The Problem of Gastroesophageal Reflux Disease and Hiatal Hernia

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17.1 Introduction

The most widely accepted definition of gastroesophageal reflux disease (GERD) is a condition that develops when stomach contents cause troublesome symptoms and/or complications [1]. GERD is a common condition with multifactorial pathogenesis affecting about 10–20% of the Western population [2]. Some contributing factors play a role in the provocation of GERD, specifically, dysfunction of the esophagogastric junction (EGJ). The EGJ consists of three components: the lower esophageal sphincter (LES), the crural diaphragm, and the anatomical flap valve. It is structurally and functionally designed to act as an antireflux barrier in which the tonically contracted smooth muscle of the LES is surrounded by oblique gastric fibers that are attached to the striated muscle of the crural diaphragm by the phrenoesophageal ligament [3]. The right crus of the diaphragm forms a sling that surrounds the distal esophagus and acts as an extrinsic sphincter by augmenting the high-pressure zone of the LES. When a proximal displacement of EGJ occurs, which is likely caused by the weakening or rupture of the phrenoesophageal ligament [4], a hiatal hernia is present because a spatial dissociation of the antireflux barrier at the EGJ into the intrinsic sphincter and extrinsic sphincter crural diaphragm exists [5]. The most comprehensive classification scheme [6] recognizes four types of hiatal hernia (Fig. 17.1):

- *Type I* or sliding hernia (>95% of cases) is characterized by a widening of the muscular hiatus and circumferential laxity of the phrenoesophageal membrane, allowing a portion of the gastric cardia to herniate upward.
- *Type II* is characterized by a localized defect in the phrenoesophageal membrane, while the gastroesophageal junction remains fixed to the preaortic fascia and the median arcuate ligament.

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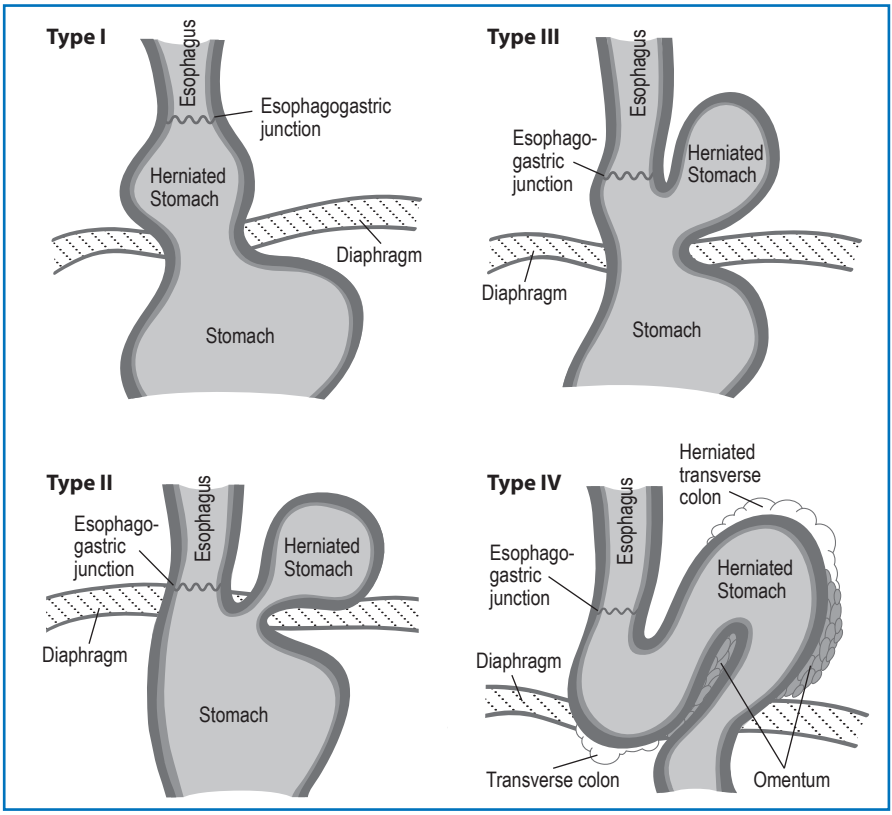

Fig. 17.1 Schematic representation of different types of hiatal hernia

- *Type III* has elements of both types I and II, with progressive enlargement of the hiatus that allows increasing amounts of fundus and LES to migrate through the hiatus.
- *Type IV* is associated with a massive defect in the phrenoesophageal membrane, allowing not only the LES and gastric fundus to herniate, but also other abdominal organs, such as the pancreas, spleen, omentum, and/or small and/or large intestine.

