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Endoscopic findings and gastroesophageal reflux changes after sleeve gastrectomy: Results of 18-month follow-up

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ABSTRACT

Introduction: Obesity is now a critical public health challenge globally. Laparoscopic Sleeve Gastrectomy (LSG) is a widely performed obesity surgery known for its effectiveness in promoting weight loss and improving obesity-related conditions. The aim of study was to assess the presence of gastroesophageal reflux symptoms (GERD) symptoms after LSG.

Materials and Methods: This retrospective observational study included patients who underwent LSG for morbid obesity between October 2022 and March 2024. Demographic characteristics, body mass index (BMI), percentage of excess weight loss (%EWL), and comorbidities were assessed preoperatively and at 6, 12, and 18 months postoperatively. Esophagogastroduodenoscopy (EGD) was performed before surgery and at the 18-month follow-up.

Results: Eighty-six patients met the inclusion criteria (52 females, 60.4%), with a mean age of 39±8 years. Mean preoperative BMI was 43.8±14 kg/m², which decreased to 28.6±6 kg/m² at 18 months, corresponding to a mean %EWL of 61±8.4%. Preoperative GERD symptoms were present in 19 patients (22.1%), increasing to 25 (29.1%) postoperatively ($p>0.05$). EE was identified in 14 patients (16.2%) before surgery and in 25 patients (29.1%) after surgery ($p<0.001$). De novo EE developed in 22 patients (25.5%), mostly Grade A (72.7%). HH prevalence increased from 20.9% to 30.2% ($p<0.001$).

Conclusions: In summary, our 18-month follow-up demonstrated an increase in gastroesophageal reflux and endoscopic evidence of esophagitis after sleeve gastrectomy.

Keywords: Erosive esophagitis, gastroesophageal reflux symptoms, hiatal hernia, laparoscopic sleeve gastrectomy, obesity

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Introduction

Obesity is now a critical public health challenge globally.[1,2] The rise in obesity correlates with increased rates of non-communicable diseases such as diabetes, cardiovascular diseases, and some cancers, contributing to an estimated 1.6 million premature deaths annually linked to overweight and obesity.[1] Laparoscopic sleeve gastrectomy (LSG) has gained wide acceptance as a primary bariatric procedure due to its technical simplicity, safety, and metabolic efficacy.[3,4] However, LSG can have significant effects on the anatomy and function of the stomach.[5] The operation involves longitudinal resection of approximately 75–80% of the stomach, resulting in a tubular gastric remnant along the lesser curvature.[4,5] This technique of sleeve gastrectomy may influence the development or worsening of gastroesophageal reflux symptoms (GERD).[6-8] Gastroesophageal reflux symptoms typically include heartburn, regurgitation, chest pain, and other discomforts caused by the backflow of stomach acid into the esophagus. Although obesity itself is a major risk factor for GERD due to factors like increased intra-abdominal pressure and altered gastroesophageal anatomy,[7] the anatomical changes after LSG—such as removal of the gastric fundus and alterations in lower esophageal sphincter (LES) pressure—can either exacerbate or, in some cases, improve reflux symptoms.[6-9] The interaction between LSG and GERD remains a critical consideration when evaluating surgical options and managing postoperative outcomes in obese patients. Erosive esophagitis (EE) and hiatal hernia (HH) may accompany GERD; for this reason, the identification of HH and esophagitis is important preoperatively.[9,10]

This study aimed to evaluate the progression of GERD symptoms, EE, and hiatal hernia in patients undergoing LSG, with a particular focus on endoscopic outcomes during an 18-month follow-up period.

Materials and Methods

This study was approved by the Istanbul Yeni Yüzyıl University Ethics Committee (No:2025/10-1663, Date: 07/10/2025) and conducted according to the Helsinki Declaration.

This retrospective study included patients who underwent LSG for morbid obesity between October 2022 and March 2024 at the Bariatric Surgery Department. Inclusion criteria were: Age 18–60 years, BMI ≥35 kg/m² with comorbidities or ≥40 kg/m² without comorbidities, and complete pre- and postoperative follow-up data. Patients with prior upper gastrointestinal surgery, active peptic ulcer disease, or incomplete endoscopic data were excluded.

