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De novo gastroesophageal reflux disease after sleeve gastrectomy: role of preoperative silent reflux

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Abstract

Background Laparoscopic sleeve gastrectomy (LSG) has become the most frequently performed bariatric procedure to date. However, LSG is known to worsen pre-operative and result in de novo gastroesophageal reflux disease (GERD). Pre-operative evaluation reveals a high percentage of silent GERD of so far unknown influence on post-operative GERD.

Methods Prospective data of patients undergoing primary LSG between 01/2012 and 12/2015 were evaluated. Pre-operative GERD-specific evaluation consisted of validated questionnaires, upper endoscopy, 24 h-pH-manometry, and esophagograms. Patients were followed-up with questionnaires every 6 months, upper endoscopies after 1 year and 24 h-pH-metry after 2 years. Silent GERD was defined as esophagitis grade > B and/or abnormal esophageal acid exposure in absence of symptoms. LSG was performed over a 32F bougie, hiatal hernias > 1 cm were addressed with posterior hiatoplasty. Excluded were patients with hiatal hernias > 4 cm, patients with incorrect anatomy (stenosis, fundus too large) and conversion to RYGB for early leaks.

Results 222 patients were included. Mean follow-up was 32 ± 16 months, mean preoperative body mass index 49.6 ± 7.2 kg/m^2. 116 patients (52%) presented with post-operative GERD-symptoms, of which 85 (73%) had de novo symptoms. Of those, 48 (of 85, 56%) had no preoperative GERD and 37 (of 85, 44%) silent GERD. 57 patients (26%) had neither pre- nor post-operative GERD, 7 (3%) had silent pre-operative and no postop GERD, and in 19 patients (9%) GERD was cured with LSG. 31 patients (14%) stayed symptomatic. Of 56 patients (25%) with pre-operative silent GERD, 37 (of 54, 66%) became symptomatic.

Conclusion LSG leads to a considerable rate of post-operative GERD. De novo-GERD consist of around half of pre-operative silent GERD and completely de novo-GERD. Most patients with pre-operative silent GERD became symptomatic.

Keywords Gastroesophageal reflux disease · Obesity · Body mass index · Sleeve gastrectomy · Asymptomatic diseases · 24-h pH-monitoring

Gastroesophageal reflux disease (GERD) and morbid obesity are closely intertwined, a higher body mass index (BMI) and in particular central obesity are associated with higher rates of GERD [1]. Several factors favor the development of GERD in morbidly obese patients: higher intraabdominal pressure with subsequent disruption of the hiatal region leading to an increased prevalence of hiatal hernias [2, 3], higher frequency of transient lower esophageal sphincter relaxations [4], type 2 diabetes and possible subsequent gastroparesis with delayed gastric emptying [5].

Bariatric surgery is the only sustainable method to address morbid obesity and its resulting comorbidities. Its use parallels the rise of the worldwide obesity epidemic. In 2014, there were well over half a million procedures performed worldwide [6]. In recent years, laparoscopic sleeve gastrectomy (LSG) has become the most frequently performed bariatric procedure [6–8]. Maintained anatomy, absence of anastomoses, shorter operative times and decreased technical difficulty contribute to its popularity. It can be used as stand-alone procedure or in multi-step concepts [9, 10]. So far, mid-term results show a comparable weight loss and resolution of comorbidities to Roux-Y Gastric Bypass (RYGB),

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except for GERD [11, 12]. LSG leads to de novo-GERD and might worsen pre-existing GERD, even though data are heterogeneous [13–22].

A high rate of "asymptomatic" or silent GERD is found in the preoperative evaluation of bariatric patient with reported rates up to 45% [23–28]. Decreased esophageal sensitivity seems to play a major role in its pathogenesis [25]. So far, there is no data on evolution of preoperative silent GERD and its relevance to the contribution of "de novo" GERD.

