Perioperative screening, management, and surveillance of Barrett's esophagus in bariatric surgical patients

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Short title: Barrett's Esophagus and Bariatric Surgery

Abstract

Obesity is a strong risk factor for Barrett's esophagus (BE), the only proven precursor lesion to EAC. Bariatric surgery is currently the only reliable treatment that achieves of long-term sustained weight loss, however, it can markedly affect the development of *de novo* BE, and the progression or regression of existing BE. Bariatric procedures may also have implications on future surgical management of any consequent EAC. In this review, we examine current evidence and published guidelines for BE in bariatric surgery. Current screening practices before bariatric surgery vary substantially, with conflicting recommendations from bariatric societies. If diagnosed, the presence of BE may alter the type of bariatric procedure. A selective screening approach prevents unnecessary endoscopy, however, there is poor symptom correlation with disease. Studies suggest that sleeve gastrectomy predisposes patients to gastroesophageal reflux and *de novo* BE. Conversely, Roux-en-Y gastric bypass is associated with decreased reflux and potential improvement or resolution of BE. There are currently no guidelines addressing surveillance for BE following bariatric surgery.

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BE is an important consideration in the management of bariatric surgical patients. Evidence-based recommendations are required to guide procedure selection and postoperative surveillance.

Keywords: Barrett's esophagus; bariatric surgery; endoscopy; esophagus; stomach

Introduction

Worldwide rates of obesity have tripled over the last few decades, with over 13% of the adult population now classified as obese (body mass index (BMI) over 30kg/m^2).[1] Bariatric surgery is currently the only reliable means of achieving substantial and sustained weight loss accompanied by remission and reduced incidence of metabolic comorbidities in morbidly obese individuals [2,3].

Esophageal pathology is relatively common in obesity,[4,5] and potentially exacerbated by bariatric surgical procedures. This includes Barrett's esophagus (BE), the only known precursor lesion of esophageal adenocarcinoma (EAC). There are multiple mechanisms, both mechanical and metabolic, for this association in obesity. The incidence of EAC in Western society has increased up to five-fold in the last few decades,[6] closely mirroring the rising rates of obesity.

It is important to recognize BE in the bariatric surgical population. Preoperative endoscopy may be an opportune time to screen an already at-risk population, and its detection can suggest a change of surgical strategy. Second, bariatric surgical procedures can impact gastroesophageal reflux (GER) and, therefore, theoretically affect the development of *de novo* BE, or progression and regression of existing BE. Finally, bariatric surgery can significantly impact future curative treatments for esophageal adenocarcinoma (EC) that may develop from BE.

In this review, we discuss the link between BE, obesity, and bariatric surgery, and review the evidence around perioperative screening and surveillance.

BE and obesity

BE is a premalignant condition characterized by the conversion of the normal esophageal squamous epithelium to the metaplastic columnar epithelium containing goblet cells.[7] This is thought to be in response to prolonged exposure to acid reflux. BE can progress from metaplasia to dysplasia, and subsequently EAC.

Central adiposity and high BMI are risk factors for BE. A meta-analysis by Singh *et al.* showed a significant increase in odds of BE with central adiposity (OR = 1.98; 95% CI: 1.52–2.57), with a strong dose–response relationship.[8] This association remains significant after controlling for gastroesophageal reflux disease (GERD). Higher BMI is also significantly correlated with increased length of BE (r = 0.25, P < 0.0001) and greater rates of long-segment BE.[9]

Various mechanisms have been implicated in the development of BE in obesity. These can be divided into mechanical, metabolic, and dietary factors.[5]

Mechanically, obesity increases intragastric pressures and disrupts the antireflux mechanisms, both of which encourage acid reflux into the esophagus. High BMI and waist circumference are independently associated with dysfunction of the GE function, including separation of the diaphragmatic hiatus and increased frequency of lower esophageal sphincter relaxation,[10,11] allowing passage of the refluxate into the esophagus. Intragastric pressures from visceral abdominal fat further exacerbate acid reflux.[12]

