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REVIEW ARTICLE

Gastroesophageal Reflux Disease and Sleeve Gastrectomy

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Abstract Gastroesophageal reflux disease (GERD) and/or hiatus hernia (HH) are one of the most common disorders of the upper gastrointestinal tract. Despite the positive effect of sleeve gastrectomy (SG) regarding weight loss and improvement in obesity co-morbidities, there are concerns about the development of de novo gastroesophageal reflux disease or worsening the existing GERD after this bariatric operation. Furthermore, controversy exists on the consequences of SG in lower esophageal sphincter function and about the ideal procedure when a hiatus hernia is preoperatively diagnosed or discovered during the laparoscopic SG. This review systematically investigates the incidence, the pathophysiology of GERD and/or HH in morbidly obese individuals before and after SG, and the treatment options for concomitant HH repair during laparoscopic sleeve gastrectomy.

Keywords Bariatric surgery · Sleeve gastrectomy · Hiatus hernia · Gastroesophageal reflux disease · GERD

GERD and Obesity

The prevalence of gastroesophageal reflux disease (GERD) is estimated at a range of 20 % in the general population [1]. Obesity is a known risk factor for GERD and/or hiatus hernia (HH) as well as for erosive esophagitis and esophageal adenocarcinoma [2, 3]. Approximately 50–70 % of patients undergoing bariatric surgery for morbid obesity have symptomatic reflux, while a concomitant HH is present in 15 % of patients with BMI >35 kg/m^2 [1]. According to reported studies, 79 % of morbidly obese patients had heartburn, 66 % regurgitation, 49 % presented endoscopic findings of esophagitis with 18 % short segment Barrett's esophagus, and 9 % long segment Barrett's esophagus at the moment of preoperative workup for bariatric surgery. In patients with severe esophagitis submitted to Roux-en-Y gastric bypass (RYGBP), the incidence of GERD is as high as 50–100 % [3, 4].

Although obesity, particularly waist circumference (an index of central adiposity), rather than BMI is strongly associated with GERD [5], absence of classical reflux symptoms,

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such as heartburn, non-cardiac chest pain, recurrent posterior laryngitis are often missing or underestimated in obese individuals. This makes the diagnosis and assessment of GERD difficult, particularly when manometry and/or pH metry is not performed as routine [2].

The pathophysiological mechanisms of the obesity-related GERD are still incompletely understood [1, 6]. The more likely hypothesis is that obesity causes a chronic increase in the intra-abdominal pressure, inducing reflux of gastric content through an ineffective lower esophageal sphincter (LES). The increased intra-abdominal pressure is also leading to delayed gastric emptying and the development of HH [7]. The latest conditions could further impair the anti-reflux mechanisms, worsening the GERD [1, 6, 8]. The mechanisms for developing HH may include esophageal shortening, congenital defect, and increase of intra-abdominal pressure [7].

It has also been proposed that patients with GERD have altered autonomic nervous function and more specifically have reduced parasympathetic activity. Obese individuals also have shown diminished parasympathetic activity, which may be reversed after weight loss through exercise, diet control, and bariatric surgery. Given that contraction and relaxation of LES are vagally mediated, the question that arises is whether the autonomous nervous system is in fact the missing link between obesity and GERD [9].

GERD After Sleeve Gastrectomy

On-site anonymous survey during the Second International Consensus Summit for Sleeve Gastrectomy reported that the mean prevalence of postoperative GERD was 6.5 %, ranging from 0 to 83 % [10]. In selected series involving more than 100 SG patients, symptomatic GERD was reported to occur in 7.8–20 % of cases at 12–24 postoperative months [5]. Cottan et al. [11] reported a series of 126 patients who underwent SG and found a 20 % incidence of GERD at 12 months postoperatively. Hamoui et al. [12] reported 131 SG patients with a 12.7 % incidence of GERD at 13 months; Nocca et al. [13] reported 163 SG patients with an 11.8 % incidence of GERD at 24 months; and Soricelli et al. [1] reported 264 SG patients with a 7.8 % incidence of GERD at 24 months. On the other hand, Daes et al. [5] reported a very low incidence of 1.5 % GERD in a series of 234 patients, by detecting and systematically repairing hiatus defect and by careful attention to surgical technique, avoiding torsion or narrowing of the sleeve.

