Management of Failed Anti-Reflux Surgery

Essay

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Abstract

Background: Failure of anti-reflux surgery means persistence of the previous symptoms or appearance of new symptoms after surgery and this needs a careful evaluation. Investigations should be directed at both the original preoperative evaluation and current situation. Esophageal manometry might show borderline function predisposing to persistent symptoms postoperatively.

Aim of the work: The diagnosis of the causes of failed anti-reflux surgery and select patients who are in need for re-do surgery.

Summary: GERD is the most common condition affecting the upper gastrointestinal tract. This is partly due to the declining incidence of peptic ulcer as the incidence of infection with Helicobacter pylori has reduced as a result of improved socioeconomic conditions along with a rising incidence of GERD in the last 20–30 years.

Key words: GERD – Esophagus – Manometry – Endoscopy – Hiatus hermia – Nissen fundoplication – Laparoscopy – Esophagram.

INTRODUCTION

Laparoscopic fundoplication became the gold standard in the surgical therapy of GERD. In comparison with open procedure, Laparoscopic anti-reflux surgery has a lower morbidity rate, a better early and late postoperative outcome and is more cost-effective. Laparoscopic 360 degrees Nissen fundoplication with crurorаphy is the standard procedure, whereas the 270 degrees Toupet technique is the technique of choice for esophageal motility disorder ***(Wehrli, 2005).***

Despite the variety of surgical operations and strategies employed, the overall failure rate is constant at approximately 11% - 14 % and almost 4% - 7% of dissatisfied patients require a re-operation ***(Migliore, 2009).***

Failure of anti-reflux surgery means persistence of the previous symptoms or appearance of new symptoms after surgery and this needs a careful evaluation. Investigations should be directed at both the original preoperative evaluation and current situation. Esophageal manometry might show borderline function predisposing to persistent symptoms postoperatively ***(Zhou et al., 2013).***

Unsuccessful results of surgery for GERD whether associated with hiatal hernia or not, is a frustrating event for the surgeon as well as far for the patient because of the well-known poor quality of life ***(Furnée et al., 2009).***

Aim of the Work

The aim of this essay is the diagnosis of the causes of failed anti-reflux surgery and select patients who are in need for re-do surgery.

Chapter 1

ANATOMY

Anatomy of the esophagus

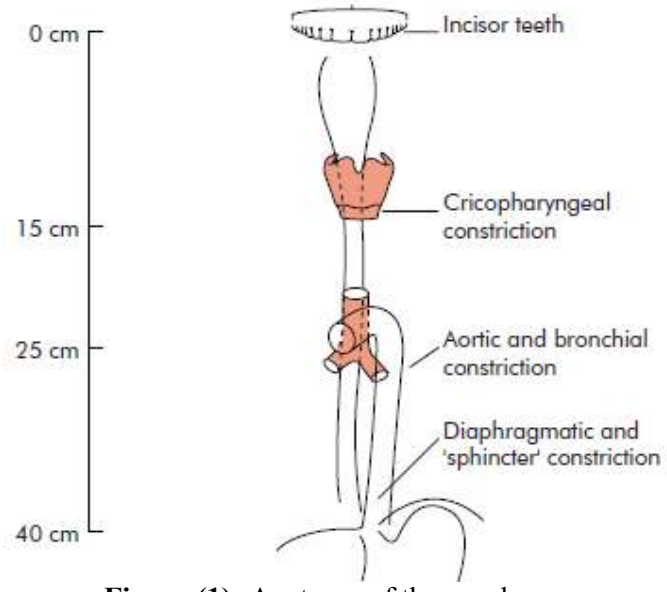

Figure (1): Anatomy of the esophagus.

- The esophagus develops from the cranial portion of the foregut and is recognizable by the 3rd week of gestation.
- The normal esophagus is a hollow, highly distensible muscular tube that extends from the epiglottis in the pharynx, at about the level of C6 vertebra, to the gastroesophageal junction at the level of the T11 or T12.
- Measuring between 10 and 11cm in the newborn, it grows to a length of about 25cm in the adult.