Metabolic factors are also important mechanisms in the development of BE and neoplasia. Overexpansion of adipose tissue in obesity produces a state of low-grade systemic inflammation, owing to changes in immune profile to a proinflammatory state and over-expression of inflammatory cytokines, such as tumor necrosis factor-α, interleukin (IL)-1β, and IL-6.[13] The paracrine and endocrine profile of adipose tissue changes with increased leptin and decreased adiponectin. Increasing lipolysis results in the release of saturated free fatty acids, and excess adiposity results in ectopic fat storage in depots, such as the liver, pancreas, and muscle, leading to organ dysfunction and insulin resistance.[14] These changes in leptin, adiponectin, insulin resistance, and systemic inflammation have all been linked to the development and progression of BE.[5]

Various dietary factors have been associated with the development of BE, many of which are associated with obesity. Meta-analyses show that low dietary fiber intake is associated with an increased risk of BE and EAC [15]. Composition of dietary fats, towards high animal fat intake, has been shown to change bile acid composition and increase rates of metaplasia, dysplasia, and adenocarcinoma (AC) in animal models [16]. Alternatively, a high intake of beta-carotene (for example, from carrots, dark leafy greens, and cantaloupe) is protective from dysplastic BE [17].

Bariatric surgery and reflux disease

GERD refers to pathological, retrograde passage of gastric and/or biliopancreatic contents into the esophagus, such that symptoms or pathology occurs.[18] In the general population, it is one of the most common diagnoses during endoscopy, with a prevalence of between 10 and 20%. This prevalence increases with obesity and may be up to 60%.[19]

Trends in bariatric surgery have changed dramatically over the last 10–20 years. In this time, sleeve gastrectomy (SG) has increased from 3.0% in 2008 to 54% of all cases performed in the United States in 2014. Laparoscopic adjustable gastric banding (LAGB) has fallen from 29% to 1% of bariatric cases, and Roux-en-Y gastric bypass (RYGB) decreased from 52% to 32%.[20] Randomized control trials have shown similar weight loss outcomes for SG and RYGB.[21,22] These trends are likely due to a combination of reasons, such as procedural complexity, insurance coverage, complication profile, and surgeon/patient preferences.

Bariatric surgery can have profound effects on the mechanisms promoting or alleviating GERD. RYGB is a popular choice for obese patients with significant reflux, with studies consistently showing symptomatic relief, reduction in acid suppressor medication, improvement in acid exposure, and

esophagitis.[23] Mechanistically, RYGB creates a small low-pressure gastric pouch with few acid-producing parietal cells and a long Roux limb that prevents reflux of duodenal contents into the esophagus. Prevention of bile reflux is important, given the link between BE and the exposure of the esophageal mucosa to the biliopancreatic fluid.[24] Studies on LAGB have conflicting results, with both symptomatic relief and reduction in medication, as well as studies reporting exacerbation of reflux.[23]

The majority of studies that have assessed reflux after SG report an increase in GERD symptoms and objective evidence of acid reflux, such as erosive esophagitis.[25] Proposed mechanisms that exacerbate reflux include an increase in intragastric pressures, intrathoracic sleeve migration, fundal dilatation, delayed gastric emptying, and esophageal motility disorders.[23] However, there are some studies that have shown the opposite, with improvement in GERD and the esophagitis.[26–28] This could be due to faster gastric emptying, alterations in the gastrointestinal (GI) hormonal milieu, decreased acid secretion, and postoperative weight loss. Technical factors, such as concomitant hiatus hernia repair, may also contribute to differences between studies.

With the link between obesity, BE and EAC well established, diagnosing BE in bariatric surgical patients is important. Understanding the effect of bariatric procedures on BE is vital for tailoring surgery to individuals and counseling patients on the risk. The presence of BE in patients undergoing bariatric surgery is particularly important, as bariatric procedures can significantly influence surgical options for curative management of EAC.

The primary aim of this review was to summarize the current evidence on:

1. Screening for BE before bariatric surgery;
2. The effects of bariatric surgery on BE;
3. Surveillance for patients who have/develop BE following bariatric surgery.