Materials and methods

Data of patients undergoing primary LSG in a university hospital between January 2012 and December 2015 were recorded in a prospective computer database and reviewed retrospectively. Excluded were patients without hiatal hernia closure despite hiatal hernias > 1 cm, with hiatal hernias > 4 cm, conversion to RYGB due to early leak, incorrect post-operative anatomy in esophagogram (stenosis, fundus too large [29]), refusal of preoperative functional evaluation, and lost to follow-up before post-operative endoscopy. The local ethics committee approved this study.

Patients were seen at least twice before and 3 weeks, 3, 6, 12, 18 months after LSG and on a yearly basis thereafter or more often if needed.

GERD-specific, pre-operative evaluation consisted in assessment of symptoms with standardized questionnaires (frequency and severity scores) [30, 31], upper endoscopies, esophagograms, and 24 h-pH-impedance-manometries off-proton pump inhibitors (PPI). Assessment of symptoms was performed every 6 months, upper endoscopies after 1–2 years and 24-pH-impedance-manometries after 2–3 years in patients without GERD-symptoms. Helicobacter pylori was eradicated prior to LSG.

Patients were stratified into three groups according to the results of upper endoscopy, pH-metry and questionnaire: no GERD, silent GERD and (symptomatic) GERD.

No GERD patients had no symptoms (up to 2 GERD-episodes per week), esophagitis Los Angeles (LA) grade A or less, and no pathological esophageal acid exposure (> 4% of time esophageal pH < 4).

Silent GERD was defined as objective evidence of GERD (esophagitis LA grade ≥B and/or pathological esophageal acid exposure) in absence of symptoms.

GERD patients had both, symptoms and objective evidence of GERD.

For LSG, a standardized technique was used described in detail elsewhere [10]. In short, after establishing a pneumoperitoneum, the greater curvature was resected alongside a 32F bougie beginning 5 cm oral to the pylorus up to the angle of His. Hiatal hernias > 1 cm were addressed with posterior hiatoplasty using non-absorbable sutures. PPI were prescribed routinely for 6 weeks postop, thereafter only in symptomatic patients.

Indications for LSG were high-risk patients [10], patients with higher BMI (> BMI 50 kg/m^2) and patient's wish.

Values are reported as means with standard deviations.

Results

Of 247 operated patients, 222 patients met the inclusion criteria, 103 (46%) of which were male. Of the 25 excluded patients, 4 had incorrect anatomy, 2 were converted to RYGB, the others were either lost to follow-up or refused repeated functional testing. Mean follow-up was 32 ± 16.1 months. Mean preoperative BMI was 49.6 ± 7.2 kg/m^2, mean BMI after 2 years 32.3 ± 12.2 kg/m^2. Mean age was 43.1 ± 12.8 years.

Post-operative upper endoscopies were performed in 100% of patients after a mean 1.6 years; 24 h-pH-impedance-metries in 139 patients (63%) after a mean 2.3 years.

41 patients (18%) had insulin-dependent diabetes mellitus, 87 (39%) were under antidepressants.

Rates of pre-and post-operative GERD are detailed in Fig. 1. 48 patients (42%) of 114 patients without pre-operative GERD became symptomatic. Of 56 patients (of 222, 25%) with pre-operative silent GERD, 37 (66%) became symptomatic and 7 (13%) showed no postop GERD. 52 patients (23%) had pre-operative GERD, of those, 31 (60%) stayed symptomatic and 19 (37%) had no post-operative GERD.

Overall, 116 patients (52%) presented with post-operative GERD-symptoms, of which 85 (73%) had de novo symptoms (Fig. 2).

Preoperative GERD-symptoms were typical in 52 patients (100%), respiratory in 8 (15%), obstructive in 3 (6%) and pain-related in 9 (17%).

116 patients had postoperative GERD-symptoms; they were typical in 108 (93%), respiratory in 7 (6%), obstructive in 12 (10%), and pain-related in 13 patients (11%).

Discussion

This study addresses the impact of pre-operative silent GERD on GERD-symptoms after LSG. 25% of patients presented with pre-operative silent GERD, of those, 66% became symptomatic. Overall, GERD-symptoms were present in 23% of pre-operative and 52% of post-operative patients.