- For the endoscopist, the esophagus is recorded as the anatomic distance between 15 and 40cm from the incisor teeth, with the gastroesophageal junction located at the 40-cm point.
- Several points of luminal narrowing can be identified along its course-proximally at the cricoid cartilage, midway in its course alongside the aortic arch and at the anterior crossing of the left main bronchus and left atrium, and distally where it pierces the diaphragm.
- Although the pressure in the esophageal lumen is negative compared with the atmosphere, manometric recordings of intraluminal pressures have identified two higher-pressure areas that remain relatively contracted in the resting phase.
- A 3-cm segment in the proximal esophagus at the level of the cricopharyngeus muscle is referred to as the upper esophageal sphincter (UES).
- The 2 to 4cm segment just proximal to the anatomic gastroesophageal junction, at the level of the diaphragm, is referred to as the lower esophageal sphincter (LES). Both sphincters are physiologic, in that there are no anatomic landmarks that delineate these higher-pressure regions from the intervening esophageal musculature (Figure 1).

(Stadlhuber et al., 2009)

Histological structure of esophagus

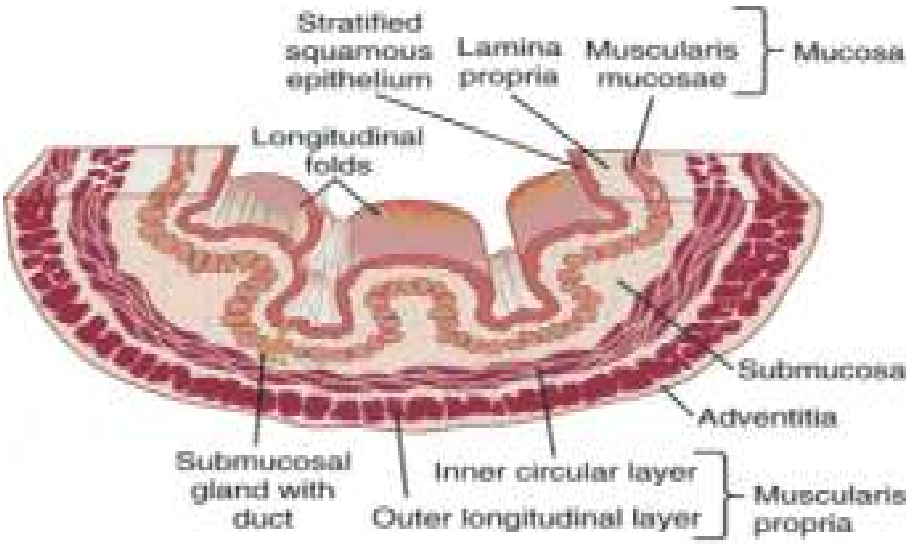

Figure (2): Histological structure of esophagus.

- The wall of the esophagus consists of a **mucosa**, **submucosa**, **muscularis propria/ externa**, and **adventitia**, reflecting the general structural organization of the gastrointestinal tract (Figure 2).
- The **mucosa** has a smooth, glistening, and pink-tan surface. It has three components;
  - A non-keratinizing stratified squamous epithelial layer.
  - Lamina popria.
  - Muscularis mucosa.

1. The epithelial layer has mature squamous cells overlying basal cells. The basal cells, constituting 10% to 15% of the mucosal thickness, are reserve cells with great proliferative potential.
   - A small number of specialized cell type, such as melanocytes, endocrine cells, dendritic cells, and lymphocytes, are present in the deeper portion of the epithelial layer.
2. The lamina propria is the non-epithelial portion of the epithelial layer, above the muscularis mucosae.
   - It consists of areolar connective tissue and contains vascular structures and scattered leukocytes.
   - Finger-like extensions of the lamina propria, called papillae, extend into the epithelial layer.
3. The muscularis mucosa is a delicate layer of longitudinally oriented smooth-muscle bundles.
   - The submucosa consists of loose connective tissue containing;
     - Blood vessels.
     - A rich network of lymphatics.
     - A sprinkling of leukocytes with occasional lymphoid follicles.
     - Nerve fibers (including the ganglia of Meissner plexus).
     - Submucosal glands.