Literature search

We performed a comprehensive literature search to identify studies reporting any experiences or outcomes with BE and bariatric surgery. We searched MEDLINE, PubMed, and the Cochrane Library (until January 2020). The search items used were derivatives of BE and bariatric surgery. Original research, clinical trials, review articles, and meta-analyses were all included.

Screening for BE before bariatric surgery

The role of routine endoscopy before bariatric surgery remains controversial. There is a wide variety of practices, with many centers performing routine endoscopy while others practice selective endoscopy, often based on symptoms.[29] This is reflected by varying recommendations among surgical societies (Table 1). The European Association for Endoscopic Surgery (EAES) recommends routine upper GI (UGI) endoscopy or upper GI series for all bariatric patients.[30] By contrast, the American Society for Metabolic and Bariatric Surgery (ASMBS) and the American Society for Gastrointestinal Endoscopy (ASGE) both suggest an individualized approach, with investigations performed for those with clinically significant symptoms.[31,32] The evidence underlying these recommendations are somewhat weak.

Advocates of selective endoscopy argue that clinically important findings are not common in the bariatric population, and therefore endoscopy can be offered based on the symptoms and the risk. Current reports suggest that the proportion of patients where endoscopy alters surgical approach or has a clinical impact varies from <1% to 24.6% of patients.[33–37] There is significant heterogeneity between studies around the definition of "clinically significant" impact, with some defining this as change or delay in surgery, while others include the change in medical management as significant.

Many studies have reported a lack of correlation between symptoms and detection of BE (Table 2).[36,38–41] Up to 60% of patients diagnosed with BE on preoperative endoscopy are asymptomatic. Saarinen *et al.*[36] examined the endoscopy results of 1275 bariatric patients, and reported a rate of BE of 3.7% (*n* = 47), of whom 28 (59.6%) were asymptomatic. Similarly, D'Silva *et al*.[41] studied 675 preoperative endoscopies and found that 60% of those findings consistent with GERD, including BE and reflux esophagitis, were asymptomatic. The lack of symptoms together with the lack of accurate noninvasive tests is likely to decrease the efficacy of a selective approach to endoscopy before bariatric surgery.

Endoscopy has the advantage of detecting mucosal lesions, such as BE or early AC, which are often imperceptible or too subtle to be diagnosed by other means. In individual studies, BE was found in <1% to 15% of preoperative endoscopy (Table 2).[36,37,39–48] Two systematic reviews and meta-analyses have examined the utility of preoperative endoscopy.[38,49] Parikh *et al.* reported an overall incidence of BE of 1% (45 of 4511 patients), with EAC found in 0.9% (4 of 4511). Bennett *et al.* combined the findings of 19 studies with 5802 patients, finding an incidence of BE of 2.1% and esophageal cancer in 0.2%. Data on how many patients were symptomatic is lacking, as only two studies in the meta-analysis made the distinction between those with and without symptoms. Both these systematic reviews have advocated for selective endoscopy based on relatively low numbers.

Given the number of bariatric operations performed annually worldwide, and the implications for BE screening and early cancer detection, over 2% of bariatric patients with BE is a clinically significant number. The clinical significance is further increased given that majority of bariatric patients are relatively young, which leads to an increased lifelong risk of cancer.

Additionally, bariatric surgery has implications for curative surgical procedures and conduit reconstruction. Resectional procedures, such as SG or resectional RYGB, eliminate the use of the stomach as a future reconstructive option during esophageal resection procedures. Reconstruction with other conduits, such as colon or small bowel, are technically more demanding and have an increased risk.[50] Additionally, any gastric procedure may cause scarring or stricturing, increasing the chance of complications if future surgery is required.[51] In this regard, excluding preexisting BE is vital before committing to bariatric surgery.