In a recent review of the literature, an increase in reflux symptoms after SG was reported in four studies [5], while seven studies reported a reduction in GERD symptoms postoperatively [14]. Most studies reported an increase in reflux symptoms during the first postoperative year followed by a gradual decrease in symptoms up to the third postoperative year [15].

Trying to explain the post-sleeve gastrectomy GERD, some studies have suggested that anatomical changes associated with SG may exacerbate GERD symptoms or induce de novo GERD in previously asymptomatic patients [16]. The factors that increase GERD after SG include reduction of LES pressure, possibly from division of ligaments and blunting of the angle of His, reduction in gastric compliance, increased sleeve pressure with an intact pylorus due to the use of bougie <40 Fr, decreased sleeve volume and distensibility, and dilated upper part of the final shape with a relative narrowing of the mid-stomach, without complete obstruction.

Following SG induced by the above factors, GERD is equilibrated by the rapid gastric emptying and the postoperative weight loss. Finally, the resolution of GERD in the long term can be achieved by removal of the fundus, which is the source of relaxation waves to the LES, increased gastric compliance and emptying, reduction of acid secretions, and restoration of the angle of His at 3 years after SG [5, 8, 14, 17–19].

The presence of HH is further complicating the issue of GERD following SG. Symptomatic HH is present in 15 % of patients with body mass index 35 kg/m^2 and is closely associated with GERD and its complications [1]. The diagnosis of HH is based on indirect techniques as the double-contrast barium swallow, the upper gastrointestinal endoscopy, and the esophageal manometry, which do not allow the direct assessment of the gastroesophageal junction. This area is also easily disclosed intra-operatively during upper abdominal surgery procedures [20]. Many surgeons will simultaneously perform the laparoscopic sleeve gastrectomy (LSG) and restore the stretched hiatus by hiatoplasty in case that HH is diagnosed pre- or intra-operatively [21].

Lower Esophageal Sphincter After Sleeve Gastrectomy

For the most important barriers that protect the esophagus from reflux, the LES is modified when a sleeve gastrectomy is performed, dividing the sling fibers and provoking a decrease in the LES resting pressure, as shown by Braghetto's group [22]. Manometric changes occur in LES after sleeve gastrectomy. Mean LES resting pressure (LESRP) decreased significantly after SG from 14.2±5.8 to 10.5±6.06 mmHg (P=0.01). Fifteen percent of patients presented normal LESRP (23.1±3.7 mmHg) and 85 % were hypotensive, with a mean resting pressure of 8.3±2.6 mmHg. After surgery, the length of the high-pressure zone was also affected, with 45 % of patients with shortened total length (shorter than 3.5 cm) and 70 % with abdominal length shorter than 1 cm [3]. The presence of increased GERD with scintigraphic assessment, endoscopic erosive esophagitis, and cardia dilatation was also observed [22].

Other authors have reported similar results. Burgerhart [23] observed a decrease in LES resting pressure from 18.3±9.2 to 11.0±7.0 mmHg (p=0.02) measured by high-resolution manometry.

On the contrary, Del Genio et al. [24] with high-resolution manometry showed an unchanged LES function, increased ineffective peristalsis, and incomplete bolus transit. Interestingly, in the same study, 24-h pH/impedance metry showed an increase of both acid exposure of the esophagus and the number of non-acid reflux events in postprandial periods. The explanation for these findings could be that transient relaxations induce an increase in reflux episodes.

On the other hand, Petersen et al. [6] reported that SG significantly increased LESRP independent of weight loss and they suggest that this surgery may protect obese patients from GERD. They think that this difference is due to technical issues, explaining that the position of the stapler in relation to the angle of His is an important factor. The lesser curvature open inner transverse C-shaped muscle (sling) fibers are approximated, increasing intraluminal tension (Laplace's law). However, the authors described that gastroscopy demonstrated cardiac insufficiency, esophagitis, and HH in most patients. It is hard to find an explanation to these findings if those patients had increased LESRP.

