Preoperative Workup of GERD

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Amy Cha and Victoria M. Gershuni

Definition of Gastroesophageal Reflux Disease

The Montreal consensus definition of gastroesophageal reflux disease (GERD) is a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications. Manifestations of GERD can be classified as esophageal or extraesophageal syndromes, with or without evidence of esophageal mucosal injury. This classification allows symptoms to define the disease but permits further characterization if mucosal injury is found on further study. Mucosal injury from GERD can progress to the well-recognized complications of esophagitis, stricture, intestinal metaplasia or Barrett's esophagus (BE), and adenocarcinoma [1].

Anatomy and Physiology of the Gastroesophageal Junction

The anatomical antireflux barrier at the gastroesophageal junction (GEJ) is created by the coordinated action of the lower esophageal sphincter (LES), diaphragmatic crura, segment of intra-abdominal esophagus, the angle of His, and peristaltic action propelling acid forward. At rest, the LES remains tonically contracted (10–30 mmHg) to create a zone of increased pressure compared to intraluminal gastric pressure (5 mmHg). The LES relaxes upon swallowing in advance of the peristaltic wave [2]. The crura of the diaphragm respond to changes in intra-abdominal pressure and can amplify LES contraction. Other components of acid clearance include saliva, gravity, and esophageal motility. Gastric dysmotility and delayed gastric emptying can likewise predispose to GERD. The symptoms and/or mucosal injury in GERD are attributed to increased esophageal exposure to gastric acid, often due

A. Cha (✉) · V. M. Gershuni
Department of Surgery, Hospital of the University of Pennsylvania, Philadelphia, PA, USA
e-mail: amycha@pennmedicine.upenn.edu

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to changes in the function of the LES. LES deterioration over time leads to decreased barrier of the esophagus from acid and bile exposure [2]. The disease includes both esophageal and extraesophageal syndromes and can progress from symptoms without esophageal injury (nonerosive) to complicated GERD. Up to 70% of patients who present with symptoms of GERD in the primary care setting do not have evidence of tissue injury [3].

Clinical Presentation

The Montreal classification divides GERD manifestations into esophageal and extraesophageal syndromes.

The esophageal syndromes include the typical reflux syndrome defined by the presence of troublesome heartburn and/or regurgitation. Heartburn and regurgitation can sometimes be accompanied by other symptoms such as epigastric pain or sleep disturbance due to nighttime heartburn. GERD can also cause episodes of chest pain that resemble ischemic cardiac chest pain, which is called reflux chest pain syndrome and considered an esophageal syndrome as well. Persistent or progressive dysphagia is a symptom of an esophageal syndrome with mucosal injury, as it is a warning symptom or alarm symptom for stricture or cancer of the esophagus which warrants investigation.

The extraesophageal syndromes include reflux-related cough, laryngitis, asthma syndromes, and associations with dental erosion, sinusitis, aspiration, pulmonary fibrosis, pharyngitis, hoarseness, globus sensation, or recurrent otitis media. It is important to remember that an association of these syndromes and GERD exists, but it is rare for extraesophageal syndromes to occur in isolation without concomitant manifestations of typical esophageal syndrome. These syndromes are usually multifactorial with GERD as only one of several potential triggers, and data showing a benefit of reflux treatments on these syndromes are weak.

Due to the wide range of presentation in GERD patients, it is important that other etiologies such as cardiopulmonary disease, other foregut disease, and motility disorders also be ruled out prior to surgical treatment of GERD. Although GERD is a common entity, the signs and symptoms are nonspecific. The presentation is heterogeneous and dependent on the patient's perception of symptoms, which can overlap the symptoms of other upper gastrointestinal disorders like achalasia, gastroparesis, and functional dyspepsia [3].

Epidemiology

Recent epidemiologic studies have reported the prevalence of GERD with at least one episode of heartburn and/or regurgitation weekly to be as high as 30% in Western countries, which is up from 20% in 2005 [4]. Evidence suggests disease burden may be increasing worldwide, even as the range of geographical areas studied has expanded considerably. Studies have demonstrated that as many as 7% of

Americans have daily episodes of heartburn and 42% of Americans suffer from at least one episode per month [5]. These data suggest that GERD is likely to remain a common reason for physician office visits, both primary and referral, around the world.

