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REVIEW ARTICLE

Gastroesophageal reflux disease and morbid obesity: evaluation and treatment

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Abstract

Gastroesophageal reflux disease (GERD) is markedly increased among the obese population being recognized as one of the many obesity-related comorbidities. This concept should raise awareness, making physicians investigate more profoundly about this disease in this kind of patients. Currently, bariatric surgery is considered the gold standard treatment for morbid obesity. However, not all the operations are appropriate for the treatment of GERD and not all the patients are willing to receive bariatric surgery for the treatment of GERD. Even though sleeve gastrectomy has emerged as a suitable treatment option for morbid obesity, it has been related to development of de novo GERD or worsening the pre-existing one. Conversely, results after Roux-en-Y gastric bypass have been encouraging in this aspect, and it seems to be the best option for patients who suffer both diseases. Therefore, the presence of GERD should not be ignored at the time of deciding which type of surgery will be offered to the patient.

Keywords Gastroesophageal reflux disease · Barrett's esophagus · Obesity · Bariatric surgery · Sleeve gastrectomy · Roux-en-Y gastric bypass

Introduction

Gastroesophageal reflux disease (GERD) is markedly increased among the obese population. While the prevalence of GERD in non-obese persons ranges from 15 to 20%, the estimate is between 50 and 100% within the obese population. Even more worrisome becomes the fact that Barrett's esophagus (BE) can be diagnosed in approximately 1.2% of non-obese individuals, while the incidence rises up to 9% in obese patients [1].

GERD is currently recognized as one of the many obesity-related comorbidities. Patti et al. found that for each five-point increase in BMI, the DeMeester score was expected to increase by 3 U [2].

GERD is defined as the failure of the antireflux barrier that allows abnormal reflux of gastric contents into the esophagus. Considering GERD in the context of obesity, several factors such as increased intraabdominal pressure, reduced esophageal clearance, increased transient relaxations of the lower esophageal sphincter (LES), distorted anatomy of the gastroesophageal junction [e.g., hiatal hernia (HH)], and high fat-containing diet undoubtedly play a role in the genesis of this disease [3].

Having said that, it becomes evident that obesity is one of the major factors involved in the origin of GERD. Consequently, treating one of the main sources of the problem seems a reasonable approach to these patients.

Currently, bariatric surgery is considered the gold standard treatment for morbid obesity. However, not all the operations are suitable for the treatment of GERD and not all the patients are willing to receive bariatric surgery for the treatment of GERD.

Along this chapter, we are going to review diagnosis of GERD, possible complications, treatment options and their results in the obese population. Since Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG) accounts for 77% of all bariatric procedures performed in the US, we are going to focus our description on these two procedures [4].

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Methods

Literature review was performed through the National Library of Medicine's Pubmed Website, using the pertinent keywords.

Definition of obesity

Body Mass Index (BMI) calculation is the most utilized method to diagnose and classify obesity. BMI results from the ratio between weight (kg)/height (m^2). Obesity is categorized as follows:

Obesity Class I: BMI 30–34.9 kg/m^2
Obesity Class II: BMI 35–39.9 kg/m^2
Obesity Class III: BMI $\geq$ 40 kg/m^2

Diagnosis of GERD

Keeping in mind that obese patients are at increased risk of having GERD, physicians should be aware to avoid missing such diagnosis.

Special attention should be taken when diagnosing GERD, since a common mistake is to assume that the presence or absence of symptoms is enough to diagnose/rule out GERD.

Four tests are mandatory while evaluating these type of patients:

Symptomatic evaluation (using a validated questionnaire)
Barium swallow (BS)
Esophagogastroduodenoscopy (EGD)
Esophageal function tests (esophageal manometry, pH monitoring/impedance)

Each of them provides different and valuable information. We describe them in the following paragraphs.

Symptomatic evaluation

A validated symptom questionnaire should inquiry about frequency and severity of symptoms. Heartburn, regurgitation and dysphagia are known as typical symptoms. Among atypical symptoms cough, wheezing, belching, chest pain, nausea and hoarseness can be listed. However, the lack of symptoms' reliability to establish diagnosis of GERD should be always reminded [5]. This becomes more evident within the obese patients who usually do not complain of GERD symptoms [6].

Symptoms are important, however, to orient the physician and to set up a baseline for future follow-up.