- Submucosal glands connected to the lumen by squamous epithelium-line ducts are scattered along the entire esophagus but are more concentrated in the upper and lower portions. Their mucin-containing fluid secretions help lubricate the esophagus.
- As is true throughout the alimentary tract, the muscularis propria/externa consists of an inner circular and an outer longitudinal coat of smooth muscle with an intervening, well-developed myenteric plexus (Auerbach plexus).
    - The muscularis propria striated muscle fibers from the cricopharyngeus muscle.
    - Besides creating a unique histologic interplay of smooth muscle and skeletal muscle fibers, this feature explains why skeletal muscle disorder can cause upper esophageal dysfunction.
- In sharp contrast to the rest of the gastrointestinal tract, the esophagus is mostly devoid of a serosal coat. Only small segments of the intra-abdominal esophagus are covered by serosa; the thoracic esophagus is surrounded by fascia that condenses around the esophagus to form a sheath like structure.
- In the upper mediastinum, the esophagus is supported by this fascial tissue, which forms a similar sheath around adjacent structures, the great vessels and the tracheobronchial tree. This intimate anatomic proximity

to important throracic viscera is of significance in permitting the ready and widespread dissemination of infections and tumors of the esophagus into the posterior mediastinum. The rich network of mucosal and submucosal lymphatics that runs longitudinally along the esophagus further facilitates spread.

(Muller-Stich et al., 2006)

As a summary, wall of esophagus composed of:

1. ***Mucosa***
   - Epithelial cells (non-keratinized stratified columnar epithelium).
   - Lamina popria.
   - Muscularis mucosa (longitudinal smooth muscle).
2. ***Submucosa***
   - Submucosal plexus (Myentric's).
3. ***Muscularis externa/popria***
   - Inner circular smooth muscle.
   - Meissner plexus (Auerbach's)
   - Outer longitudinal muscle.
4. ***Adventitia***

Physiology of esophagus

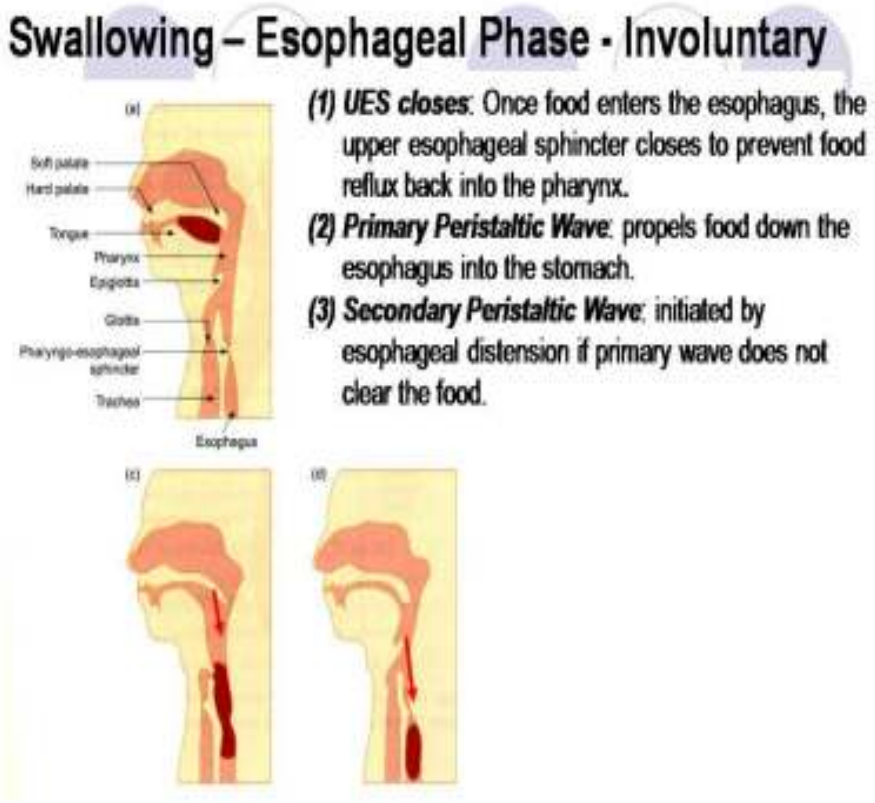

Figure (3): Physiology of esophagus.

The main functions of the esophagus are:

1. To conduct food and fluids from the pharynx to the stomach.
2. To prevent passive diffusion from the pharynx to the stomach.
3. To prevent passive diffusion of substances from the food into the blood.
4. To prevent reflux of gastric contents into the esophagus.