Treatment Options

The primary treatment options for GERD include medical therapy with proton pump inhibitors and/or H2 receptor antagonists or laparoscopic surgical reconstruction by fundoplication. Acid-suppressive therapies may reduce or eliminate the symptom of heartburn by increasing the pH of gastric secretions, but they do not address the anatomically defective antireflux barrier and esophageal exposure to any weakly acidic gastric contents that may continue to reflux in some patients. Despite adequate acid suppression, 32% of patients in randomized studies and 45% in observational studies were found to have persistent symptoms [5]. However, not all patients who fail to respond to medical therapy have GERD. It is important to study these patients to distinguish those with persistent symptoms due to GERD vs non-GERD causes. This is emphasized by the finding that nearly 30% of patients who present with a chief complaint of GERD do not end up having abnormal distal esophageal acid exposure and, thus, would not benefit from an antireflux operation [5].

When surgical treatment is considered, objective esophageal testing is imperative to document the presence of GERD. While symptoms are indicative of GERD, they are unreliable in establishing the diagnosis without additional esophageal function tests. The goal of preoperative testing is to establish the presence of abnormal esophageal acid exposure and correlate reflux events with symptoms. Laparoscopic fundoplication is highly effective in patients with documented abnormal esophageal acid exposure and typical GERD symptoms of heartburn and regurgitation. Proper patient selection by objective esophageal testing is critical to achieve excellent surgical outcomes.

Esophageal Testing

Upper endoscopy is important to assess for esophageal mucosal injury as a manifestation of GERD, namely, esophagitis and BE. The Los Angeles (LA) classification was introduced into practice to objectively describe the severity of esophagitis. LA grade A and mild B esophagitis can have wide inter-observer variability and be diagnostically nonspecific, so the Esophageal Diagnostic Advisory Panel recommends these patients require pH testing to document the presence of GERD. Patients with LA grade C or D esophagitis do not require pH testing, as long as achalasia and pill esophagitis have been excluded. BE is defined as columnar-lined segment of esophagus visible on endoscopy in conjunction with pathologic findings of intestinal metaplasia with presence of goblet cells; it represents an advanced form of

GERD. The Prague classification is an objective description for the endoscopic appearance of BE, but there is inter-observer variability particularly in short-segment lesions <1 cm, and only 50% of short-segment BE lesions were confirmed histologically. The Esophageal Diagnostic Advisory Panel makes a distinction between short-segment BE (<3 cm) requiring pH testing to document the presence of GERD before antireflux surgery [5]. Patients with long-segment BE (≥3 cm) do not require pH testing prior to antireflux surgery [5]. Endoscopic findings of BE or a stricture are the most sensitive indicators of short esophagus that will require Collis gastroplasty. Finally, upper endoscopy is useful in eliminating errors in pH testing such as misplaced pH probe or capsule, especially important in the diagnosis of patients who may have nonerosive reflux disease. This distinct subgroup of GERD patients has no mucosal injury on endoscopy but can be further subcategorized with careful pH testing. Patients with abnormal pH test but no mucosal injury are commonly encountered, requiring additional testing to document pathological GERD. Particular attention should be paid to obtaining thorough surgical history and history of other gastrointestinal symptoms in these patients to consider whether antireflux surgery may worsen the non-GERD symptoms. Patients with no mucosal injury and a normal pH test but with symptoms and reflux events that temporally correlate may have acid hypersensitivity. Patients with no mucosal injury with a normal pH test and no symptom correlation with reflux events by definition must have a non-GERD etiology for their symptoms. These two groups of patients with no mucosal injury and negative pH test might not be adequately treated with antireflux surgery, and in these patients surgery should be avoided [5].

