tr>
<td>Australia</td>
<td>5.4</td>
<td>2.1</td>
</tr>
<tr>
<td>Canada</td>
<td>3.9</td>
<td>2.0</td>
</tr>
<tr>
<td>Sweden</td>
<td>3.7</td>
<td>2.9</td>
</tr>
<tr>
<td>USA: White</td>
<td>6.0</td>
<td>1.6</td>
</tr>
<tr>
<td>Black</td>
<td>19.5</td>
<td>4.2</td>
</tr>
</tbody>
</table>
### TABLE 12-5. MANNEL’S STAGING BASED ON JAPANESE SYSTEM

<table>
<thead>
<tr>
<th>Tumor (T)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tm, Tsm</td>
<td>Confined to mucosa, submucosa</td>
</tr>
<tr>
<td>Tmp</td>
<td>Confined to muscularis propria</td>
</tr>
<tr>
<td>T1</td>
<td>Invasion reaching adventitia</td>
</tr>
<tr>
<td>T2</td>
<td>Invasion into adventitia</td>
</tr>
<tr>
<td>T3</td>
<td>Invasion into neighboring structures</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lymph node metastases (N)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>N0</td>
<td>No metastases</td>
</tr>
<tr>
<td>N1</td>
<td>Metastases to paraesophageal nodes of involved segment</td>
</tr>
<tr>
<td>N2</td>
<td>To nodes of esophagus adjacent to tumor</td>
</tr>
<tr>
<td>N3</td>
<td>To paraesophageal or periesophageal nodes of distant esophageal segments</td>
</tr>
<tr>
<td>N4</td>
<td>To nodes beyond N3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Organ metastases (M)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
<td>None</td>
</tr>
<tr>
<td>M1</td>
<td>Present</td>
</tr>
</tbody>
</table>

Pleural dissemination (P)

<table>
<thead>
<tr>
<th>PL0</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>PL1</td>
<td>Present</td>
</tr>
</tbody>
</table>

*Adapted from Mannell, 1982c.*

### TABLE 12-6. THOMPSON’S STAGING BASED ON AMERICAN JOINT COMMITTEE

<table>
<thead>
<tr>
<th>Tumor (T)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>T0</td>
<td>No demonstrable tumor</td>
</tr>
<tr>
<td>T1s</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>T1</td>
<td>Length 5 cm or less, no obstruction, not circumferential, no extraesophageal spread</td>
</tr>
<tr>
<td>T2</td>
<td>Length over 5 cm, with obstruction and is circumferential but no extraesophageal spread</td>
</tr>
<tr>
<td>T3</td>
<td>Tumor extension into mediastinal structures</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lymph nodes (N)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical esophagus (neck nodes)</td>
<td>N0   No node metastases</td>
</tr>
<tr>
<td>N1</td>
<td>Unilateral movable nodes</td>
</tr>
<tr>
<td>N2</td>
<td>Bilateral movable nodes</td>
</tr>
<tr>
<td>N3</td>
<td>Fixed nodes</td>
</tr>
<tr>
<td>Thoracic esophagus (excludes neck and abdominal nodes)</td>
<td>N0   No node involvement</td>
</tr>
<tr>
<td>N1</td>
<td>Nodal metastases</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Distant metastases (M)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
<td>None</td>
</tr>
<tr>
<td>M1</td>
<td>Metastases present (includes neck and abdominal nodes for thoracic tumor)</td>
</tr>
</tbody>
</table>

*Adapted from Thompson, 1983.*