Barium swallow (BS)

This test only provides anatomic information. It is worth of mention that the absence of HH does not exclude the diagnosis of GERD.

Esophagogastroduodenoscopy (EGD)

EGD offers information about the presence/absence of any mucosal injury such as esophagitis, Barrett's esophagus, strictures, ulcers or tumors. It should be noticed that the absence of esophagitis on EGD does not exclude the diagnosis of GERD [7].

Esophageal manometry (EM)

Esophageal motility and lower esophageal sphincter status can be studied through this test. EM is particularly important to rule out the presence of primary esophageal motility disorders. Missing diagnosis such achalasia, diffuse esophageal spasm or nutcracker esophagus would be extremely harmful to the patient [8]. On the other hand, EM allows identification of the LES location for correct placement of the 24 hs. pH monitoring catheter (5 cm above the upper border of the LES).

pH monitoring/multichannel intraluminal impedance-pH (MII-pH)

This is the only test that can provide precise information about the presence/absence of GERD. Either 24 hs. pH monitoring or Bravo® (wireless 48 hs. pH monitoring) will serve for this purpose. This study also establishes correlation between symptoms and episodes of reflux.

MII-pH monitoring can detect liquid, gas or mixed reflux in addition to acid, weakly acidic or weakly alkaline reflux. MII-pH monitoring can also record the direction of flow and the height of reflux. MII-pH is useful to study patients with persistent symptoms in spite of adequate treatment with proton pump inhibitors (PPI's) and normal findings on EGD.

Patient selection

The National Institute of Health NIH criteria [9] are still being used for patient selection although this might be changing with the introduction of the concept of metabolic surgery.

Results

Treatment options

Once the diagnosis of GERD has been established, the surgeon needs to face the following clinical scenario: he/she needs to treat a patient who has two simultaneous diseases: GERD and obesity. As it has been mentioned before, not every patient will accept to undergo surgery for obesity when his/her reason for consultation is GERD.

In the following paragraphs, different surgical techniques and their results will be described.

Antireflux surgery

One might think that if the patient is determined not to have any kind of bariatric surgery or his/her BMI is borderline; an antireflux procedure could be an option for that patient.

Several investigators published their experience in performing antireflux procedures in morbidly obese patients. Perez et al. reported their results in 224 patients who underwent either Nissen or Belsey fundoplication for the treatment of GERD. Subjects were stratified according to their BMI as follows: (a) normal weight, (b) overweight, and (c) obese. Recurrence rate was 4.5%, 8% and 31%, respectively ($p=0.001$ obese vs. normal weight and obese vs. overweight). They concluded that obesity negatively affects the outcome of antireflux surgery. They theorized that increased intraabdominal pressure in obese patients augments the usual wear and tear on the surgical repair and contributes to loosening of the crural closure and fundoplication [10].

Conversely, Winslow et al. published their experience on 505 patients undergoing antireflux procedures. Patients were also distributed according to their BMI in (a) normal weight, (b) overweight, and (c) obese. They found that even though operative time was longer for the obese group (115±42 vs. 137±55 min, $p=0.003$) and the operation was technically more demanding, complication rate, success rate and patient satisfaction were similar among groups. They concluded that obesity should not be a contraindication for antireflux surgery [11].

Sleeve gastrectomy (SG)

SG has been gaining ground during the last years, being recognized by the ASMBS as an acceptable option as a primary bariatric procedure [12]. Although this procedure has shown excellent results in terms of weight loss and resolution/improvement of comorbidities, its effect on GERD is still a matter of debate. Again, similar to the antireflux surgery phenomenon exposed above, expert opinions are conflicting. Some authors advocate that decreased LESP, distortion of the angle of His, diminished gastric volume and compliance, and increased intragastric pressure might play a detrimental role on the antireflux barrier. On the contrary, some others affirm that accelerated gastric emptying, weight loss, decrease in acid production, and removal of the gastric fundus, which is the source of transient lower esophageal sphincter relaxations might contribute to the improvement of GERD status.

Upon literature review, there are several publications concerning this subject. However, just a few of them studied patient's outcomes using objective tests.

Among the most popular publications, there are eight interesting studies supporting the fact that SG has a negative influence on GERD (Table 1). For instance, Himpens, Carter and Howard presented their different experiences, and they all observed that the percentage of patients with GERD like symptoms preoperatively was significantly increased after the operation [13–16]. However, these three studies based their conclusions only on preoperative and postoperative symptoms.