Effect of bariatric surgery on BE

SG

Current published literature studying BE in SG patients strongly suggests an increased rate of *de novo* BE after SG. A recent systematic review by Yeung *et al.* (2020) reported on the impact of SG on

reflux-related measures, including symptomatic reflux, erosive esophagitis, and BE[25] (Table 3). Five studies examined the incidence of BE after SG. Over a follow-up period of 24 months or more, the rate of *de novo* BE was 8% (95% CI: 4–13%, $P < 0.0001$). This review showed significant increases in *de novo* reflux (rate of 20%), erosive esophagitis (28%), and PPI use (36%). Furthermore, 4% of patients underwent conversion to RYGB for the treatment of severe reflux. All studies were retrospective or cohort studies with no randomized control trials, and there was a large amount of heterogeneity in all analyses.

Table 4 shows a summary of recent individual studies examining the incidence of BE after SG. Participant numbers range from 43 to 231 and a follow-up varied from 58 months to over 10 years. Rates of *de novo* BE ranged from 1.2% up to 18.8%, with between 5 to 10 years of a follow-up.

Roux-en-Y gastric bypass

RYGB appears to be beneficial in the setting of BE. A systematic review by Adil *et al.* in 2019 reported on 117 patients with biopsy-proven preoperative BE and endoscopic follow-up at over 1 year after RYGB surgery. Meta-analysis showed significant regression in BE after RYGB, with a risk difference of -0.56 (95% CI: -0.69 to -0.43, $P < 0.001$). This corresponded to 64 participants (54.7%) showing histological regression, with two (1.7%) showing progression. This effect was seen in both short- and long-segment BE (risk difference of -0.51 and -0.46, respectively, $P < 0.001$ for both).[52,53] Heterogeneity between studies was moderate, with some publication bias owing to a small study size (Table 3).

Results of individual studies are seen in Table 5. All studies report improvement in BE after RYGB. Felsenreich *et al.* studied 10 patients who underwent RYGB as a revisional procedure for SG after a diagnosis of BE. They reported 80% remission, and objective improvements in acid exposure (36.8% to 2.8% after a 24-h acid exposure) and DeMeester score (100 to 16.3). Individual studies suggest better rates of resolution in short- versus long-segment BE [52,53]. The effects of weight loss quantity on the resolution of BE has not previously been studied, but evidence suggests that greater weight loss may contribute to the resolution of BE.[9] These studies further support the role of RYGB as a combined bariatric and antireflux procedure.

Many have advocated for RYGB as a revision procedure for patients who experience significant GERD post-SG. Felsenreich *et al.* (2019) studied 10 patients who underwent RYGB after SG due to severe reflux and BE.[54] At a mean follow-up time of 33.4 months, they noted a significant decrease in acid exposure (36.8% to 3.8% 24-h acid exposure) and drop in DeMeester score (100 to 16.3). Furthermore, BE resolved in eight patients, including in one patient with small areas of dysplasia on preoperative endoscopy.

Other procedures

Few studies have examined the effect of other bariatric procedures on BE. There is only one case report of BE,[55] and reports within larger outcomes studies of BE observed[56,57] after LAGB. Due to paucity in the literature, the specific incidence of BE after gastric banding is not known.

We could not find studies specifically addressing the incidence of BE after other procedures, including single anastomosis gastric bypass, single anastomosis duodenal bypass, or biliopancreatic diversion.

Surveillance for *de novo* BE after bariatric surgery

There are currently no society guidelines specifically addressing potential *de novo* BE and surveillance postbariatric surgery. Current guidelines focus on known risk factors for BE, including chronic GERD, older age, male gender, central obesity, Caucasian race, and tobacco use.[59]

The Interdisciplinary European Guidelines on Metabolic and Bariatric Surgery encourage "regular lifelong qualified surveillance" for all patients after bariatric procedures, including clinical, metabolic, and nutritional assessment. In particular, persistent severe GI symptoms should be followed up with endoscopy and/or CT as a first diagnostic option.[60] Similarly, the American Society of Gastrointestinal Endoscopy (ASGE) recommends endoscopy as the first-line diagnostic study for bariatric patients with abdominal pain, nausea, or vomiting.[32] These recommendations prevent unnecessary delay in the diagnosis of significant pathology due to erroneously attributing them to a normal postoperative course after a bariatric procedure. The emphasis on symptoms, however, is likely to miss most cases of BE. No routine endoscopic follow-up schedule has been recommended by any society.

