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Medical treatment versus surgery for treatment of gastroesophageal reflux disease

Parth J. Parekh, MD[a,1], David A. Johnson, MD, MACG, FASGE, FACP[b,1,*]

[a] Department of Internal Medicine, Tulane University, New Orleans, Louisiana
[b] Department of Internal Medicine, Eastern Virginia Medical School, 885 Kempsville Rd, #114 Norfolk, Virginia, VA 23502

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ABSTRACT

Gastroesophageal reflux disease has significant and profound effect on the population at large. This increasingly prevalent disease has been shown to have adverse effect on quality of life and work productivity, interference with daily living, and much less the direct and indirect diagnostic and therapeutic costs. Effective medical and surgical therapies are available. This discussion focuses on evidence for justifying therapy—with both a risk and reward assessment of outcomes. Best practice recommendations and caveats for each pathway of therapy are discussed.

1. Introduction to the treatment of gastroesophageal reflux disease

Gastroesophageal reflux disease (GERD) is characterized by troublesome symptoms of heartburn and regurgitation and, if any, complications that are caused by the reflux of stomach contents into the esophagus [1]. This is a common condition affecting approximately 10%-20% of the population in the Western world [2], and its symptoms can negatively affect patients' health-related quality of life (QOL) and well-being, as well as productivity at work, resulting in substantial societal burden and employer costs [3].

Proton pump inhibitors (PPIs) are considered the most effective form of pharmacologic therapy for GERD. Although surgical treatment of GERD is a well-established option when done by an expert surgeon, current GERD treatment guidelines recommend the use of PPIs as the preferred method of disease management of patients with moderate or severe manifestations [4]. However, 20%-40% of patients with GERD continue to experience persistent and intense reflux symptoms while receiving standard-dose PPI therapy [5,6]. This article reviews and compares the published literature for both surgical treatment and medical treatment of GERD and highlights best practice recommendations for both approaches.

2. Goals of therapy

According to the most recent consensus definition of GERD [4], the disease is defined as a condition that develops when the reflux of stomach contents causes troublesome symptoms or complications or both. According to this definition, “troublesome” symptoms are those that adversely affect an individual's well-being. The goal of therapy is to control symptoms and prevent complications.

“Typical GERD” symptoms (such as heartburn and acid regurgitation) and their frequency to be “troublesome” have also been defined [1]. The typical symptoms, for example, heartburn and acid regurgitation, have been defined by consensus only and currently lack adequate validation [1]. The attempt of the Montreal process to define an overall sensitivity and specificity of these 2 symptoms for the diagnosis of GERD has failed, for the most part owing to the lack of a gold standard and nonhomogeneity of the trials. Therefore, the definition and diagnosis of typical GERD are based solely on expert consensus. Symptoms that may accompany typical GERD symptoms include epigastric pain, sleep disturbances, dyspepsia, dysphagia, odynophagia, nausea, vomiting, and others. “Atypical GERD” involves signs or symptoms considered to be associated with or caused by GERD such as noncardiac chest pain, chronic cough (especially nocturnal), chronic laryngitis, and asthma.

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* Corresponding author.
E-mail address: dajevms@aol.com (D.A. Johnson).

[1] Authorship contributions: Parekh PJ and Johnson DA contributed equally to this work.

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The main complications of GERD can include reflux esophagitis, the development of strictures, Barrett esophagus, and esophageal adenocarcinoma. In rare cases, esophagitis may also lead to clinically significant bleeding. Despite its potentially serious sequelae, GERD usually presents as a relatively benign condition. Most patients with GERD experience nonprogressive disease (ie, the progression from nonerosive to erosive disease, to severe erosive disease, to Barrett esophagus, and ultimately esophageal adenocarcinoma or other complications), and thus GERD has been traditionally viewed as a nonprogressive disease without significant associated mortality.

3. New concepts of pathophysiology

There are several underlying mechanisms working individually or in concert contributing to the pathogenesis of GERD. If envisioned to be a single plumbing circuit, as described by Stein and Demeester [7], the esophagus acts as an antegrade pump, the lower esophageal sphincter (LES) as a valve, and the stomach as a reservoir. Abnormalities can stem from any or all components of the system and ultimately contribute to the pathogenesis of GERD and its sequelae. The effect of esophageal dysfunction [8] and resultant damage to pre-epithelial [8,9], epithelial [8], and post-epithelial [8] defense mechanisms; LES dysfunction [10]; abnormal LES pressures [11-15]; and structural abnormalities (eg, hiatal hernia) [16-18] has been well demonstrated in the pathogenesis of GERD. Emerging data have shifted focus on to the role of transient LES relaxations (TLESR) and the acid pocket as a potential underlying mechanism in the pathogenesis of GERD.