Braghetto et al. found that symptoms and esophagitis appeared the novo after SG; moreover, LES status changed from normotensive in 100% of patients to hypotensive in 85% of the patients postoperatively; pH studies were performed in a few patients and only after the operation, so no preoperative data were available for comparison [17, 18].

The interesting aspect of the next three publications remains in the objective data they offer. Burgerhart et al. reported their experience on 20 patients who underwent SG. They found that although there was no statistically significant difference among preop and postop symptoms, LESP dropped from 18.3 to 11 mmHg ($p=0.03$), that % of time pH < 4 increased from 4.1 to 12 ($p=0.004$) and the duration of the reflux episodes was longer [19]. Del Genio et al. published their data on 25 patients. While LESP remained stable, there was increased ineffective esophageal motility (IEM: ≥ 30% of the contractions, with amplitude < 30 mmHg), alteration of the esophageal clearance, and the DeMeester score escalated from 9 to 18.2 mmHg ($p=0.04$) [20]. Finally, we investigated outcomes on 14 patients undergoing SG. We found that LESP dropped from 17.1 to 12.4 mmHg ($p<0.05$), % time pH < 4 increased from 3.8 to 7.7 ($p<0.05$), and the DeMeester score rose from 12.6 to 28.4 ($p<0.05$) Analyzing GERD status after SG, we encountered that 5 (36%) patients had de novo GERD, in 3 (21%) GERD worsened, 1 (7%) remained with GERD (same status) and 5 (36%) patients remained without reflux [21].

In another study, we were able to analyze 109 patients before and after SG. We observed that the prevalence of esophagitis and HH increased significantly after LSG (20.1% vs. 33.9%; $p<0.05$), and (22% vs. 34.8%; $p<0.05$), respectively. GERD like symptoms also increased although the difference was not statistically significant (33% vs.

Table 1 Literature review: positive association between SG and postoperative GERD

Author	SG		SX		EGD		EM		pH	
	#pt	F/U	Preop (%)	Postop (%)	Preop	Postop	Preop	Postop	Preop	Postop
Braghetto et al. [17]	20	6 mo	–	–	–	–	LESP: 14.2	LESP: 10.5 Hypot.: 85% pts	N/A	N/A
Braghetto et al. [18]	167	N/A	0	27.5	WNL	15% pt esophagitis	WNL	Hypot.: 74% pts	N/A	ABN: 65.3% pt
Himpens et al. [13, 14]	53	6 yrs	3.3	23	–	–	–	–	–	–
Carter et al. [15]	176	–	34.6	47.2						
Howard et al. [16]	28		0	22						
Burgerhart et al. [19]	20	3 mo	–	–	–	–	LESP: 18.3	LESP: 11	% time pH < 4: 4.1	% time pH < 4: 12
Del Genio et al. [20]	25	13 mo	–	–	–	–	LESP: 21	LESP 22	DeMeester 9	DeMeester 18
Gorodner et al. [21]	14	14 mo	–	–	–	–	LESP: 17.1	LESP: 12.4	DeMeester 12.6	DeMeester 28.4

SG sleeve gastrectomy, *SX* symptoms, *EGD* esophagogastroduodenoscopy, *EM* esophageal manometry, *pH* pH monitoring, *pt* patients, *mo* months, *yrs* years, *WNL* within normal limits, *ABN* abnormal, *LESP* lower esophageal sphincter pressure, *Hypot* hypotensive

44%) (p = NS). De novo esophagitis was present in 25% of patients, HH in 16% and symptoms in 35% of patients. We concluded that the prevalence of esophagitis, HH and GERD like symptoms was increased after surgery. Still, it must be mentioned that postoperative esophagitis was non-severe in the majority of patients and symptoms were mild, sporadic and were relieved by proton pump inhibitors (PPI's) [22].

Some other authors went even further in their conclusions, based on their alarming findings. Genco et al. studied 110 patients undergoing SG. They performed EGD preoperatively and at 5-year follow-up. EGD findings revealed that 19 (17%) patients had de novo Barrett's esophagus. They suggested that postoperative surveillance should be mandatory regardless the presence/absence of GERD symptoms [23]. Moreover, Csendes et al. studied 231 patients before and 1 year after SG. They observed that 3 (1.3%) patients had developed de novo Barrett's esophagus after the operation [24]. In our experience (data non-published) 2 (0.4%) out of 433 patients evolved to the same unfortunate situation.