There is no consensus on screening and surveillance for BE in the general population, and little evidence to support its use. Most societies have used a cut-off prevalence of BE of 10% to recommend screening [61,62]. Several risk factors have been identified, including the history of GERD, male gender, white race, age >50 years, smoking, obesity, and family history. The American Society of Gastrointestinal Endoscopy (ASGE) recognizes high-, moderate-, and low-risk groups. High-risk populations include those with a family history of EAC or BE, where screening is recommended. The moderate-risk group includes patients with GERD and another additional risk factor, including age >50, obesity/central adiposity, history of smoking, or male gender. The screening was not recommended for low-risk populations. When applied to the bariatric population, those with symptoms of GERD will be of "moderate-risk" and, therefore, warrant screening and surveillance.

One concerning feature of acid reflux and BE after SG is the lack of correlation between endoscopic findings and reported symptoms.[25,63,64] Soricelli *et al.* studied the association between symptoms of GER and endoscopic esophageal lesions after SG in 249 patients over a mean follow-up of 66 months.[64] They found no significant correlation between the symptoms of GERD and the severity of esophagitis or the presence of BE. In a study on GERD after SG, Genco *et al.* reported highest symptomatic reflux scores for Grade A and B esophagitis (visual analogue scale (VAS) of 3.1–3.4), lower reflux scores for Grade C and D esophagitis (VAS = 2.4–2.8), and the lowest reflux scores in patients found to have BE (VAS = 1.3).[63] These studies highlight the difficulties in triaging patients with post-SG for selective gastroscopy.

Given current evidence suggesting the development of reflux and *de novo* BE in sleeve patients, it may be prudent to advocate for surveillance of patients 5–10 years after SG, or sooner if they are symptomatic.

Summary

Preoperative screening

GERD and BE are often asymptomatic. Given the implications of some bariatric procedures on the development of reflux and BE and also on future cancer treatment, endoscopy plays a vital role in preoperative assessment. Other modalities, such as UGI series, are of little utility for the detection of GERD or mucosal abnormality. If significant pathology, such as esophagitis or BE is found, the patient and surgeon should strongly consider RYGB over other procedures.

Effect of bariatric surgery

Current evidence indicates that SG predisposes to *de novo* GERD and BE. On the other hand, RYGB is associated with resolution or reduction in length of BE, as well as reduced GERD. Therefore, RYGB is ideal for those with preoperative BE or GERD. Unless there is a specific indication for resection, the bypassed stomach should be left *in situ*, as it may be used as a conduit if an esophagectomy is required in the future.

Postoperative surveillances

Routine postoperative surveillance should strongly be considered in all patients, particularly those with SG. Reliance on symptomatology to select patients for surveillance may be unreliable and could result in missed disease.

Conclusion

BE is an important consideration in patients undergoing bariatric surgery. Development and progression of BE is intrinsically linked with obesity, and the level of adiposity correlates with the risk. Bariatric procedures can influence the progression of BE, and can also impact future cancer surgery. Current evidence suggests BE is more likely to develop or progress after SG, while reports have shown regression and resolution after RYGB. Screening before bariatric surgery may be valuable as, theoretically, it helps guide procedure selection and may inform future BE surveillance. Strong consideration should be given to endoscopic surveillance after SG, given the reported rates of development of BE at 5–10 years. Further prospective studies are required to strengthen these conclusions and facilitate the development of evidence-based guidelines for perioperative management of BE in bariatric patients.

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Table 1. Current published guidelines on use of endoscopy before bariatric surgery