Comorbidities were assessed using the following criteria:

- **Hypertension (HT):** Based on Brethauer criteria.[11]
- **Diabetes (DBT):** According to the American Diabetes Association guidelines.[12]
- **Obstructive Sleep Apnea (OSA):** Evaluated clinically, focusing on improvements in daytime sleepiness and nocturnal snoring.
- **Fatty Liver, Metabolic syndrome**

Gastroesophageal reflux disease (GERD) was defined according to the *Montreal Consensus*.[13] GERD-like symptoms were assessed using a dedicated questionnaire administered both preoperatively and at the 18-month follow-up (Table 1).

Esophagogastroduodenoscopy (EGD) was performed before surgery and at the 18-month follow-up. The presence of **EE)** and **HH** was documented. EE was classified

Table 1. Symptomatic assessment of patients

Symptoms (heartburn and/or regurgitation)				
Frequency	**Score**	**Intensity**	**Score**	**Category**
Never	0	None	0	—
1 or 2 days/week	1	Does not cause discomfort, does not interfere with daily activities	1	Mild: 1–2
3 or 5 days/week	2	Causes some discomfort but does not interfere with daily activities	2	Moderate: 3–4
6 or 7 days/week	3	Interferes with daily activities	3	Severe: 5–6

using the *Los Angeles Classification*.[14] HH was diagnosed following the guidelines of the *Society of American Gastrointestinal and Endoscopic Surgeons (SAGES)*, either during preoperative endoscopy or through intraoperative visual inspection of the anterior hiatus.[15]

The crura were not routinely dissected to search for HH. Importantly, there was a 100% correlation between preoperative EGD findings and intraoperative observations.

Proton pump inhibitors (PPIs) were routinely prescribed for 12 months following laparoscopic sleeve gastrectomy (LSG), regardless of the presence or absence of symptoms. If erosive esophagitis was detected during the 18-month follow-up EGD, PPIs were reintroduced for 12 weeks at a dosage of pantoprazol 40 mg/day. A repeat EGD was performed after this treatment period only in patients with EE classified as grade B or C.

Helicobacter pylori (HP) infection was assessed via gastric biopsies obtained according to the Sydney protocol. If HP was detected, patients received eradication therapy consisting of amoxicillin 2 g/day, clarithromycin 500 mg/day, and pantoprazol 40 mg/day for 14 days prior to LSG.

Esophageal biopsies were obtained only when Barrett's esophagus (BE**)** was suspected, following the Seattle protocol. Morbidity and mortality outcomes were also evaluated.

Surgical Technique

A 12-mm optical trocar was inserted under direct vision at the supraumbilical midline for camera access. Subsequently, two 5-mm trocars were placed—one in the left upper quadrant and the other in the right upper quadrant—and a 10-mm trocar was positioned in the left flank. A Nathanson liver retractor was introduced through the epigastrium.

The short gastric vessels were divided using a harmonic scalpel. Dissection of the greater curvature of the stomach was carried out up to the left crus. Gastric sleeve calibration was performed using a 40 French bougie.

Vertical gastric transection was completed using 4 to 5 linear staplers (Covidien 60; Endosurgery), beginning 4–6 cm from the pylorus along the greater curvature and proceeding parallel to the lesser curvature toward the angle of His. Staple cartridge selection—black (4.4 mm), purple (4.1 mm) was based on the thickness of the gastric wall. The resected stomach was retrieved using a specimen bag, and a 10 mm Jacksson Prat drain was placed.

In cases where a hiatal hernia was identified, repair involved dissection of the crura, repositioning of the stomach into the abdominal cavity, and posterior closure of the hiatal defect with interrupted 2-0 polyester sutures (ETHIBONDÒ 2-0; Ethicon). The same 40 Fr bougie was used for calibration during hernia repair.

Statistic Methods

Data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The Kolmogorov–Smirnov test was used to assess the normality of data distribution.

Comparisons between preoperative and postoperative values were conducted using the paired Student's t-test for normally distributed continuous variables and the Wilcoxon signed-rank test for non-normally distributed variables. The chi-square test or Fisher's exact test was applied to evaluate associations between categorical variables, including the evolution of GERD symptoms, EE, and hiatal hernia before and after sleeve gastrectomy. A p-value of <0.05 was considered statistically significant.

Results

A total of 86 patients who met the inclusion criteria were enrolled in the study. The cohort consisted of 52 women (60.4%), with a mean age of 39±8 years. The average preoperative BMI was 43.8±14 kg/m². At 18-month follow-up, the mean BMI decreased to 28.6±6 kg/m², and the mean percentage of excess weight loss (%EWL) was 61%±8.4%. Helicobacter pylori (HP) infection was identified in 21 patients (24.4%) during preoperative screening; all received eradication therapy prior to surgery.