Recently, Rebecchi et al. [25] published the first prospective study with 2 years follow-up, using a clinical validated questionnaire and evaluating 24 h pH monitoring and esophageal manometry, in 71 patients submitted to LSG. The comparative analysis of preoperative and 2 years postoperative clinical and laboratory findings demonstrated an improvement of symptom score in the subgroup with preoperative reflux. Real "de novo" GERD developed in 5.4 % of the asymptomatic subgroup with normal preoperative pH metry and manometry. The authors conclude that LSG did not impair the LES and should be considered an effective option for the treatment of morbidly obese patients with GERD.

Another interesting effect of sleeve gastrectomy is the distortion in the intragastric pressure. Yehoshua et al. [26] observed that basal intragastric pressure does not change after sleeve gastrectomy, but after the occlusion of the stomach and filling with saline increased significantly, implying an important decrease in gastric distensibility. This phenomenon might produce an increase in gastroesophageal pressure gradient after meals, augmenting the regurgitation of gastric content. This might explain why Himpens observed decreasing of GERD symptoms at 3 years after an initial increase. After 1 year, the sleeve remains narrow, but after 3 years, the gastric tube gets wider and compliance is heightened, reducing the intragastric gradient pressure and, therefore, the gastroesophageal reflux or decrease LESP with time [27]. Braghetto's group have also studied patients 5 years after sleeve gastrectomy, and patients with reflux symptoms present a LESRP of 9.8±2.1 mmHg (range 9.6 to 10,9 mmHg) and most of them were converted to laparoscopic Roux-en-Y gastric bypass, due to severe acid reflux on 24 h pH monitoring (De Meester score 25–52). On the contrary patients without reflux present a LESP of 18.3±4.2 mmHg (range 12.2 to 18.3) [28]. Gorodner [29] recently published that LESP is significantly decreased after LSG, while DeMeester score significantly increased.

Hiatus Hernia Repair During Laparoscopic Sleeve Gastrectomy

An aggressive policy of hiatal area exploration during LSG is advocated by several authors [1, 30, 31]. The intraoperative diagnosis of hiatus hernia/ hiatus defect reaches 35 % of cases [1].

However, there are only a few studies with more than 100 patients, investigating the effect of concomitant HH repair (HHR) during LSG on GERD. All with one exception and despite some methodological limitations suggest an improvement of symptoms postoperatively.

Soricelli et al. reported that among 378 patients that underwent LSG, 60 of them (15.8 %) had symptomatic GERD and in 42 patients (11.1 %) a HH was diagnosed preoperatively. In another 55 asymptomatic patients (14.5 %), HH was diagnosed intra-operatively. Therefore, LSG plus HHR was performed in 97 patients. The mean follow-up period in this study was 18 months. GERD remission occurred in 44 patients (73.3 %). In the remaining 16 patients, an improvement in the severity of GERD symptoms and in the anti-reflux medications was evident. De novo reflux symptoms were developed in 22.9 % of the patients who underwent SG alone compared with 0 % of the patients who underwent SG plus HH repair [8].

Daes et al. [5] reported a series of 134 morbidly obese patients that underwent SG and followed up for 6–12 months. Two years later, the same group [32] reported a cohort of 382 patients that underwent LSG, with 373 of them having completed 6–22 months of follow-up. There were 170 patients (44.5 %) with GERD and 197 patients (51.6 %) with preoperative diagnosis of HH. Intra-operatively in 142 patients (37.2 %), HH was confirmed, from which 126 had GERD symptoms preoperatively. All patients with HH confirmed intra-operatively had cruroplasty without mesh reinforcement, even for large hernias. During the follow-up period, only 10 patients (2.6 %) experience GERD symptoms, 8 of which had HH and crural repair.

