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Problems of Distribution and Pathophysiology of Gastroesophageal Reflux Disease

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ABSTRACT: This article provides an overview of the literature data on the problem of the prevalence of gastroesophageal reflux disease. Some pathophysiological aspects of this pathology are analyzed. The authors conclude that gastroesophageal reflux disease is a common clinical problem that affects millions of people around the world, according to some reports, up to 25–30% of the adult population is affected by it. Patients are recognized by both classic and atypical symptoms. GERD is associated with lifestyle factors, in particular obesity and tobacco smoking, which also threatens overall health.

KEYWORDS: Complications, Endoscopy, Gastroesophageal reflux disease, GERD, GERD pathophysiology, Prevalence, Reflux esophagitis, Risk factors.

INTRODUCTION

According to WHO, there is an increase in diseases associated with the pathology of the esophagus, and in particular gastroesophageal reflux disease. GERD is consistently the most commonly diagnosed gastrointestinal disease in the US and results from incompetent resistance to the retrograde movement of gastric contents into the esophagus. GERD is the most common gastrointestinal disease; its prevalence is 10% to 20% in western countries, but somewhat lower in Asia. Its clinical manifestations classically include heartburn and regurgitation, although a wide range of symptoms can also be associated with GERD, including dysphagia, odynophagia, watery rash, lump sensation, atypical chest pain, chronic cough, hoarseness, and wheezing [19, 24].

For many years, the mainstay of initial therapy for GERD has been medication. This includes lifestyle and dietary changes, such as sleeping with your head up, cutting out unhealthy foods, and not eating for 3–4 hours after going to bed. Treatment options included antacids for mild symptoms, switching to histamine–2 receptor antagonists or proton pump inhibitors (PPIs) for more severe symptoms. PPI acid suppression remains the mainstay of treatment [9, 10, 17, 20, 21], but 10% to 40% of patients with GERD do not respond to this treatment, causing refractoriness to PPI therapy. Refractory GERD is defined as less than 50% improvement in reflux symptoms, including heartburn, despite at least 12 weeks of double–dose PPI therapy [2, 6, 25]. For those patients who have contraindications to drugs or simply find it inappropriate to continue drug therapy for a long time, the next step has usually been a surgical antireflux procedure, the classic hiatal repair with Nissen fundoplication. Recently, however, a variety of endoscopic techniques have emerged to bridge the gap between medical and surgical treatment of GERD.

This need to fill the therapeutic gap arises from both the patient's and the clinician's points of view. Surgeons performing laparoscopic antireflux surgery (LARS) tend to refuse 360° Nissen fundoplication due to a higher incidence of postoperative gas/bloating and dysphagia [4, 18, 22]. While some of these problems can be partially addressed with a partial fundoplication (Dor or Toupet), these procedures are less standardized and long–term results may be less predictable.

Endoscopic treatment of GERD is currently considered appropriate for patients with early stage GERD as well as for patients with altered anatomy where standard laparoscopic surgical approaches are limited. Currently, the US Food and Drug Administration (FDA) has approved three endoscopic devices that are used to treat GERD: Stretta® for radiofrequency therapy (Restech, Houston, Texas), Esophyx–Z® for transoral non–surgical fundoplication (TIF) (EndoGastric Solutions, Redmond, Washington) and Overstitch® for endoscopic suturing (Apollo Endosurgery, Austin, TX).

To appreciate the role of endoscopic antireflux procedures, one must first understand the anatomy of the gastroesophageal junction (GEJ) and the pathological abnormalities that then lead to GERD [1, 5, 8, 12, 15, 24]. Pressure gradients between the abdominal stomach and the thoracic esophagus would favor the retrograde movement of gastric contents into the esophagus during

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most human activity, rather than the complex anti–reflux mechanism at the junction of the esophagus, stomach, and diaphragm. The lower esophageal sphincter complex is one of the two main components of this antireflux barrier and is an integral part of the esophagus and includes the lower esophageal sphincter (LES) and the esophagogastric junction. The second component is the shin diaphragm, which in normal people acts in conjunction with the LES to open during swallowing and then contract to compress the esophagus to maximize the threshold to prevent gastric reflux. The two components taken together make up the high pressure zone (HPA) found on esophageal manometry.

The LES consists of two components: the proximal part consists of the proper muscles of the distal esophagus, and the distal part consists of the ligamentous fibers of the proximal stomach [11, 16, 23]. Mechanically, the LES can be thought of as the "internal sphincter", while the calf diaphragm is the "external sphincter". The diaphragmoesophageal ligament attaches the distal esophagus to the tibia of the diaphragm, thus connecting the internal and external sphincters.

Transient or permanent dysfunction of one or both components constitutes the pathophysiology of GERD. In the early stages of GERD, the transient opening of ILI is too frequent and too often accompanied by reflux of gastric contents, not just air. Whether this is due to a neurological reflex or due to transient shortening of the lower esophageal sphincter resulting in loss of sphincter capacity is still unclear; however, antireflux procedures such as the Nissen fundoplication have been found to reduce both the frequency of these transient events and the amount of gastric acid reflux during these transient events. Transient relaxation of the lower esophageal sphincter (TRNS), one of the main mechanisms of reflux, especially diurnal reflux, is neurologically mediated [7, 14, 23]. Afferent signals for such relaxation may come from the pharynx, larynx, or stomach. The efferent pathway is located in the vagus nerve and nitric oxide is a postganglionic neurotransmitter. In advanced stages of GERD, chronic loss of length and pressure of the lower esophageal sphincter, as well as separation of the crus from the LES due to hiatal hernia, can lead to more severe reflux.

Although the PPI class is the mainstay of GERD treatment, these drugs do not reduce the incidence of reflux events, and persistent symptoms associated with ongoing reflux often require physical revision of the compromised anatomy. LARS is considered the "gold standard" procedure for repairing the antireflux barrier because it repairs both the lower leg component by hiatal hernia repair and the lower esophageal sphincter by creating a flap valve through fundoplication. However, both the level of invasiveness and the side effects of gas swelling associated with the supercompetent flap valve have prompted physicians and patients to seek alternative interventions.

In patients with a largely intact sphincter (i.e., no or very limited hiatal hernia, Hill grade 1 or 2), there is potential for an endoluminal approach to repair the lower esophageal sphincter. Conceptually, this could entail a decrease in the extensibility of the entire or only the lower part of the LES to prevent shortening and loss of LES capacity during gastric distension, an increase in resting pressure of the LES, and strengthening of the sling fibers in the CEP [3, 13].

CONCLUSION

Gastroesophageal reflux disease is a common clinical problem that affects millions of people around the world, according to some estimates, up to 25–30% of the adult population is affected by it. Patients are recognized by both classic and atypical symptoms. GERD is associated with lifestyle factors, in particular obesity and tobacco smoking, which also threatens overall health. Early recognition of symptoms is integral to preventing the complications of GERD, and advances in diagnostic and therapeutic methods have improved the ability to detect and treat these complications.

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