The presence of hiatal hernia is considered an independent risk factor for GERD [7]. It has been reported that ~75% of individuals with esophagitis and 90% of patients with Barrett's esophagus (BE) have a hiatal hernia [8, 9] Among the different types of hiatal hernia, type I (sliding) is closely associated with GERD [10]. Although sensitivity and specificity are not ideal, heartburn and regurgitation are considered typical symptoms sufficient to make a presumptive diagnosis of GERD [1], taking into account patient age and in the absence of other concerning symptoms or signs (the so-called alarm signs), which include dysphagia, odynophagia, weight loss, gastrointestinal (GI) bleeding, vomiting, family history of cancer, and epigastric mass. In such situations, upper-GI

endoscopy should be considered. However, the most sensitive and objective means for assessing reflux is ambulatory 24-h pH impedance monitoring. Classically, the diagnosis of hiatal hernia relies on its presence during endoscopy or barium swallow study [11, 12]. The diagnosis of hiatal hernia with these techniques has several limitations. One limitation is that these are snapshot techniques, and the presence of a hiatal hernia is consequently considered an all-or-nothing phenomenon. Furthermore, diagnosis of a small hiatal hernia could be challenging [13]. A recent study suggests that high-resolution manometry (HRM) has a high sensitivity and specificity (92 and 93%, respectively) for detecting a hiatal hernia [13], allowing a dynamic evaluation of EGJ with a more accurate analysis. The combination of endoscopy and HRM could reach a sensitivity of 98% in the diagnosis of hiatal hernia, making it redundant to perform an additional barium esophagogram for the preoperative diagnosis of hiatal hernia. Nowadays, intraoperative findings of hiatal hernia are considered the reference standard for its diagnosis and classification [14].

17.2 GERD and Hiatal Hernia in the Obese Population

Obesity is considered an independent risk factor for GERD. Obese patients tend to have more severe erosive esophagitis and have a greater risk of developing BE and adenocarcinoma of the esophagus compared with individuals of normal weight [15, 16]. The exact pathophysiological link between obesity and GERD has not been completely determined as yet. Among the proposed mechanisms are the greater frequency of transient low esophageal sphincter relaxation (TLESR), increased prevalence of esophageal motor disorders, diminished LES pressure, as well as presence of hiatal hernia [16, 17]. Obese patients, in fact, are more than three times as likely to have hiatal hernia compared with nonobese individuals [18]. A recent study on 142 obese patients who were candidate to primary bariatric surgery [19] reveals the presence of hiatal hernia in about 23% of cases, which were, for the most part, asymptomatic. Although ~50–70% of patients undergoing bariatric surgery have asymptomatic hiatal hernia [20], obese patients show higher symptoms score, abnormal acid exposure at 24-h ambulatory pH-metry, and lower LES pressure compared with controls [21]. Other authors confirmed these data, showing that 73% of morbidly obese patients had some abnormal 24-h pH monitoring findings and 51.7% had an elevated DeMeester score [22].

In conclusion, GERD with or without hiatal hernia can manifest in a variety of forms: from no visible esophageal injury at endoscopy, also called nonerosive reflux disease (NERD), to esophageal injury or erosive reflux disease (ERD); from metaplasia of the squamous esophageal mucosa to a columnar phenotype or BE. It is not clear whether these manifestations are part of a continuous spectrum or distinct phenotypes of GERD [23, 24]. However, this wide range of clinical

conditions increases the need of more preoperative investigations and influences the choice of procedure.

17.3 GERD and Bariatric Surgery

17.3.1 Gastric Banding

Literature data shows that ~80% of patients with gastric banding (GB) report resolution of GERD symptoms at short-term follow-up [25, 26]. GERD remission after GB was also evaluated using 24-h pH and manometry recordings at a mean follow-up of 19 months, revealing a significant decrease in total number of reflux episodes, total reflux time, and DeMeester score [27]. In patients with preoperative hiatal hernia, GB was performed with concomitant hiatal hernia repair with good results [28]. It has been hypothesized that the unfilled Lap-Band, when placed more proximally at the EGJ, could be an effective antireflux device, probably because it creates a longer intra-abdominal pressure zone or pulls the stomach more into the abdomen in the presence of a hiatal hernia [29]. Three years after GB, some authors described the resolution of GERD symptoms [30], whereas others reported new-onset GERD in 20.5% of patients [31]. Pouch formation is a crucial event in the occurrence of GERD after GB. Uncorrected band placement is considered the primary cause of early pouch dilation, whereas late pouch dilation is attributed to the inclusion of fundus above the band [29]. Another possible cause of GERD after GB is esophageal dilation. It is hypothesized that the inflated band reduces trans-stomal flow, causing reduced esophageal clearance, stasis of ingested food, and subsequently its reflux [32]. Given data on increased adverse outcomes, including GERD. in the long term with laparoscopic adjustable gastric banding (LAGB), as well as the high rate of reoperation or conversion to a more definitive bariatric surgery, the procedure is expected to be used less frequently going forward [33].