Gibson et al. reported a series of 500 patients that underwent SG with a mean follow-up of 14 months. During SG, anterior repair of hiatal laxity was performed in 265 patients and posterior repair in 30 patients. Postoperatively, the incidence of GERD reduced from 45 to 6 % and symptoms were well controlled with PPI. The authors do not clearly

describe how many patients underwent HH repair and remained with reflux symptoms postoperatively [33].

Recently, Santonicola et al. compared the clinical outcome of 102 patients submitted to LSG with 78 patients submitted to LSG and concomitant HH repair. Crural defect repair was carried out using non-absorbable (0 Ethibond) interrupted sutures reinforced with a 1×1 cm pledget of Marlex, calibrated on a 40-French orogastric bougie. GERD symptoms were assessed using a standardized questionnaire to evaluate the prevalence of typical symptoms (heartburn and/or regurgitation). The data analysis demonstrated that LSG has a beneficial effect on relieving GERD symptoms, although the underlying mechanisms are still unclear. Conversely, the HH repair did not induce any improvement in GERD symptoms [20]. These findings are in contrast with all of the previous reports that recommended the repair of hiatal defect as a method to prevent/reduce de novo GERD, indicating that detailed knowledge is scanty and this topic remains controversial.

However, in a more recent systematic review on simultaneous LSG and HH repair, the authors found 17 papers including 737 patients. The reported postoperative GERD at a mean follow-up of 24 months was 12.6 %. Sixteen out of 17 papers recommend simultaneous repair of HH during LSG [34].

Mesh Reinforcement of HH Repair

SAGES guidelines 2013 recommend the use of mesh reinforcement in case of large defects and types III and IV HH. Although, the mesh reinforcement of the cruroplasty during anti-reflux surgery carries out low recurrence rates at mid-term follow-up, the type (not-absorbable/absorbable), size of the mesh, shape, and fixing method remain controversial [30]. Severe complications (erosion into the esophagus, aorta, diaphragm, and esophageal stenosis) following the use of polypropylene mesh have been reported [35, 36]. These events, although very rare, require extremely complex management. On the other hand, polytetrafluoroethylene (PTFE) and PTFE composites induce minimal tissue reactions but are opaque and it is difficult to achieve an accurate/adequate fixation. To reduce the risk of erosion, other materials have been tested as follows: ligamentum teres, acellular dermal matrix biodegradable patch, small intestine submucosa, and synthetic bioabsorbable mesh [31, 37, 38]. The recent meta-analysis of Antoniou et al. show that mesh-reinforced HHR in normal weight population is associated with a fourfold decreased risk for anatomic recurrence compared with simple crural closure, without advice of the best type of mesh to be used [39].

In 2014, Silecchia's group published a series of 43 patients with GERD, submitted to LSG (primary or revisional) or anti-reflux surgery (laparoscopic 360° fundoplication), using a synthetic bio-absorbable mesh (BIO-A tissue reinforcement) fixed with absorbable devices (tacks and/or fibrin glue) for hiatal defect ≥4 cm^2. Remission of GERD symptoms was observed in 39 patients (91 %), without mesh-related complications at a mean follow-up of 17.4 months and recurrence rate of 2.3 %. The main steps of the procedure include an accurate hiatal area dissection with reduction in the abdomen of the lower esophagus for at least 5 cm, complete and anatomical vision of the hiatus with right approach (*pars flaccida technique*), and hiatoplasty with non-absorbable stitch reinforced with the absorbable mesh [38].

Based on this evidence, the authors carried out a prospective comparative study about HH repair during LSG performing simple cruroplasty for HH defect <4 cm^2 (group A N=49) and cruroplasty reinforced with BIO-A mesh for HH defect ≥4<8 cm^2 (group B N=38). The hiatal area measurement was performed using a triangle area formula. After a mean follow-up of 16 months, they registered significant statistical differences in terms of postoperative recurrences (group A 16.3 % vs group B 7.9 %; $p<0.05$), GERD symptoms resolution (group A 82.3 % vs group B 94.4 %; $p<0.05$), and transient dysphagia (group A 10.2 % vs group B 18.4 %; $p<0.01$). The latest study underlines the importance of hiatal area exploration during LSG in all cases (high incidence of intra-operative diagnosis of hiatal hernia, 37 %), showing and confirming the safety and efficacy of HH repair with absorbable mesh (no mesh-related complications, low recurrence rate, 7.9 %) in case of hiatal defect <8 cm^2 [unpublished data]. A recent systematic review on concomitant HH repair and LSG revealed 7 studies with only 31 reported cases with mesh reinforcement, 29 biological and 2 polypropylene. All studies report a satisfactory outcome even for large defects [34].