Guideline	Year	Recommendation	Selective or routine
The American Association of Clinical Endocrinologists, the Obesity Society, and the American Society for Metabolic and Bariatric Surgery (AACE/TOS/ASMBS)[31]	2013	Clinically significant GI symptoms should be evaluated before bariatric surgery with imaging studies, UGI series, or endoscopy (Grade D recommendation)	Selective
The European Association for Endoscopic Surgery (EAES)[30]	2005	Upper GI endoscopy or upper GI series is advisable for all bariatric procedures (Grade C recommendation), but is strongly recommended for gastric bypass patients (Grade B recommendation)	Routine
The American Society for Gastrointestinal Endoscopy (ASGE)[32]	2015	The decision to perform preoperative endoscopy should be individualized in patients scheduled to undergo bariatric surgery after a thorough discussion with the surgeon, taking into consideration the type of bariatric procedure performed (low-quality evidence) Patients with symptoms of GERD, such as heartburn, regurgitation, dysphagia, or any postprandial symptoms that suggest a foregut pathology and/or who chronically use antisecretory medications should have a UGI endoscopic evaluation before bariatric surgery.	Selective

Abbreviations: GI, gastrointestinal; UGI, upper gastrointestinal.

Table 2. **Studies examining preoperative screening endoscopy for Barrett's esophagus in bariatric surgical patients**

Study	Type	*N*	*N* demographics	Population included	Prevalence of BE	Normal	Clinical impact	Recommendation for endoscopy
Abou Hussein[42]	Retrospective	1278	61% female, BMI = 43.7 ± 8	All bariatric	3.0%	10.6%		Routine
Azagury,[39]	Retrospective	319		Asymptomatic bariatric	0.31%	54%	4%	Selective
Bradley, 2015 [65]	Retrospective	79	76% female, median BMI = 44.6	All bariatric with reflux, dysphagia, or hiatal hernia	16.4%	-	19%	Selective
Carabotti[66]	Prospective	142	82% female, median BMI = 44	All bariatric	0	53%	3.5%	Routine
Coblijn[67]	Prospective	523	76.5% female, mean BMI = 46.5	Primary bariatric	1%	49%	62%	Not recommended
Csendes[68]	Prospective	426	78% female	All bariatric	5.8%	45%	-	Routine
D'Hondt[43]	Retrospective	652	Age 39.5 ± 11.3, 70.9% female, BMI = 42.8 ± 5.0	Consecutive RYGB	0.8%	31.9%	7.8%	Routine
D'Silva[41]	Prospective	675	Age 45 ± 9.1, BMI = 43.9 ± 6.9	All bariatric	1.8%	78.5%	9.9%	Routine
Endo[69]	Retrospective	155	59% female, BMI 45 ± 8	All bariatric	0.6%	34%	10%	Routine
Estevez-Fernandez[70]	Retrospective	331	82% female, mean BMI = 47.5	All bariatric	0.6%	-	52.6%	Routine
Fernandes[71]	Retrospective	613	78% female, mean = BMI 44.7	All bariatric (selective endoscopy)	0.5%	43.7%	-	-
Heimgartner[72]	Prospective	100	68% female, mean BMI = 44.9 ± 6.9	All bariatric	6%	29%	-	Routine
Humphreys[40]	Retrospective	371		LAGB (selective endoscopy)	0.8%	56%	-	Routine
Kavanagh[73]	Retrospective	133	68% female, mean BMI = 46.8	All symptomatic bariatric	8%	-	69.9%	Selective
Masci[74]	Prospective	1049	78% female, mean BMI = 45.1 ± 4.6	LAGB (selective endoscopy)	0	91.8%	2.3%	-
Mong[34]	Retrospective	272	87% female, BMI = 48 ± 7.95	All bariatric (selective endoscopy)	3.7%	88%	0.3%	Routine
Moulla[44]	Retrospective	636	BMI = 49 (range 31–92)	All bariatric (selective endoscopy)	15.0%	-	1.6%	Selective
Munoz[75]	Retrospective	626	72% female, BMI 42 ± 6.5	RYGB (selective endoscopy)	0.16%	54%	> 45%	Routine
Ozeki[45]	Retrospective	260	Age 54.0 ± 9.0, 25% female, BMI = 44.9 ± 7.0	All Veterans undergoing RYGB or SG (routine)	7.4%	33%	-	Selective
Parikh[49]	Meta-analysis	6616	Mean BMI = 47 ± 3.2	Review of 28 studies on preoperative endoscopy	0.1%	92.4%	7.6%	Selective
Saarinen[36]	Retrospective	1275	Age 48.5 ± 9.1, 72.6% female, BMI = 46.1 ± 7.0	All bariatric (selective endoscopy)	3.7% (2.2% asymptomatic)	50.7%	24.6%	Routine
Santo[76]	Retrospective	717		All bariatric	0.28%	-	-	-