Conversely, the following four authors rejected the association between LSG and GERD (Table 2). Melissas et al. described 50% resolution/improvement of symptoms, although they found 8.7% de novo GERD symptoms. They attributed these positive results to patients' weight loss and accelerated gastric emptying [25]. Likewise, Weiner et al. showed even better results, with 43% improvement and 57% resolution of symptoms [26]. Petersen et al. performed preoperative and postop manometry on SG patients, although they did not perform pH monitoring. They found that LESP significantly increased from 8.4 to 21 mmHg at 8-month follow-up [27].

Again, Rebecchi et al. were the only authors reporting objective data. In this study, patients were split based on their preoperative GERD status into group A (pathologic, n = 28) and group B (normal, n = 37). Symptoms improved in group A, decreasing their symptom score from 53.1 to 13.1 ($p < 0.001$). The DeMeester score and % time pH < 4 decreased in group A as follows: DeMeester score went from 39.5 to 10.6 ($p < 0.001$) and % pH < 4 from 10.2 to 4.2 $p < 0.001$). De novo GERD occurred in 5.4% group B. No significant changes in LESP and esophageal peristalsis amplitude were found in both groups. They concluded that SG improves symptoms and controls reflux in most morbidly obese patients with preoperative GERD and that the occurrence of "de novo" reflux is uncommon [28].

Roux-en-Y gastric bypass

RYGB has been proposed as the best treatment option for obesity combined with GERD. The anatomic modifications including the creation of a small gastric pouch, the exclusion of the fundus and most part of the body, where parietal cells are concentrated, and the Roux-en-Y configuration result in

Table 2 Literature review: negative association between SD and postoperative GERD

Author	SG		SX		EGD	EM		pH	
	#pt	F/U	Preop	Postop		Preop	Postop	Preop	Postop
Melissas et al. [25]	23	N/A	N/A	50% pt resolved/improved 8.7% de novo GERD	N/A	–	–	–	–
Weiner et al. [26]	120	2 yrs	35%	43% improvement 57% resolution		–	–	–	–
Petersen et al. [27]	17	6 days	45%	N/A		LESP: 21	LESP: 24	–	–
Petersen et al. [27]	20	8 mo				LESP: 8.4	LESP: 11	–	–
Rebecchi et al. [28]	65	2 yrs	Score 53	Score 13	–	–	–	DeMeester 39	DeMeester 10

SG sleeve gastrectomy, *SX* symptoms, *EGD* esophagogastroduodenoscopy, *EM* esophageal manometry, *pH* pH monitoring, *pt* patients, *mo* months, *yrs* years, *LESP* lower esophageal sphincter pressure, *Hypot* hypotensive

decreased gastroesophageal reflux, independently of weight loss occurrence. Several studies confirmed this observation [29, 30].

After reviewing the literature, we found that in fact, the majority of authors concur that RYGB is the best treatment option for obese patients with GERD. However, some studies demonstrated that de novo reflux esophagitis might occur after RYGB, and that there is still some acid secretion in the gastric pouch despite of the reduction in the quantity of parietal cells [31].

Some of the studies lack of objective data, but many of them base their conclusions on objective evaluations. Next, we describe some of their findings.

Frezza et al. published their experience on 152 obese patients with diagnosis of GERD undergoing RYGB. They observed that > 90% of patients reported either resolution or improvement of their symptoms. The use of proton pump inhibitors (PPI's) decreased from 44 to 9% ($p < 0.01$), while the use of H2 blocker therapy decreased from 60 to 10% ($p < 0.001$). Again, even though these results are encouraging, they were based on symptomatic assessment and use of medication, but objective tests were not performed after the operation [32].

Instead, Mejia-Rivas et al. investigated the effect of RYGB on GERD on 20 patients using objective measurements. Similar to Frezza, they observed that symptoms were resolved in 90% of the cases. On EM, LES pressure was slightly increased postoperatively, being 18 ± 11 and 20.1 ± 5.6 mmHg before and after the RYGBP, respectively ($p = \text{NS}$). On pH monitoring, the DeMeester score significantly decrease from 48.3 to 7.7 ($p < 0.001$). Only one patient (5%) had abnormal esophageal acid exposure with heartburn as the main symptom [33].