GERD Complicating Sleeve Gastrectomy: Roux-en-Y Gastric Bypass or Re-sleeve (ReSG)?

Surgery is the last option for GERD complicating LSG. GERD should be carefully defined with an exhaustive workup including upper GI endoscopy, high-definition manometry, and pH impendancometry [24]. If required, computerized tomography (CT) scan volumetric assessment of the gastric sleeve should be performed [40].

These investigations are meant to demonstrate the correlation between symptoms reported by patients and GERD. Preoperative workup is also intended to identify anatomical anomalies as strictures resistant to endoscopic treatment or functional strictures including the twisted sleeve [41] that are formal indication to surgery. Surgical treatment should be thus reserved to patients with proven GERD resistant to full dose of PPIs. Once the indication for surgery has been established, the second step concerns the choice of the procedure.

The gold standard of anti-reflux procedure in the morbidly obese remains the standard RYGBP. Indeed, the latter presents several mechanisms that prevent GERD. The small lesser

curvature-based gastric pouch has virtually no acid content as the acid-producing mucosa of the fundus is excluded. The Roux-en-Y loop avoids biliary reflux and exerts a downward pulling effect that retains the gastric pouch into the abdomen. For these reasons, when doing a conversion from LSG into RYGB [42], the pouch must be resized and any persistent gastric fundus has to be resected [43]. The hiatus should be carefully inspected for the presence of hiatal hernia as we found that in the long term the latter may be responsible for the migration of the gastric pouch into the thorax, although this condition may cause dysphagia which requires surgical treatment [44].

ReSG has been introduced a few years ago as a rescue procedure for weight loss failure or regain after SG [45]. With time, it has been clear that this procedure can be effective mainly in patients with what has been called primary dilation. The last consists in a gastric sleeve that has been fashioned too large at primary surgery. The question has been addressed if ReSG may be used in patients showing GERD after LSG. Noel et al. recently used this strategy in two patients and reported favorable short-term results [18].

The rational for this strategy relies in the resection of an exceedingly large residual gastric fundus that is supposed to be responsible for GERD symptoms. Preoperative CT scan volumetric assessment of the gastric tube is of mainstay importance to demonstrate a large persistent fundus. The resection of the exceeding fundus results in the reduction of acid secretion in the gastric tube. Furthermore, any regurgitation due to stasis of the bolus into the residual fundus is also eliminated. If hiatal hernia is found, it should be fixed, as it may be responsible for GERD in itself.

Conclusion

Although GERD is a common disease in morbidly obese patients, there is lack of common policy concerning the required investigations for precise diagnosis and accurate assessment of this condition in patients scheduled for bariatric surgery. Furthermore, since preoperative diagnosis of HH is often missing, the need for meticulous intra-operative evaluation of the hiatus is indispensable.

Sleeve gastrectomy may improve or aggravate existing GERD or may lead to “de novo” reflux. There is no consensus among investigators on the effects of SG in LES function and on the mechanisms responsible for the outcome of existing GERD and the development of de novo GERD. However, most authors agree that, with the exception of severe reflux and Barret esophagus, SG can be safely and effectively performed when bariatric surgery is indicated.

Additionally, there is no universally accepted policy for the need and technical methods for simultaneous SG and HH repair, although most authors agree that posterior hiatus repair is necessary when HH is diagnosed pre- or intra-operatively.

In our opinion, long-term randomized control trials are required to highlight all debated issues and allow definitive conclusions.

Compliance with Ethical Standards This is a review article and no ethical approval is required.

Conflict of Interest The authors declare that they have no competing interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human participants or animals performed by any of the authors. No additional informed consent is required.

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