				(selective endoscopy)				
Schigt[37]	Prospective	523	Age 44.3, 76.7% female, BMI = 46.6	All bariatric (routine endoscopy)	1.3%	49.1%	-	Selective
Schlottmann[77]	Retrospective	193	64% female, mean = BMI 44.5	All bariatric (selective endoscopy)	1.6%	64%	-	Routine
Schneider[46]	Retrospective	1190		All bariatric (selective endoscopy)	0.4%	39.5%	2.4%	Routine
Sharaf[47]	Retrospective	195	Age 41.2, 78.5% female, BMI = 48.9	All bariatric (selective endoscopy)	3.1%	10.3%	61.5%	Routine
Soricelli[64]	Prospective	144	Mean BMI = 46.2 ± 7.2	SG (routine endoscopy)	13%	-	-	Consider wider screening
Suter[78]	Prospective	345	80% female, mean BMI = 44.7	Patients being evaluated for bariatric surgery	1.2%	-	-	-
Wolter[48]	Retrospective	801	Age 43.8 ± 11.4, BMI = 50.1 ± 12.7	All bariatric (routine endoscopy)	2.1%	34.3%	-	Routine
Zeni[33]	Retrospective	169	82% female, mean BMI = 49.7	All bariatric	1.3%	33.3%	9.4%	Routine

Abbreviations: BMI, body mass index; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy; LAGB, laparoscopic adjustable gastric band

Table 3. Systematic reviews on bariatric procedures and their impact on BE

Systematic review	Aims	Study details	Results	Conclusions
Adil, 2019[52]	Effect on RYGB on BE with pre- and postendoscopy and biopsy	Eight studies 10,779 patients 117 patients with BE with follow-up >1 year	Regression of BE after RYGB (risk difference -0.56 (95% CI: -0.69 to -0.43), $P < 0.001$): • Short-segment BE (-0.51 (95% CI: -0.68 to -0.33), $P < 0.001$) • Long-segment BE (-0.46 (95% CI: -0.71 to -0.21), $P < 0.001$) Strongly associated with regression of BE versus progression (OR = 31.2 (95% CI: 11.37 to 85.63), $P < 0.001$) Improvement in GERD (-0.93 (95% CI: -1.04 to -0.81), $P < 0.001$	RYGB improves BE significantly at a >1-year follow-up
Yeung, 2020[25]	Effect of SG on GERD, esophagitis, and BE to clarify long-term clinical sequelae	46 studies (all GERD related pathology) 10,718 patients Five studies with BE with long-term results	The long-term prevalence of BE was 8%. High heterogeneity ($I^2 = 94.1\%$) Increase in postoperative GERD after the sleeve was 19% *De novo* reflux was 23% The long-term prevalence of esophagitis was 28% 4% required conversion to RYGB for severe reflux Symptoms do not always correlate with pathology	Prevalence of BE following SG is significant Surgical decision making and consent should consider these long-term complications