In morbidly obese patients, a high rate of asymptomatic patients despite objective evidence of GERD is not a novel finding. In several studies, strictly symptom-based diagnosis of GERD turned out to be unreliable [23–28, 32,

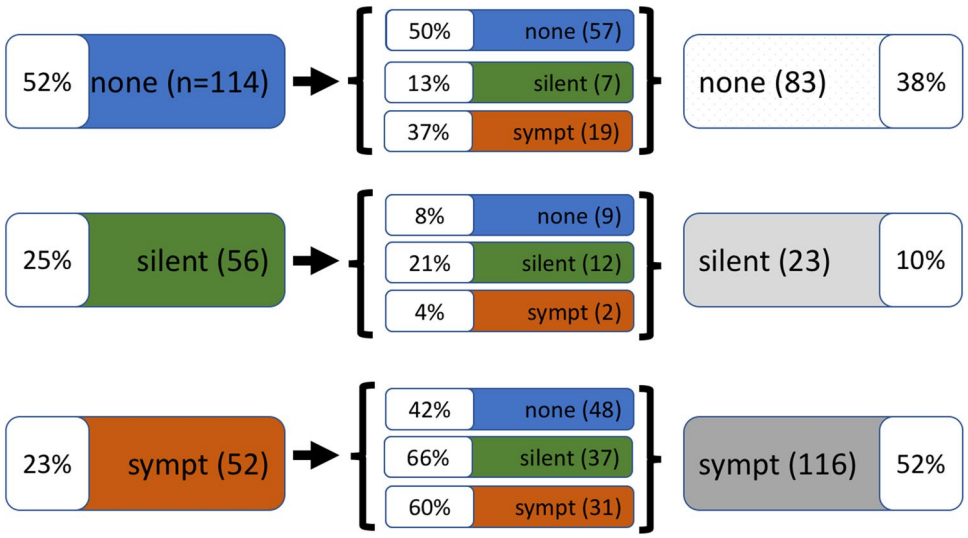

Fig. 1 Evolution of GERD from prior (left) to after (right) LSG, stratified into three groups (*sympt* symptomatic). Percentages in right and left columns refer to overall number of patients ($n = 222$), percentages in middle column to subcategory

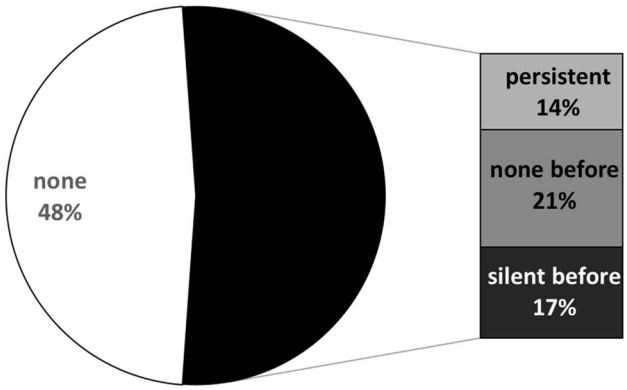

Fig. 2 Patients with de novo symptoms of GERD after LSG

33]. Amongst others, Jaffin et al. reported 61% abnormal manometric findings in 111 pre-operative patients; 59% had no symptoms [32]. In 116 pre-operative morbidly obese patients, Koppman et al. found a prevalence of 41% of abnormal manometric findings, but only one patient with symptoms [33]. Ortiz et al. found a sensitivity of only 29.3% for typical symptoms to diagnose GERD in 158 morbidly obese patients. 47% had abnormal esophageal acid exposure and/or esophagitis, 71% were asymptomatic [34]. In a group of 45% asymptomatic out of 88 pre-operative bariatric patients, Martin-Perez et al. reported 50% abnormal pH-metries [26]. Yet the reasons for this high rate of silent GERD in the morbidly obese are less elucidated. In another study, Ortiz et al. compared 30 morbidly obese patients to 28 controls, they were found to be significantly less symptomatic (14 vs. 96%) and to have decreased esophageal sensitivity (57 vs. 14%) when instilled in the esophagus with acid during pH-metry [25]. Further, Frokjaer et al. reported a decreased esophageal sensitivity in patients with diabetes mellitus together with a reduced compliance of the gastroesophageal junction and increased stiffness of the esophagus [35]. Altered autonomic nervous system in the morbidly obese may serve as another explanation. Manabe et al. found autonomic dysfunction as cause for esophageal motility disorders in normal weight patients with GERD [36]. In 22 morbidly obese patients, Surrenti et al. showed reduced esophageal peristaltic waves and delayed esophageal transit in presence of autonomic nerve dysfunction when compared to controls [37].

