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In Brief

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Gastroesophageal reflux disease (GERD) is defined by "symptoms or complications resulting from the reflux of gastric contents into the esophagus or beyond, into the oral cavity (including larynx) or lung."[1,2] GERD symptoms include heartburn, regurgitation, dysphagia, wheezing, and sleep disturbances, among a spectrum of other symptoms. Atypical symptoms of GERD include asthma, chronic cough, and non-cardiac chest pain. Of the atypical presentations, asthma seems to be the most highly associated pulmonary manifestation of GERD.[3]. In fact, an estimated 50%-80% of asthmatics in the United States are also suspected to have GERD.[3] It is similarly possible to have GERD without objective symptoms. GERD can range in severity from mild, nonerosive reflux disease to erosive esophagitis. The mucosal damage seen in erosive esophagitis and Barrett's esophagus, is a potential hallmark of GERD, however, a majority of symptomatic patients do not have mucosal erosion, as is the case in non-erosive reflux disease.[4]

GERD pathophysiology is multifactorial and complex. The symptoms or mucosal damage apparent in GERD, or both, are thought to result from incompetent antireflux mechanisms, imbalances in acid production, and structural abnormalities in the stomach, esophagus, or surrounding anatomy. Abnormal physiologic relationships between the stomach and esophagus, abnormal acid production, delayed gastric emptying, and irregular esophageal motor function may each contribute to the development of GERD and hint at a multifactorial pathogenesis. Ingestion of caustic agents, disorders in mucosal integrity, hypersensitivity, and genetics may also have a role in the development and progression of GERD.

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A GERD diagnosis is made if reflux symptoms are present more than twice a week for a few weeks, and reflux symptoms must be present repeatedly over time in order to classify as GERD. GERD is often associated with other disorders and diseases, such as gastroparesis, eosinophilic esophagitis, and esophageal dysmotility, and these conditions must be taken into account when performing esophageal testing, classification, and imaging. The standard first-line treatment for GERD is medical therapy. Due to the well-documented harms of long-term antireflux pharmacologic therapy, endoscopic therapy, and antireflux surgery are increasingly beneficial options for patients with GERD symptoms. Surgical candidacy involves several factors, including staging of the disease, esophageal testing, and patient symptoms.

Objective measures used in the diagnosis of GERD include esophagogastroduodenoscopy (EGD), pH testing, esophageal manometry, and x-ray of the upper gastrointestinal system. EGD visualizes the mucosa of the esophagus and allows for evaluation of the degree of reflux severity.

Due to the diverse presentation of GERD, including no mucosal damage, a normal endoscopy does not rule out GERD. Despite the low sensitivity for the diagnosis of GERD, EGD is the most appropriate examination to evaluate for complications of GERD such as severe mucosal damage, Barrett's esophagus, esophageal strictures, and the presence of hiatal hernias.[5]

Biopsy specimens obtained via EGD are an important component of the evaluation for patients with GERD, due to the direct visualization of histologic change. New, noninvasive technology utilizing saliva and sputum samples have emerged to aid in the diagnosis of GERD.[6] One such test focuses on pepsin, which is ordinarily found only in the stomach. The presence of pepsin in the saliva indicates that stomach contents may have refluxed and traveled upward.

The main endoscopic therapies currently available and approved by the US Food and Drug Administration for GERD are transoral incisionless fundoplication (TIF) (EsophyX; EndoGastric Solutions, Redmond, WA), Medigus Ultrasonic Surgical Endostapler (MUSE, Medigus, Omer, Israel), and radiofrequency ablation (Stretta; Mederi Therapeutics, Norwalk, CT).

TIF aims to reconstruct the LES anatomy and help restore the LES function as a reflux barrier. The Stretta system utilizes radiofrequency ablation to deliver low-power, low-temperature energy to the LES, and gastric cardia, which leads to tissue remodeling in the affected areas. These treatments can result in improved functioning of the LES and a decrease in LES relaxation events that exacerbate reflux symptoms.

Ideal patients for these endoscopic reflux therapies are those with minimal anatomic change at the gastroesophageal junction and moderate-to-severe GERD symptoms that are responsive to proton pump inhibitor therapy. Additionally, patients must be candidates for general anesthesia. These options have a demonstrable safety track record and are viable therapeutic option for patients with chronic GERD unwilling to proceed with surgery or to take life-long medications.

Surgical treatment of reflux is indicated in conditions of medical management failure, patients experiencing complications of medical therapy, atypical symptoms, patients with large hiatal hernias, or patients who wish to discontinue medical therapy. The primary goal of antireflux surgery (ARS) is to re-establish a mechanical barrier to acid reflux. Existing literature suggests that individuals who demonstrate typical GERD symptoms that are exacerbated in the supine position have a better prognosis compared to those with extraesophageal symptoms.

A Nissen total fundoplication is generally performed in the presence of normal esophageal motility. If preoperative manometry is indicative of an esophageal motility disorder, a partial fundoplication is preferred.[7] In general, patients carrying diagnosis of high-grade esophageal dysplasia or cancer are not candidates for ARS. If esophageal pathology is able to be successfully treated with endoscopic ablation or resection, ARS could then be considered. A Nissen fundoplication is a 360° wrap of the fundus around the posterior aspect of the esophagus. Two partial fundoplication procedures include the toupet and Dor fundoplication techniques. The toupet fundoplication is similar to a Nissen as it is also a posterior wrap. This partial fundoplication is positioned between 180° and 270°. A Dor fundoplication is a partial anterior wrap that does not require disruption of the posterior attachments to the esophagus.

Most patients undergoing primary ARS report mild, transient dysphagia that resolves in 2-4 weeks. The most common etiology of early postoperative dysphagia is tissue edema involving the wrap at the esophageal hiatus. Less frequently, dysphagia is due to esophageal or gastric

hematoma formation adjacent to sutures. In either case, additional intervention is typically not indicated as patients are usually able to tolerate liquids with significant improvement over the next few days. Severe dysphagia with inability to tolerate liquids should be studied with an upper gastrointestinal series in order to rule-out anatomic abnormalities, including an early hiatal hernia or obstruction at the gastroesophageal junction. In many cases, prolonged dysphagia is due to an overly tight wrap which can be treated with endoscopic dilation. If slippage or re-herniation has occurred, reoperation is necessary.

The LINX Reflux Management System (Torax Medical, Inc., Shoreview, MN) is a device developed to address the therapy gap between patients with good symptomatic control on medication and patients who require a Nissen fundoplication.[8] The LINX device involves magnetic sphincter augmentation (MSA) of the LES. The LINX device is a ring of magnets that are connected by titanium wires that is placed laparoscopically around the LES. It reinforces the intrinsic sphincter of the LES but does not cause the supraphysiologic valve pressures seen with Nissen fundoplication.[9] Currently, the only absolute contraindication to MSA placement is allergy to titanium, stainless steel, or ferrous material, which can be tested by giving the patient a trial device to wear 3-4 days prior to surgery.[9]

MSA outcomes are similar to those observed with Nissen fundoplication.[9-11] The most common postoperative complication is dysphagia, which often improves over the first 6 months. MSA patients often require more endoscopic dilations than patients who have undergone Nissen fundoplication.[12]