All patients who are considered for antireflux surgery require barium esophagram. A barium esophagram provides the surgeon with useful anatomic and functional information. It will reveal the presence and size of hiatal hernia, diverticulum, stricture, esophageal length, and even gastroesophageal reflux events with provocative maneuvers. It is not, however, a reliable predictor of short esophagus as the endoscopic findings of stricture or BE are [5]. Barium esophagram can further differentiate between a type I sliding hiatal hernia and paraesophageal hernias (types II, III, IV). Paraesophageal hernias may be associated with increasing GERD symptoms and gastric volvulus may result in catastrophic complications. The workup may require barium esophagram, upper endoscopy, and manometry because an antireflux procedure is performed as an intergral part of the procedure. pH testing is not required in these patients.

Ambulatory pH testing is the gold standard for determining presence of pathological GERD. It is required for all patients being considered for antireflux surgery [6] with very few exceptions: type III paraesophageal hernia which must be repaired regardless of GERD, long-segment BE (≥3 cm), or LA grade C or D esophagitis if achalasia and pill esophagitis have been excluded. The Esophageal Diagnostic Advisory Panel consensus was that pH testing off acid suppression at least 7 days [6] should be performed in all patients with nonerosive GERD, those with LA grade A or mild B esophagitis, and those with short-segment BE (<3 cm). pH testing off acid suppression is an important measurement in the management of patients with GERD not responding to PPI therapy as well; those who have a normal pH study may then stop PPI therapy which is of no benefit to them [6]. pH testing can be

performed by transnasal catheter for 24 h or wireless pH capsule for 48 h. An abnormal 24-h pH test in a PPI-dependent patient with typical symptoms predicts successful outcomes with antireflux surgery, whereas those with typical symptoms without abnormal pH test are less likely to have successful surgical outcomes. 48-h pH testing can increase detection accuracy and sensitivity for abnormal esophageal acid exposure by as much as 22% [5]. Multichannel intraluminal impedance (MII)-pH is a promising tool to detect any type of reflux event regardless of acid or nonacid pH, especially in patients refractory to PPI therapy. Additional studies are needed to clarify the value of 24-h MII-pH (on acid suppression) in predicting outcomes of antireflux surgery. The Esophageal Diagnostic Advisory Panel maintains that testing off acid suppression should be used to determine if there is pathologic GERD [5]. Finally, the symptom index (SI) and symptom association probability (SAP) are the symptom association values calculated by the analysis software to evaluate the temporal association between clinical symptoms and reflux events. The SI is a measure of the strength of the association between symptoms and reflux events; ≥50% is considered positive. The SAP determines whether this relationship could have occurred by chance; >95% is statistically significant. These calculated values have only been validated for acid-related heartburn, regurgitation, and chest pain, and not nonacid by MII-pH. The values are also highly dependent on the numbers of symptoms noted by patients during the testing period [5].

Manometry should also be performed in all patients being considered for antireflux surgery to exclude achalasia or other underlying esophageal motility disorder which may have been misdiagnosed as GERD. Sixty percent of GERD patients might have defective LES on manometry, and impaired esophageal motility is associated with the severity of esophagitis as well. Now, 32-channel high-resolution manometry is easier, faster, and more accurate. Manometry can be used to assess peristaltic coordination and contractile force of the esophageal body, which can guide the surgeon in choosing the type of antireflux procedure. Patients with frequent failed or weak peristalsis might have less dysphagia with partial fundoplication, but no controlled data support the practice of tailoring the degree of fundoplication to the preoperative esophageal motility. The precise location of the LES can be measured for accurate pH capsule or catheter placement. Nevertheless, manometry is also not a reliable predictor of the short esophagus as the upper endoscopic finding of BE or stricture [5].

