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Therapeutic flexible endoscopy replacing surgery: Part 2—Gastroesophageal reflux disease and its complications☆

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ABSTRACT

Gastroesophageal reflux disease (GERD) is a complex entity whereby gastric contents reflux into the esophagus owing to either a lack of a natural barrier between the stomach and esophagus or a dysfunction of foregut motility. In many cases, elements of both are present. GERD presents with either typical or atypical symptoms and can lead to metaplasia of the esophageal epithelium, a precancerous condition called Barrett's esophagus (BE). GERD is a chronic disease, and like other chronic diseases, it requires a thorough evaluation to deduce the exact etiology and also necessitates a dynamic and evolving spectrum of therapy. Until relatively recently, the spectrum of treatment for chronic GERD and BE included lifestyle modification, pharmacotherapy, and laparoscopic fundoplication. Considering the effect of chronic GERD on health and quality of life, and with further progress in understanding the disease process, newer and less invasive techniques for treatment have emerged and developed over the past decade. The 2 current FDA-approved therapies for the endolumenal treatment of GERD are transoral incisionless fundoplication with the Esophyx2 device and the Stretta system. If chronic GERD has lead to BE, endolumenal therapies include radiofrequency ablation with the HALO system or endoscopic resection of the metaplastic changes. Data regarding the outcomes of each of these procedures are accumulating rapidly; however, further data and evaluation are necessary to determine the most appropriate place for these procedures in the spectrum of therapy for chronic GERD and BE.

1. Introduction

Gastroesophageal reflux disease (GERD) is a complex entity whereby gastric contents reflux into the esophagus owing to either a lack of a natural barrier between the stomach and esophagus or a dysfunction of foregut motility. In many patients, both elements are present. Proper function is dependent upon the anatomy and physiology of the esophagus, stomach, and diaphragm, working in concert to promote antegrade flow and prevent retrograde flow of the intraluminal contents. The anatomy and interactions of these organs form the basis for understanding GERD and lead us toward a better understanding of its treatment. This article briefly reviews the pathophysiology of GERD and describes the role endolumenal therapies play in treating this disease and one of its complications—Barrett's esophagus (BE).

2. Manifestations of chronic GERD

2.1. Typical symptoms

The symptoms of GERD can be classified as typical and atypical. For a list of the typical symptoms, please refer to Table 1.

2.2. Atypical symptoms

Atypical symptoms, which are classically the extraintestinal manifestations of GERD and include laryngopharyngeal reflux symptoms, are not as straightforward in their contribution to an accurate diagnosis. Often, the pathway to diagnosis is made easier when typical symptoms are present in addition, but it can be quite circuitous when they are the primary manifestations. Table 2 lists the most common atypical symptoms.

2.3. Barrett's esophagus

BE is another manifestation of GERD and is considered absolute proof of an ineffective antireflux barrier [2]. It is the condition in which metaplastic columnar epithelium replaces the stratified squamous epithelium that normally lines the distal esophagus. The diagnosis strictly mandates the presence of intestinal

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Table 1
Typical symptoms of GERD. (Adapted from Bhanot and Soper [1].)

Typical symptoms
Heartburn
Regurgitation
Water brash
Dysphagia
Odynophagia

metaplasia, because it is the only columnar epithelium that has a clear predisposition for esophageal adenocarcinoma [3]. BE is caused by chronic GERD and may progress from metaplasia through low-grade dysplasia (LGD), high-grade dysplasia (HGD), and ultimately to adenocarcinoma.

2.4. GERD as a chronic disease

GERD is more commonly thought of as a chronic disease. The signs and symptoms generally develop over an extended period of time, rather than acutely. Surgical intervention is based upon the reconstruction of the body's natural antireflux barrier, which, in patients suffering from GERD, is very often due to an anatomical derangement [4]. Carrying this principle forward, as with other chronic diseases, GERD should then be thought of as a disease that exists on a spectrum, thereby requiring a spectrum of therapy. Just as coronary artery disease may necessitate medication, percutaneous intervention, or surgery, GERD may require similarly planned interventions, whether this is pharmacotherapy, endolumenal therapy, or surgery.