Evolution of comorbidities after LSG

Among preoperative comorbidities, type 2 diabetes was present in 12 (13.9%) patients, hypertension in 16 (18.6%), and OSA in 19 (22.1%). Resolution or improvement occurred in 66.6%, 75%, and 84.2%, respectively.

Morbidity and mortality

Postoperative complications were observed in 3 patients (3.48%), all of whom developed hematomas. All of patients were managed conservatively. There were no mortalities in this series. Additionally, no patients required conversion to Roux-en-Y gastric bypass (RYGB) due to

Table 2. Resolution of comorbidites after LSG

Comorbidity	Before LSG, n (%)	Resolution (%)	Same (%)	Worse
Type 2 DM	12 (13.9)	8 (66.6)	4 (33.3)	–
HT	16 (18.6)	12 (75)	4 (25)	–
OSA	19 (22.09)	16 (84.2)	3 (15.7)	–

DM: Diabetes mellitus; HT: Hypertension; LSG: Laparoscopic sleeve gastrectomy; OSA: Obstructive sleep apnea.

gastroesophageal reflux disease (GERD) symptoms or endoscopic findings.

Evolution of GERD symptoms after LSG

The number of patients reporting GERD symptoms increased from 19 (22.09%) preoperatively to 25 (29.06%) postoperatively; however, this change was not statistically significant. Among the 19 patients with preoperative symptoms, 6 (31.5%) experienced resolution after LSG, while 13 (68.4%) reported persistent symptoms. Notably, 12 of the 86 patients (13.9%) who were asymptomatic before surgery developed de novo GERD symptoms following the procedure (Fig. 1).

In the group of 19 patients with preoperative symptoms, endoscopic findings included erosive esophagitis (EE) in 32%, hiatal hernia (HH) in 39%, and normal EGD in 29%. Postoperatively, among the 25 symptomatic patients, EE was found in 8 (32%), HH in 4 (16%), and normal EGD in 13 (52%). A significant association was observed between GERD symptoms and the presence of EE on postoperative EGD ($p<0.05$). The most commonly reported symptom was heartburn (22 patients, 88%), followed by regurgitation (3 patients, 12%). Of the symptomatic patients reported their postoperative GERD symptoms as mild in intensity and infrequent, all of whom achieved complete relief with standard-dose proton pump inhibitors (PPIs). Four patients (16%) experienced symptoms at least three times per week (moderate) and also responded well to standard-dose PPIs. The remaining patients reported daily symptoms (severe) and were treated with double-dose PPIs for six months, resulting in effective symptom control in all cases.

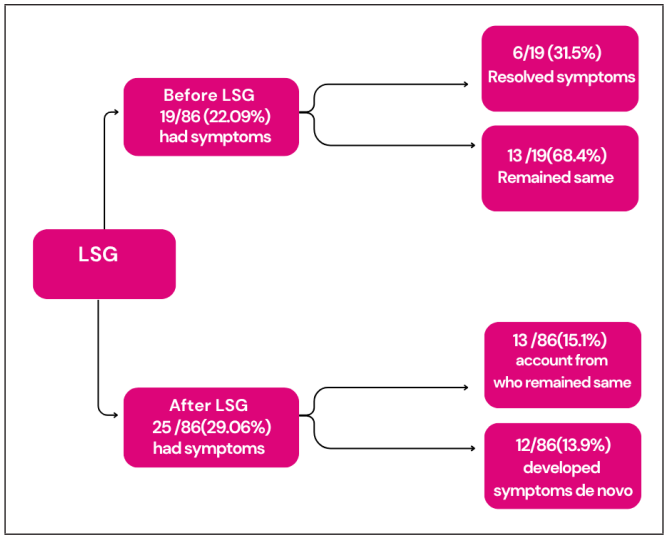

Figure 1. Assessment of symptoms after LSG.

Evolution of EE after LSG

During the preoperative evaluation, 14 patients (16.2%) were diagnosed with erosive esophagitis (EE), all classified as Grade A. These patients were offered Roux-en-Y gastric bypass (RYGB) as an alternative surgical option due to the risk of worsening esophagitis following LSG. Only 2 patient accepted RYGB surgery.

At the 18-month follow-up, EE was identified in 25 patients (29.06%) on endoscopy ($p<0.001$). Among these, 19 patients (76%) had Grade A esophagitis, 5 (20%) had Grade B, and 1 (4%) had Grade C.