Gastric emptying study should be obtained selectively. Delayed gastric emptying symptoms include bloating, abdominal distension, and nausea, but these are nonspecific and overlap with the symptoms of functional dyspepsia. Even 30% of patients with functional dyspepsia will have delayed gastric emptying, and the study does not distinguish gastroparesis from functional dyspepsia. Twenty percent of patients with GERD have delayed gastric emptying which improves with fundoplication, reducing the capacity of the fundus and the radius of the proximal stomach, generating a higher intraluminal pressure and promoting the passage of food bolus. Persistent delayed gastric emptying can worsen gas bloat after antireflux surgery, but 31% of patients were found to have delayed gastric emptying preoperatively that did not predict the outcome of fundoplication [5, 7]. Currently there are

no established gastric emptying study values that predict the worsening of gas bloat postoperatively. It should be obtained selectively in patients with significant nausea, vomiting, and bloating or those with retained food on endoscopy [7].

Laryngopharyngeal reflux (LPR) symptoms may be a result of irritation of the hypopharynx by acid reflux, but other causative factors include tobacco, alcohol, allergies, postnasal drip, and chronic sinusitis to name a few. Empiric PPI therapy is usually recommended but demonstrates no therapeutic benefit in recent meta-analyses [1]. Outcomes of antireflux surgery performed for LPR symptoms are less favorable compared with those achieved in patients with typical GERD symptoms. In an attempt to measure LPR events from acid reflux, the oropharyngeal pH catheter and hypopharyngeal MII (HMII)-pH catheter have been introduced and investigated. There is a lack of data to support the use of oropharyngeal pH or HMII-pH testing for improving patient selection for antireflux surgery. For patients with LPR symptoms who undergo these tests, a positive pH test documenting pathologic acid exposure in the distal esophagus is still required to justify antireflux surgery.

Indications for Surgery

From a surgical perspective, GERD is a mechanical failure of the lower esophageal sphincter (LES), appropriate gastric emptying, and coordinated esophageal peristalsis. A single test cannot make the diagnosis alone; rather, the several diagnostic studies taken together provide a full picture of GERD to determine whether it is amenable to surgical treatment. Based on SAGES guidelines, objective evidence of esophageal reflux must be demonstrated prior to surgery. These include any mucosal break from adjacent normal-appearing esophageal mucosa in a symptomatic patient, peptic stricture in the absence of malignancy, biopsy-proven Barrett's esophagus (BE), or prolonged exposure to acidic pH as demonstrated by esophageal pH monitoring probe.

Only after successful objective identification of pathologic acid exposure should surgery be pursued in the following situations [7]:

1. Patients who have failed conservative therapy with lifestyle change and medical management as determined by inadequate symptom management, severe regurgitation despite acid suppression, or side effects secondary to acid-suppressing medications.
2. Patients who wish to pursue surgery for quality of life considerations (cost, need for lifelong medication use, etc.) despite adequate medical management.
3. Demonstration of GERD complications, including BE or peptic stricture.
4. Extra-intestinal manifestations of GERD: asthma, pulmonary fibrosis, throat clearing, aspiration, cough, etc.

References

1. Wayman J, Myers JC, Jamieson GG. Preoperative gastric emptying and patterns of reflux as predictors of outcome after laparoscopic fundoplication. Br J Surg. 2007;94:592–8.
2. Mikami DJ, Murayama KM. Physiology and Pathogenesis of Gastroesophageal Reflux Disease. Surg Clin N Am. 2015:515–25.
3. Vakil N, van Zanten SV, Kahrilas P, et al. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol. 2006;101:1900–20.
4. El-Serag HB, Sweet S, Winchester CC, Dent J. Update on the epidemiology of gastro-oesophageal reflux disease: a systematic review. Gut. 2014;63(6):871–80.
5. Jobe BA, Richter JE, Hoppo T, et al. Preoperative diagnostic workup before antireflux surgery: an evidence and experience-based consensus of the esophageal diagnostic advisory panel. J Am Coll Surg. 2013;217(4):586–97.
6. Richter JE, Pandolfino JE, Vela MF, et al. Utilization of wireless pH monitoring technologies: a summary of the proceedings from the Esophageal Diagnostic Working Group. Dis Esophagus. 2012;26:755–65.
7. Stefanidis D, Hope WW, Kohn GP, et al. Guidelines for surgical treatment of gastroesophageal reflux disease. Surg Endosc. 2010;24:2647–69.