Furthermore, Csendes et al. performed preoperative and postoperative EGDs on 130 patients undergoing RYGB. The average number of postoperative EGDs was 3.6 per patient with a mean follow-up of 92 months. Before the surgery distal erosive esophagitis was present in 23.8% of patients. The consecutive EGDs showed that esophagitis healed in 93% of these patients. However, three patients (3%) with normal preoperative EGD presented de novo erosive esophagitis 66 months after surgery [34].

Madalosso et al. studied the effect of banded RYGB on 53 patients also through objective evaluations preoperatively, and at 6 and 39 months postoperatively. They observed that the prevalence of typical reflux syndrome was significantly reduced from 58% preoperatively to 9% during last follow up ($p < 0.001$). On EGD, the prevalence of reflux esophagitis decreased from 45% preoperatively, to 19% in the last follow up ($p = 0.001$). However, de novo reflux esophagitis appeared in 17% of the patients at 6 months, but this percentage was reduced to 7% at 39-month follow-up. They believed that disruption of integrity of the esophageal sphincter due to dissection of the phrenoesophageal membrane for stapler placement close to the angle of His might play a role. Neither esophageal stricture nor Barrett esophagus was observed at any evaluation. DeMeester score decreased from 28.6 preoperatively to 1.2 at 39-month follow-up ($p < 0.001$). They assumed that reduction in abdominal pressure, improvement in gastric emptying after weight loss, and reduced gastric output might explain these findings [35].

Roux-en-Y gastric bypass and Barrett's esophagus

A special section should be dedicated to this delicate situation, since BE appears as a consequence of GERD. Obese patients who have been diagnosed with BE might represent a challenge.

Surely, if the principle for the treatment of BE is stopping the reflux, offering a RYGB might be the best option for these patients. RYGB would diminish the amount of acid that could be refluxed into the esophagus by creation of a small gastric pouch while the Roux-en-Y configuration would divert the bile avoiding bile reflux.

There are a few reports concerning this subject.

For instance, Houghton et al. reported their experience on five RYGB patients who had long segment Barrett's esophagus (LSBE). Two out of five patients had complete regression of BE, while two other patients showed regression of dysplasia to intestinal metaplasia (BE). This means that 4/5 (80%) had either total or partial regression of their BE after RYGB [36].

Braghetto et al. published their series of 21 patients with LSBE who underwent laparoscopic resectional RYGB (LRRYGB), showing regression of BE in 61.9% of cases. They concluded that this should be the operation of choice for obese patients with LSBE [1].

Finally, we investigated the effect of RYGB on BE in 11 patients. Esophageal biopsy demonstrated remission in 4 (36%) cases, three short segment BE (SSBE) and one long segment BE (LSBE). One patient was indefinite for dysplasia and remained the same after the operation. We concluded that RYGB was a suitable treatment option for obese patients with BE. Although BE persisted in the remaining patients, no progression to dysplasia was observed [37].

Conclusions

Literature review indicates that the effect of SG on GERD is still a matter of debate, mainly because of the heterogeneity of the published data. Although SG has been accepted by the ASMBS as a primary bariatric procedure due to its encouraging results in terms of weight loss and resolution/improvement of comorbidities, patients should be warned that they might need PPI's after the operation. They should also be informed that symptoms are not reliable to evaluate the presence/absence of GERD so they might need a closer follow-up, including EGD surveillance due to the risk of developing Barrett's esophagus. We recommend to objectively rule out the presence of GERD in SG candidates, since this procedure might worsen the status of the disease.

It seems that RYGB remains the treatment of choice for obese patients with GERD, since it has demonstrated to objectively improve/resolve the status of the disease in the majority of patients. However, some reports make physicians aware that reflux esophagitis might remain present in postsurgical RYGB patients and that it might even appear de novo in a few percentage of cases. Lastly, the most interesting feature that RYGB can offer is the possibility of regression or stopping the progression of BE.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Research involving human participants and/or animals Ethical approval: for this type of study formal consent is not required.

Informed consent As a review article, no informed consent was obtained.