Of the 14 patients with preoperative EE, 8 (57.1%) showed complete resolution postoperatively, while the remaining 6 (42.8%) had persistent EE of the same grade. Also, 2 patients who performed RYGB for EE showed complete resolution after surgery.

De novo EE developed in 22 patients (25.5%). Among them, 16 (72.7%) had Grade A, 5 (22.7%) had Grade B, and 1 (4.5%) had Grade C esophagitis. All patients received a 12-week course of proton pump inhibitors (PPIs). Follow-up endoscopy was performed in those with Grade B or C findings. Among the 5 patients with Grade B EE, 4 (80%) showed resolution, while 1 (20%) had persistent disease. Patient with Grade C EE showed no improvement. None of the patients developed Barrett's esophagus. The remaining 64 patients (74.4%) showed no evidence of EE on follow-up endoscopy.

Evolution of HH after LSG

The prevalence of hiatal hernia (HH) demonstrated a significant postoperative increase, rising from 18 patients (20.9%) before surgery to 26 patients (30.2%) after the procedure (p<0.001). Among the 18 patients with preoperative HH, concomitant hiatal hernia repair during laparoscopic sleeve gastrectomy (LSG) was performed in only 4 cases (22.2%). Recurrence of HH was identified in 1 of these 4 patients (25%) on follow-up esophagogastroduodenoscopy (EGD), with all recurrent hernias measuring approximately 2 cm in size. Interestingly, *de novo* HH developed in 8 patients (9.3%) postoperatively. Evaluation of erosive esophagitis (EE) among the 26 patients with postoperative HH revealed that 9 (34.6%) had normal endoscopic findings, whereas 10 (38.4%) presented with EE. The temporal evolution of clinical symptoms, EE, and HH is summarized in Table 3.

Discussion

The association between laparoscopic sleeve gastrectomy (LSG) and postoperative gastroesophageal reflux disease (GERD) remains controversial. This study demonstrated a significant increase in endoscopic evidence of GERD, erosive esophagitis, and hiatal hernia following LSG, despite substantial weight loss and improvement in metabolic comorbidities.

Obesity is a well-established risk factor for the development of GERD and its associated complications, including esophagitis, Barrett's esophagus, and esophageal adenocarcinoma.[16,17] Despite the efficacy for weight reduction and metabolic improvement of LSG, may predispose patients to de novo or worsened reflux symptoms and esophageal mucosal injury.

Table 3. Symptoms of patients before and after LSG

Variables	Before LSG (n=86) (%)	After LSG (n=86) (%)	p
GERD symptoms	19 (22.09)	25 (29.06)	0.212
EE	14 (16.2)	25 (29.06)	p<0.001
Grade A	14 (100)	19 (76)	
Grade B	0	5 (20)	
Grade C	0	1 (4)	
Hiatal hernia	18 (20.9%)	26 (30.2%)	p<0.001

EE: Erosice esophagitis; GERD: Gastroesophageal reflux disease; LSG: Laparoscopic sleeve gastrectomy

Several studies have reported similar trends of increased reflux and esophagitis after LSG. Himpens et al.[18] followed patients for five years after LSG and noted that 21.8% developed new-onset GERD symptoms, while 8.6% had endoscopic evidence of erosive esophagitis. Braghetto observed an even higher incidence, reporting that up to 30% of patients developed de novo GERD at two years postoperatively.[6] A systematic review and meta-analysis by Qumseya et al.[19] found that the pooled prevalence of new-onset GERD after LSG was 23%, and the incidence of erosive esophagitis increased significantly compared with preoperative values. Our findings are consistent with these results, reinforcing that the restrictive and high-pressure nature of the gastric sleeve contributes to reflux pathophysiology.

Several studies have documented a significant rise in de novo EE after sleeve gastrectomy. According to Braghetto et al.[6] the prevalence of EE increased from 6.5% preoperatively to 27.5% one year postoperatively. Similarly, Fernández-Ananín S et al.[8] reported new-onset EE in 32% of patients following LSG. A systematic review and meta-analysis by Yeung et al.[9] demonstrated that up to 35–40% of patients may develop new or worsened EE within two years after surgery. These data indicate that LSG is not only an effective bariatric procedure but also one that carries a significant risk for postoperative reflux complications. In our cohort, some patients experienced complete resolution of EE, possibly due to and effective medical management. In our study, although some patients showed improvement in erosive esophagitis, the overall incidence of de novo esophagitis findings was higher. This observation indicates that our results are consistent with the findings reported in the literature.