3.1. Transient esophageal sphincter relaxation

The most common mechanism implicated in the pathogenesis of GERD is TLESR. Under normal circumstances, the LES relaxes following a swallow or subsequent to primary or secondary to peristalsis to allow for passage of a bolus. In the face of a normal LES pressure, TLESR occurs spontaneously independent of pharyngeal stimulation or esophageal peristalsis [19]. There is an ongoing debate as to whether an increase in duration [20] vs an increased frequency [21,22] of LES relaxation is responsible for the pathogenesis of GERD. Other studies indicate that neither the frequency nor the duration of LES relaxation is the culprit, but rather dysfunction in the motor responses that normally promote esophageal clearance of refluxate [23-25].

3.2. Acid pocket

Intragastric pH has been demonstrated to be highest after a meal, owing to the buffering effect of food [26]. The fact that most symptoms related to GERD occur during the postprandial period [27] led many investigators to further investigate this paradox. In 2001, Fletcher et al [28] detected an area at the gastroesophageal junction of highly acidic, unbuffered gastric content, which he termed the “acid pocket”. Subsequent studies have demonstrated the location and the size of the acid pocket to differ in patients with GERD as compared with the healthy cohorts [29-31]. In patients with GERD, the “acid pocket” acts as a reservoir and is prone to upward migration of the proximal margin onto the esophageal mucosa, which may be a factor contributing to the mucosal pathology [32]. In addition, the acid pocket has been demonstrated to move in conjunction with a hiatal hernia to supradiaphragmatic location increasing the propensity for GERD symptoms by mechanisms previously discussed [29,33,34].

4. Medical therapy

Medical intervention and lifestyle modifications, if applicable, are considered the first-line therapy for patients with GERD. This section focuses on the available medical therapies and the recommended lifestyle modifications in the treatment of GERD. Figure 1 outlines an algorithmic approach to the medical treatment of GERD.

4.1. Lifestyle modifications

Several lifestyle and dietary modifications are commonly recommended to patients in the clinical realm; however, for lifestyle and dietary modifications, the evidence for efficacy of these recommendations is mostly anecdotal. The general consensus appears to be that of the lifestyle and dietary modifications only weight loss, elevation of the head of the bed, and avoidance of postprandial recumbency have been shown to somewhat consistently improve esophageal pHmetry or GERD symptomatology or both [35-38]. Jacobson et al [35] found a dose-response relationship across all categories of body-mass index (BMI) in women, noting that an increase of ≥ 3.5 in BMI increased the risk of GERD symptoms by more than a factor of 2 and conversely a decrease in BMI of ≥ 3.5 resulted in nearly a 40% reduction of symptoms compared with women whose BMI remain unchanged (odds ratio = 0.64, 95% CI: 0.42-0.97). The current American College of Gastroenterology (ACG) guidelines recommend weight loss for patients with a BMI > 25 or patients with recent weight gain [4]. To date, 3 trials have demonstrated improvement in esophageal pH values and GERD symptoms with head of bed elevation [39-41], which led the ACG to recommend the use of head of bed elevation for patients experiencing nocturnal GERD [4]. Avoidance of late evening meals has been demonstrated to improve nocturnal gastric acidity [42,43] and thus the ACG recommends avoiding eating meals particularly with high-fat content within 3 hours of reclining [4].

4.2. Acid-suppressive therapy

Histamine type 2 receptor antagonists (H2RAs) are currently the first-line agents for patients with mild to moderate GERD. H2RAs act by competitively inhibiting histamine from binding to H2 receptors found on parietal cells, thus reducing acid production. In addition, they act to reduce pepsin and volume of gastric acid [44]. There are currently 4 available H2RAs, 3 of which are available over the counter. As a class, the differences in pharmacokinetics and pharmacodynamics are negligible [45]. However, recent studies have suggested clinical superiority of nizatadine compared with other H2RAs despite there being no difference in the level of acid suppression among H2RAs as a class owing to its unique ability to inhibit the rate of TLESR and acid reflux during those episodes not demonstrated [46].

The advent of PPIs in the 1980s revolutionized the approach to GERD management [37], emerging to become the first-line therapy for patients with moderate to severe GERD [47]. PPIs irreversibly antagonize the hydrogen-potassium-adenosine triphosphatase enzyme (ie, the gastric proton pump) ultimately preventing the secretion of hydrogen ions into the gastric lumen [48]. There are currently 6 available PPIs, 3 of which are now available to patients without the need of a medical prescription by a health care provider. Recognizably the prescription strength dosages are higher (with the exception of 20 mg omeprazole); patients are more likely to adhere to a prescription regimen as compared with those who are over-the-counter consumers [49]. As a class, the differences in pharmacokinetics, pharmacodynamics, and symptomatic relief are negligible [50,51].