Table 4. Studies examining effect of SG on incidence of BE

Ref.	*N*	Population	Follow-up	BE	Results
Braghetto, 2016 (prospective)[79]	231	Patients with normal preoperative endoscopy 72.7% female, BMI = 38.5 ± 3.1	5–6 years	1.2% *de novo*	Postoperatively: • BE in 3 (1.2%) (between 5 and 6) years postoperatively • Reflux symptoms in 57 (23.2%) • Erosive esophagitis in 38 (15.5%)
Csendes, 2019 (prospective)[80]	104	Consecutive SG without (group 1) and with (group 2) reflux	1.6 years	3.8-4.5% *de novo*	Group 1: Two developed BE (3.8%), 58.5% developed GERD Group 2: Two developed BE (4.5%) , 13.5% had a resolution of GERD
Felsenreich, 2017 (prospective)[81]	43	Patients with primary SG 10 years prior, without reflux or HH preoperative	10+ years	15% *de novo*	*De novo* Barrett's in 15%, *de novo* hiatal hernia in 45%. Siz patients converted to RYGB due to reflux (14.0%) over 130 months. Lower GI quality of life and higher reflux symptom index in sleeve patients.
Felsenreich, 2018 (prospective)[82]	65	Consecutive SG between January 2003 and December 2006	10+ years	9.2% *de novo*	44 patients had follow-up gastroscopy, of which six had Barrett's metaplasia
Genco, 2017 (prospective)[63]	110	Primary SG between July 2007 and January 2010	58 months	17.2% *de novo*	Increase in erosive esophagitis, GERD symptoms (68.1% versus 33.6%, $P < 0.001$), and PPI intake (57.2% versus 19.1%, $P < 0.0001$). Endoscopic findings not related to symptoms.
Sebastianelli, 2019 (prospective)[83]	90	SG who had undergone surgery at least 5 years before without preoperative BE	78 ± 15 months	18.8% *de novo*	Increase in GERD symptoms (22% to 76%), erosive esophagitis (10% to 41%), and PPI use (22% to 52%), all $P < 0.05$.

Abbreviations: RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy; BE, Barrett's esophagus; GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; EE, erosive esophagitis

Table 5. Studies examining effect of RYGB on incidence of BE

Ref.	Type of gastric bypass	*N*	Population	Follow-up	BE	Results
Andrew, 2018 (retrospective)[84]	RYGB	14	Patients with proven BE who underwent RYGB (September 2011–May 2015).	1 year	42.9% resolution	All short-segment BE. 6/14 (42.9%) complete regression to normal 13/14 (92.8%) complied with PPI for 1 year
Braghetto, 2012 (retrospective)[85]	Resectional RYGB	21	Patients undergoing antireflux operations (including LRRYGB)	1 year	61.9% regression	Regression in IM in 13 (61.9%) Significant reduction in acid reflux, symptoms and erosive esophagitis
Csendes, 2006 (retrospective)[53]	Resectional RYGB	557	August 1999 to October2004	2 years	SSBE: 57% regression LSBE: 20% regression	Twelve patients with BE (2.1%): seven with SSBE and five with LSBE Postoperative: • Regression of 57% with SSBE • Regression of 20% with LSBE • Reflux esophagitis resolved in all patients

						postoperatively
Felsenreich, 2019 (retrospective)[55]	Revision RYGB	10	Patients converted to RYGB due to BE after SG	33.4 months	80% resolution	Decrease in a 24-h mean acid exposure (36.8% to 3.8%) and mean DeMeester from 100 to 16.3.
Gorodner, 2017 (retrospective)[86]	RYGB	11	Bariatric cases with baseline BE	41 ± 31 months	36% resolution	Nine SSBE and two SBE Average length of BE 2.1 ± 1.6 cm preoperatively and 1.2 ± 1.2 cm postoperatively(P > 0.05) Remission in four cases (36%): three SSBE and one LSBE.
Houghton, 2008 (retrospective)[87]	RYGB	5	Patients with Bx proven LSBE undergoing RYGB, with a >1-year follow-up	34 months	40% resolution 80% decreased length	BE length 6 ± 2cm preoperatively to 2 ± 1 cm postoperatively: • Length had decreased/disappeared in four patients • Complete resolution of BE in two patients • Improvement in degree of dysplasia in three patients
Signorini, 2020 (retrospective)[54]	RYGB	9	Consecutive patients with RYGB	24 months	SSBE: 75% resolution LSBE: 40% resolution	Preoperative BE: SSBE 45% and LSBE 55%: • SSBE 75% resolved • LSBE 40% resolved • No progression to dysplasia

Abbreviations: RYGB, Roux-en-Y gastric bypass; LRRYGB, laparoscopic resectional Roux-en-Y gastric bypass; BE, Barrett's esophagus; SG, sleeve gastrectomy; SSBE, short-segment Barrett's esophagus; LSBE, long-segment Barrett's esophagus; IM, intestinal metaplasia; PPI, proton pump inhibitor.