Also, several studies have reported an increased incidence of HH after sleeve gastrectomy. Daes et al.[20] demonstrated that up to 25% of patients developed new or enlarged hiatal hernias postoperatively. Similarly, Braghetto et al.[6] and Fernández-Ananín S et al.[8] observed postoperative HH formation in 20–30% of patients at 1–2 years follow-up. Yeung et al.[9] in a meta-analysis, confirmed that LSG is associated with a higher prevalence of both de novo and persistent hiatal hernias compared with Roux-en-Y gastric bypass (RYGB). An important factor in the postoperative evolution of HH is the presence of an unrecognized or unrepaired hiatal hernia at the time of surgery. Undiagnosed small sliding hernias are not uncommon in obese patients, and failure to repair them during LSG can lead to

postoperative enlargement or symptomatic reflux.[10] For this reason, routine preoperative endoscopic or radiologic evaluation is recommended to identify hiatal defects. Several authors advocate concurrent cruroplasty when a hiatal hernia is present during LSG. Studies by Soricelli et al.[10] and Kumar et al.[21] demonstrated that simultaneous LSG and crural repair reduce postoperative GERD symptoms and the likelihood of HH progression. Consistent with the literature, our study also revealed a tendency for GERD symptoms to increase after LSG.

Mechanisms of Reflux after LSG

Multiple mechanisms can explain the observed increase in reflux after LSG:

1. **Anatomical alterations:** Resection of the gastric fundus eliminates the gastric reservoir and the angle of His, both of which play a protective role against reflux. The transformation of the stomach into a narrow tubular conduit increases intragastric pressure and facilitates reflux episodes.[7]
2. **Lower esophageal sphincter (LES) changes:** LSG may alter the pressure gradient across the gastroesophageal junction and impair LES function, reducing its barrier efficacy.[6]
3. **Hiatal hernia formation or unmasking:** The increased intragastric pressure and vertical orientation of the sleeve can promote hiatal hernia development or exacerbate pre-existing defects.[20]
4. **Disrupted gastric emptying dynamics:** Sleeve geometry, sleeve torsion, or narrowing near the incisura angularis may cause delayed gastric emptying and raise the risk of acid exposure to the esophagus.

These factors collectively create a high-pressure environment that promotes the backflow of gastric contents into the esophagus, explaining the increase in GERD symptoms and endoscopic findings observed in our cohort.

Correlation between Symptoms and Endoscopic Findings

Interestingly, not all patients with endoscopic esophagitis reported typical reflux symptoms, confirming the poor correlation between subjective symptomatology and objective mucosal changes.[22] This discrepancy may result from empiric PPI therapy masking symptoms or the altered perception of reflux after significant weight loss. Therefore, objective assessment—through endoscopy or 24-hour pH/impedance monitoring—is crucial in evaluating post-LSG reflux.

Clinical Implications

Given the demonstrated risk of reflux after LSG, preoperative screening for GERD and hiatal hernia is essential. Patients with severe or refractory GERD preoperatively may be better candidates for Roux-en-Y gastric bypass (RYGB), which has been consistently shown to improve reflux symptoms.[23] For patients developing significant postoperative GERD, medical management with PPIs and lifestyle modification remains first-line therapy, but revisional surgery (conversion to RYGB) should be considered for refractory cases or those with progressive esophagitis or Barrett's metaplasia.[24]

Limitations

The main limitations of our study include its single-center design and moderate sample size, which may restrict generalizability. Endoscopic evaluation was limited to the 18-month mark, and pH-impedance testing was not routinely performed, preventing correlation between physiologic reflux and mucosal injury. Nevertheless, our findings provide valuable mid-term insight into reflux evolution following LSG.

Future Directions

Future prospective, multicenter studies should incorporate combined endoscopic and physiologic reflux testing (pH-metry, manometry) before and after LSG. Long-term surveillance is warranted to assess the development of Barrett's esophagus and potential malignant transformation in this population. Further, comparative trials between LSG and RYGB will clarify the optimal bariatric approach in patients with baseline reflux.

Conclusion

In summary, our 18-month follow-up demonstrated an increase in gastroesophageal reflux and endoscopic evidence of esophagitis after sleeve gastrectomy. These findings reinforce the importance of meticulous preoperative evaluation, surgical technique optimization (including hiatal hernia repair), and structured postoperative surveillance to identify and manage reflux early and effectively.

Disclosures

Ethical Approval: This study was approved by the Istanbul Yeni Yüzyıl University Ethics Committee (No:2025/10-1663, Date: 07/10/2025)

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