3. Etiologies of Chronic GERD

The exact etiologies that result in chronic GERD provide guidance as to the best antireflux treatment for each patient. For the purposes of this article, we focus on the etiologies as they relate to endolumenal treatment, rather than medication or surgery. The principle causes of reflux are hiatal or paraesophageal hernia or a poorly functioning or weak lower esophageal sphincter (LES) [5]. It is these findings that constitute the basis and strategy for antireflux surgery, and thus, endolumenal therapy. Other diagnoses contributing to chronic GERD are global esophageal and gastric dysmotility [2]. These 2 diagnoses alone do not often lead to endolumenal or surgical intervention, but they certainly play a role in determining the best course of endolumenal therapy or surgical treatment for patients with chronic GERD. In our discussion, they have only been considered when found in conjunction with a hiatal hernia or an abnormal LES or both.

Table 2
Atypical symptoms of GERD. (Adapted from Bhanot and Soper [1].)

Atypical symptoms
Asthma
Aspiration pneumonia
Chronic bronchitis
Hoarseness
Laryngitis
Chronic sore throat
Chronic cough
Frequent swallowing
Mouth sores
Chest pain
Dental erosion

3.1. Diagnosis of a hiatal hernia

The diagnosis and measurement of a hiatal hernia is perhaps the single-most important aspect in selecting patients for endolumenal treatment of chronic GERD. The anatomical derangement that occurs disrupts the antireflux barrier and often leads to chronic GERD. The modalities for measuring a hiatal hernia primarily consist of an upper gastrointestinal contrast swallow study (UGI), esophagogastroduodenoscopy (EGD), and to a lesser extent, manometry. We discuss the first 2 modalities in the following sections.

A UGI study is a dynamic study that is best evaluated in real time. A variety of maneuvers are used including abdominal pressure and Trendelenburg position to induce reflux. If possible, the amount of gastric cardia or fundus herniating through the diaphragm is measured longitudinally from the gastroesophageal junction to the level of the diaphragm. A measurement should also be taken transversely to account for the diaphragmatic hiatus. Usually, the larger of the 2 is used as the measurement for planning an endolumenal approach. As this is a dynamic study, a small sliding-type hiatal hernia may not be visualized if it is in a reduced state during the study [6].

EGD measurements are variable, and unless the hernia is moderate to large, the size is often underestimated or missed entirely. This is especially true if the diaphragmatic fascia remains intact, despite a hernia in the underlying muscle. Measurements should be taken as the endoscope first enters the distal third of the esophagus, before gastric insufflation, as this often reduces small hiatal hernias. The size is measured from the Z line to the area of diaphragmatic narrowing, sometimes called the diaphragmatic pinch; this is a longitudinal measurement [6].

The next measurement occurs in the retroflexed view. The stomach is completely insufflated, with no rugal folds remaining, so that it completely “layers out” on the inferior surface of the diaphragm. This gives the most accurate view of the hiatus. Moderate to large defects often do not hold the insufflation, but this technique still allows for a fairly accurate measurement. Assuming that most upper endoscopes are almost 1 cm in diameter, this provides a relative scale in which the endoscopist can calculate the transverse measurement. Again, the larger of the 2 measurements should be taken, as this is the greatest amount of potential herniation possible. This view facilitates appropriate evaluation of the Hill grade, which is used by many endoscopists to measure a hiatal hernia [7] (Figure 1).

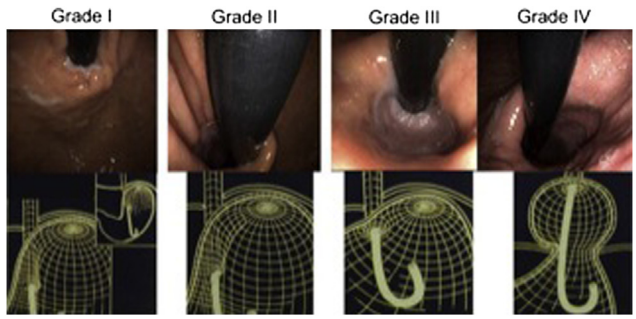

Fig. 1. Evaluation of a hiatal hernia using the Hill classification as given by Kahrilas and Pandolfino [7]. Grade I—the GEJ flap valve has a muscular ridge of tissue, which is closely approximated to the retroflexed endoscope. Grade II—the GEJ flap valve has a ridge of tissue that is less well defined than in Grade I, and it may open with respiration but then quickly closes. Grade III—the ridge of tissue is effaced, there is often failure of the GEJ to close around the endoscope, and the hiatus is patulous. Grade IV—there is no muscular ridge at all. The hiatus is continuously wide open, and a hiatus hernia is always present. (Color version of the figure is available online.)