LSG as procedure not only to encounter morbid obesity but also its associated comorbidities should therefore result in an improvement of GERD. However, as with others, this study showed an overall increase of 23–52% of patients with GERD. In a symptom-based reporting of 4832 patients, LSG was not found to reliably improve GERD and to result in de novo-GERD in 8.6% [17]. In a prospective, randomized study comparing LSG to LRYGB after 5 years, GERD worsened in 31.8% after LSG versus 6.3% after RYGB and improved in 25 versus 60% after RYGB [12]. Georgia et al. assessed pre- and post-LSG-GERD in 12 asymptomatic patients with pH-metry and found a rate of 83% of postoperative GERD, de novo in 50% [18]. Gorodner et al. assessed esophageal function pre- and post-LSG in 14 patients and found de novo-GERD in 36% and worsening in 21% [38]. Conversely, in a study using 24 h-pH-manometry involving 65 patients, Rebecchi et al. found LSG to improve GERD and to result only in 5.4% de novo-GERD Further, no significant changes in esophageal function were found [21].

In this study, in the 23% of patients with preoperative GERD, 37% had no postoperative and 60% persistent GERD. Of 52% without preoperative GERD, 42% developed symptomatic GERD; of 25% with silent GERD, 66% became symptomatic (Figs. 1, 2).

There is discussion about the necessity and extent of pre- and postoperative evaluation. In this study, 25% of patients had silent GERD, of those, 66% became symptomatic, 21%

stayed silent. There is no data on long-term consequences after LSG in patients with silent GERD. Yet as acid exposure is equal to symptomatic GERD, it might result in the same sequelae. Felsenreich et al. reported an incidence of 15% of Barrett's esophagus in patients 10 years after LSG [39]. Further studies will determine whether bariatric patients with preoperative GERD are better served with RYGB. In this case, preoperative functional evaluation to detect silent GERD will have a use in the choice of bariatric procedure. Post-operative endoscopies based strictly on GERD-symptoms certainly leaves out a significant proportion of patients at risk for Barrett's esophagus. Therefore, in our opinion, routine postoperative endoscopy should be performed in all LSG patients. Whether PPI should be administered to all patients with GERD, silent or symptomatic will remain a matter of debate until more data are available. Given the number of LSG performed to date, answers to those questions are urgently needed.

This study has several limitations. Even though data is collected prospectively, analysis is performed in a retrospective fashion. It only includes LSG patients, a selective group with a higher load of comorbidities as defined in this study and might therefore be biased. In this regard, the proportion of included men is higher than in other studies. Certainly, a dichotomous categorization of GERD with disregard to improvement or worsening does not reflect the reality. Further, a timepoint roughly 2 years after LSG is rather short and does not represent a long-term follow-up with possible changes later on.

Conclusion

LSG leads to a considerable rate of post-operative GERD. De novo-GERD consist of around half of preoperative silent GERD and completely de novo-GERD. Most patients with pre-operative silent GERD became symptomatic.

Compliance with ethical standards

Disclosures Yves Borbély, Esther Schaffner, Lara Zimmermann, Michael Huguenin, Gabriel Plitzko, Philipp Nett, and Dino Kröll declare that they have no conflict of interest or financial ties to disclose.

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