References

1. Braghetto I, Korn O, Csendes A, Gutiérrez L, Valladares H, Chacon M (2012) Laparoscopic treatment of obese patients with gastroesophageal reflux disease and Barrett's esophagus: a prospective study. Obes Surg 22(5):764–772
2. Herbella FA, Sweet MP, Tesesco P, Nipomnick I, Patti MG (2007) Gastroesophageal reflux disease and obesity. Pathophysiology and implications for treatment. J Gastrointest Surg 11:286–290
3. Suter M, Dorta G, Giusti V, Calmes JM (2010) Gastro-esophageal reflux and esophageal motility disorders in morbidly obese patients. Obes Surg 14:959–966
4. Estimate of Bariatric Surgery Numbers, 2011–2015 (2016). https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers. Accessed 4 Mar 2018
5. Galvani C, Fisichella M, Gorodner MV, Perretta S, Patti MG (2003) Symptoms are poor indicators of reflux status after fundoplication for gastroesophageal reflux disease. Arch Surg 138:514–519
6. Hong D, Kamath M, Wang S, Tabet J, Tougas G, Anvari M (2002) Assessment of the afferent vagal nerve in patients with gastroesophageal reflux. Surg Endosc 16:1042–1045
7. Jobe BA, Ritcher JE, Hoppo T, Peters JH, Bell R (2013) Preoperative diagnostic evidence and experience-based consensus of the esophageal diagnostic advisory panel. J Am Coll Surg 217:586–597
8. Koppman JS, Poggi L, Szomstein S, Ukleja A, Botoman A, Rosenthal R (2007) Esophageal motility disorders in the morbidly obese population. Surg Endosc 21:761–764
9. Gastrointestinal surgery for severe obesity (1992) National Institutes of Health consensus development conference statement. Am J Clin Nutr 55(2 Suppl):615S–619S
10. Perez AR, Moncure AC, Rattner DW (2001) Obesity adversely affects the outcome of antireflux operations. Surg Endosc 15(9):986–989
11. Winslow ER, Frisella MM, Soper NJ, Klingensmith ME (2003) Obesity does not adversely affect the outcome of laparoscopic antireflux surgery (LARS). Surg Endosc 17(12):2003–2011
12. Clinical Issues Committee of the American Society for Metabolic and Bariatric Surgery (2010) Updated position statement on sleeve gastrectomy as a bariatric procedure. Surg Obes Relat Dis 6(1):1–5
13. Himpens J, Dobbeleir J, Peeters G (2010) Long term results of laparoscopic sleeve gastrectomy for obesity. Ann Surg 252(2):319–324
14. Himpens J, Dapri G, Cadière GB (2006) A prospective randomized study between laparoscopic gastric banding and laparoscopic isolated sleeve gastrectomy: results after 1 and 3 years. Obes Surg 16:1450–1456
15. Carter P, LeBlanc KA, Hausmann MG, Kleinpeter KP, deBarros SN et al (2011) Association between gastroesophageal reflux disease and laparoscopic sleeve gastrectomy. Surg Obes Relat Dis 7:569–574
16. Howard DD, Caban AM, Cendan CJ, Ben-David K (2011) Gastroesophageal reflux after sleeve gastrectomy in morbidly obese patients. Surg Obes Relat Dis 7:709–713