- These functions require motor activity coordinated with swallowing, namely a wave of peristaltic contraction, relaxation of the LES in anticipation of the peristaltic wave, and closure of the LES after the swallowing reflex. The mechanisms governing this motor function are complex, involving both extrinsic and intrinsic innervation, humoral regulation and properties of the muscle wall itself (Figure 3).

- The control of the lower esophageal sphincter (LES) is critical to esophageal function. Maintenance of sphincter tone is necessary to prevent reflux of gastric contents, which are under positive pressure relative to the esophagus.

- During deglutition, both active inhibition of the muscularis propria muscle fibers by inhibitory nonadrenergic/ noncholinergic neurons and cessation of tonix excitation by cholinergeic neurons enable the LES to relax.

- Many chemical agents (e.g. gastrin, acetylcholine, serotonin, prostaglandin, motilin, substance P, histamine and pancreatic polypeptide) decrease the tone. However, their precise roles in normal esophageal function remain unclear.

(Gryska and Vernon, 2005)

Chapter 2

PATHOLOGY AND COMPLICATIONS OF GERD

Aetiology

Normal competence of the gastro-esophageal junction is maintained by the LES. This is influenced by both its physiological function and its anatomical location relative to the diaphragm and the esophageal hiatus. In normal circumstances, the LES transiently relaxes as a coordinated part of swallowing, as a means of allowing vomiting to occur and in response to stretching of the gastric fundus, particularly after a meal to allow swallowed air to be vented. Most episodes of physiological reflux occur during postprandial transient lower esophageal sphincter relaxations (TLESRs) ***(Moraes-Filho et al., 2002).***

In the early stages of GERD, most pathological reflux occurs as a result of an increased number of TLESRs rather than a persistent fall in overall sphincter pressure. In more severe GERD, LES pressure tends to be generally low, and this loss of sphincter function seems to be made worse if there is loss of an adequate length of intra-abdominal esophagus ***(Hoppo et al., 2010).***

The absence of an intra-abdominal length of esophagus results in a sliding hiatus hernia. The normal condensation of

peritoneal fascia over the lower esophagus (the phreno-esophageal ligament) is weak, and the crural opening widens allowing the upper stomach to slide up through the hiatus.

The loss of the normal anatomical configuration exacerbates reflux, although sliding hiatus hernia alone should not be viewed as the cause of reflux. Sliding hiatus hernia is associated with GERD and may make it worse but, as long as the LES remains competent, pathological GERD does not occur. Many GERD sufferers do not have a hernia, and many of those with a hernia do not have GERD. It should be noted that rolling or para-esophageal hiatus hernia is a quite different and potentially dangerous condition ***(Hirano and Richter, 2007).***

A proportion of patients have a rolling hernia and symptomatic GERD or a mixed hernia with both sliding and rolling components. Reflux esophagitis that is visible endoscopically is a complication of GERD and occurs in a minority of sufferers overall, but in around 40% of patients referred to hospital.

In western societies, GERD is the most common condition affecting the upper gastrointestinal tract. This is partly due to the declining incidence of peptic ulcer as the incidence of infection with Helicobacter pylori has reduced as a result of improved socioeconomic conditions along with a rising incidence of GERD in the last 20–30 years. The cause of the increase is unclear, but may be due in part to increasing obesity. The strong association

between GERD, obesity and the parallel rise in the incidence of adenocarcinoma of the esophagus represents a major health challenge for most western countries.

Clinical features

The classical triad of symptoms is retrosternal burning pain (heartburn), epigastric pain (sometimes radiating through to the back) and regurgitation. Most patients do not experience all three. Symptoms are often provoked by food, particularly those that delay gastric emptying (e.g. fats, spicy foods). As the condition becomes more severe, gastric juice may reflux to the mouth and produce an unpleasant taste often described as 'acid' or 'bitter'. Heartburn and regurgitation can be brought on by stooping or exercise. A proportion of patients have odynophagia with hot beverages, citrus drinks or alcohol. Patients with nocturnal reflux and those who reflux food to the mouth nearly always have severe GERD. Some patients present with less typical symptoms such as angina-like chest pain, pulmonary or laryngeal symptoms ***(Pandolfino et al., 2003).***

Dysphagia is usually a sign that a stricture has occurred, but may be caused by an associated motility disorder.

Because GERD is such a common disorder, it should always be the first thought when a patient presents with esophageal symptoms that are unusual or that defy diagnosis after a series of investigations.