17.3.2 Sleeve Gastrectomy

Current data about the effect of sleeve gastrectomy (SG) on GERD are still controversial [34]. At short follow-up after SG, some authors reveal a discrete percentage of GERD remission [35, 36] and others a worsening of GERD, demonstrating a high prevalence of de novo erosive esophagitis [37] or a significant decrease of LES pressure with an increase of DeMeester score [38]. Data on long-term effects of SG are still scarce: Himpens et al [39] showed a biphasic pattern of GERD prevalence after sleeve gastrectomy, with 21.8% *de novo* proton pump inhibitor (PPI) use 1 year after SG, which improved to 3.1%

at 3 years but increased again to 23% at 6 years. In our recently study [40], we described at 5-year follow-up the resolution of GERD symptoms in 65% of patients with preoperative body mass index (BMI) ≤50 kg/m^2 and 44% in those with BMI >50 kg/m^2; we also reported new-onset GERD in 15 and 8%, respectively. Other studies reported a similar percentage of GERD resolution, from 53 to 60% [41, 42], with new-onset GERD seen in 11% of patients [41]. The reduction of weight and visceral adiposity and/or an accelerated gastric emptying might explain the positive effect of SG on GERD [34, 43]. Conversely, multiple factors may explain GERD worsening after SG [34]. One proposed mechanism is alteration of the angle of His, which normally acts as a valve to prevent reflux of stomach contents into the esophagus. Himpens et al. [39] showed that after 3 years, the rate of GERD after SG decreased, potentially due to restoration of the angle of His. Another possible factor is LES dysfunction. Specifically, after transection near the angle of His during gastrectomy, the sling fibers at the fundus are divided, which can subsequently decrease LES pressure. A significant decrease in LES pressure was, in fact, reported 3 months after SG [44]. The role of concomitant hiatal hernia repair during SG is still debated. At short term, some authors found an improvement of GERD symptoms [45, 46]. However, our recent study demonstrated no significant improvement in frequency-intensity of typical GERD symptoms in obese patients with GERD and hiatal hernia who underwent SG with concomitant hiatal hernia repair 16 ± 8 months earlier [36]. Recently, Samakar et al. [47] confirmed our results, demonstrating that at mean 2-year follow-up period, two thirds of symptomatic patients remained symptomatic after LSG with concomitant hiatal hernia repair and that 15.6 % of previously asymptomatic patients developed de novo reflux symptoms. We agree with the hypothesis of authors who suggest the routine repair of small hiatal hernias may contribute to LES dysfunction by disrupting the normal anatomical barriers to reflux in order to perform the repair. Potential repair breakdown may also lead to worsening of the hiatal hernia, given the dissection that takes place in order to perform the cruroplasty.

17.3.3 Roux-en-Y Gastric Bypass

Roux-en-Y gastric bypass (RYGB) is associated with a good outcome in regard to GERD. Symptom resolution or improvement has been described in several studies [48, 49]. Madalosso et al. [49] reported a significant reduction of GERD symptoms, reflux esophagitis, and DeMeester scores after 39 ± 7 months. The positive effect of RYGB on GERD may be explained by multiple factors. Creation of a small gastric pouch and separation of most of the stomach drastically reduce the acid that could promote regurgitation. Importantly, bile reflux is also eliminated due to biliary diversion. In fact, in patients undergoing surgery for morbid obesity, RYGB is the procedure of choice for patients with concomitant severe GERD.

A recent variation on RYGB is the omega-loop gastric bypass, also known as the mini-gastric bypass or one-anastomosis gastric bypass. There have been concerns about the proximity of the biliary flow to the gastric tube in this procedure compared with RYGB and the subsequent potential for both biliary reflux and esophagitis. However, a recent study using HRM and 24-h pH impedance monitoring performed both before and 1 year after omega-loop gastric bypass demonstrated that this procedure did not cause *de novo* gastroesophageal reflux or esophagitis [50]. Other data on the risk of developing de novo GERD after this particular procedure is needed, especially as there is a lack of studies of this procedure in obese patients with GERD.

In our opinion, in patients with a moderate to large hiatal hernia and/or severe esophagitis and/or BE, RYGB with or without hiatal hernia repair should be preferred.

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