17. Braghetto I, Lanzarini E, Korn O, Valladares H, Molina JC et al (2010) Manometric changes of the lower esophageal sphincter after sleeve gastrectomy in obese patients. Obes Surg 20:357–362
18. Braghetto I, Csendes A, Korn O, Valladares H, Gonzalez P, Henriquez A (2010) Gastroesophageal reflux disease after sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech 20(3):148–153
19. Burgerhart JS, Charlotte AI, Schotborgh Schoon EJ, Smulders JF, van de Meeberg PC, Siersema PD, Smout AJ (2014) Effect of sleeve gastrectomy on gastroesophageal reflux. Obes Surg 24:1436
20. Del Genio G, Tolone S, Limongelli P, Brusciano L, D'Alessandro A, Docimo G, Rossetti G, Silecchia G, Ianelli A, del Genio A, del Genio F, Docimo L (2014) Sleeve gastrectomy and development of "de novo" gastroesophageal reflux. Obes Surg 24(1):71–77
21. Gorodner V, Buxhoeveden R, Clemente G, Sole L, Caro L, Grigaites A (2015) Does laparoscopic sleeve gastrectomy have any influence on gastroesophageal reflux disease? Preliminary results. Surg Endosc 29(7):1760–1768
22. Viscido G, Gorodner V, Signorini F, Navarro L, Obeide L, Moser F (2018) Laparoscopic sleeve gastrectomy: endoscopic findings and gastroesophageal reflux symptoms at 18-months follow-up. J Laparoendosc Adv Surg Tech A 28(1):71–77
23. Genco A, Soricelli E, Casella G, Maselli R, Catagneto-Gissey L, Di Lorenzo N, Basso N (2017) Gastroesophageal reflux disease and Barrett's esophagus after laparoscopic sleeve gastrectomy: a possible, underestimated long-term complication. Surg Obes Relat Dis 13(4):568–574
24. Braghetto I, Csendes A (2016) Prevalence of Barrett's esophagus in bariatric patients undergoing sleeve gastrectomy. Obes Surg 26(4):710–714
25. Melissas J, Koukouraki S, Askoxylakis J, Stathaki M, Daskalakis M et al (2007) Sleeve gastrectomy—a restrictive procedure? Obes Surg 17:57–62
26. Weiner R, Weiner S, Pomhoff I, Jacobi C, Makarewicz W, Weigand G (2007) Laparoscopic sleeve gastrectomy—influence of sleeve size and resected gastric volume. Obes Surg 17:1297–1305
27. Petersen W, Meile T, Küper MA, Zdichavsky M, Königsrainer A, Schneider JH (2012) Functional importance of laparoscopic sleeve gastrectomy for the lower esophageal sphincter in patients with morbid obesity. Obes Surg 22:360–366
28. Rebecchi F, Allaix ME, Giaccone C, Ugliono E, Morino M (2014) Gastroesophageal reflux disease and laparoscopic sleeve gastrectomy: a physiopathologic evaluation. Ann Surg 260(5):909–914 **(discussion 914–5)**
29. Madalosso CA, Gurski RR, Callegari-Jacques SM, Navarini D, Thiesen V, Fornari F (2010) The impact of gastric bypass on gastroesophageal reflux disease in patients with morbid obesity: a prospective study based on the Montreal Consensus. Ann Surg 251:244–248
30. Nelson LG, Gonzalez R, Haines K, Gallagher SF, Murr MM (2005) Amelioration of gastroesophageal reflux symptoms following Roux-en-Y gastric bypass for clinically significant obesity. Am Surg 71:950–953 **(discussion 953–954)**
31. Hedberg J, Hedenström H, Sundbom M (2011) Wireless pH-metry at the gastrojejunostomy after Roux-en-Y gastric bypass: a novel use of the BRAVO™ system. Surg Endosc 25(7):2302–2307
32. Frezza EE, Iframuddin S, Gourash W, Rakitt T, Kingston A, Luketich J, Schauer P (2002) Symptomatic improvement in gastroesophageal reflux disease (GERD) following laparoscopic Roux-en-Y gastric bypass. Surg Endosc 16(7):1027–1031
33. Mejia-Rivas MA, Herrera-Lopez A, Hernandez-Calleros J, Herrera MF, Valdovinos MA (2008) Gastroesophageal reflux disease in morbid obesity: the effect of Roux-en-Y gastric bypass. Obes Surg 18(10):1217–1224
34. Csendes A, Smok G, Burgos AM, Canobra M (2012) Prospective sequential endoscopic and histologic studies of the gastric pouch in 130 morbidly obese patients submitted to Roux-en-Y gastric bypass. Arq Bras Cir Dig 25(4):245–249
35. Madalosso CA, Gurski RR, Callegari-Jacques SM, Navarini D, Mazzini G, Pereira Mda S (2016) The impact of gastric bypass on gastroesophageal reflux disease in morbidly obese patients. Ann Surg 263(1):110–116
36. Houghton SG, Romero Y, Sarr MG (2008) Effect of Roux-en-Y gastric bypass in obese patients with Barrett's esophagus: attempts to eliminate duodenogastric reflux. Surg Obes Relat Dis 4:1–5
37. Gorodner V, Buxhoeveden R, Clemente G, Sanchez C, Caro L, Grigaites A (2017) Barrett's esophagus after Roux-En-Y gastric bypass: does regression occur? Surg Endosc 31(4):1849–1854