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Original article

Esophageal adenocarcinoma after sleeve gastrectomy: actual or potential threat? Italian series and literature review

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Abstract

Background: Sleeve gastrectomy (SG) leads to esophageal mucosal damage in an elevated percentage of cases, configuring a clinical condition of Barrett's esophagus (BE) in a proportion as high as 15–18.8%. BE may rarely evolve into esophageal adenocarcinoma (EAC).
Objectives: To raise awareness of BE as a precancerous lesion which may progress toward malignancy after this popular bariatric procedure.
Setting: Bariatric referral centers, Italy.
Methods: All patients referred to our bariatric center who developed an EAC after SG between 2012 and 2019 were reviewed and consecutively included in this study. The available scientific literature regarding this complication is additionally reviewed.
Results: The 3 male patients comprised in this case series underwent laparoscopic SG between 2012 and 2015 in different bariatric referral centers. Age and body mass index at baseline ranged from 21–54 years and 43.1–75.6 kg/m^2, respectively. All patients were lost to follow-up early after surgery (3.7 ± 1.4 months), and were diagnosed with EAC at a mean of 27.3 ± 7.6 months after SG. The 4 reported cases in the scientific literature developed an EAC at a mean of 32.5 ± 23 months from SG. Overall, a diagnosis of EAC was made approximately 30.3 ± 17.1 months postoperatively, which seems relatively and worryingly early after surgery.
Conclusion: Although the rate and probability of progression from BE to EAC is still not well defined, assuming that the rising popularity and execution of SG leads to a growth in the BE incidence, then the preoperative identification and stratification of cancer risk factors in this subset of patients is strongly encouraged. Clinical and endoscopic follow-ups are essential to allow for prevention and early diagnosis and for epidemiologic data collection purposes. (Surg Obes Relat Dis 2020;■:1–7.) © 2020 American Society for Bariatric Surgery. Published by Elsevier Inc. All rights reserved.

Key words: Sleeve gastrectomy; Esophageal adenocarcinoma; Barrett's esophagus; GERD

Obesity per se—specifically, a body mass index (BMI) > 40 kg/m^2—has been directly associated with a raised cancer incidence and an increased mortality rate [1,2]. Furthermore, having a high BMI increases an individual's risk of developing gastroesophageal malignancies, which has been mainly attributed to the frequent

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coexistence of gastroesophageal reflux disease (GERD) in such patients [3].

Over the past 2 decades, the number of bariatric and metabolic surgeries has been increasing as a consequence of the obesity pandemic phenomenon. Amongst bariatric procedures, sleeve gastrectomy (SG) has gained widespread popularity worldwide, allowing it to become the most commonly performed operation in this category [4]. Even though substantial surgically induced weight loss should contribute to the reduction of obesity-related GERD, SG is specifically characterized by an alteration of the normal gastric anatomy, which may in turn cause an increase in GERD. In fact, the new configuration of the gastric sleeve can result in the elimination of the angle of His following the removal of the gastric fundus, with the dissection of the phreno-esophageal ligament reducing the lower esophageal sphincter pressure and causing a mid-gastric stenosis and an intrathoracic migration of the gastric remnant [5].

Surgical modifications taking place after SG may variably contribute to a new onset of GERD or an aggravation of pre-existing GERD, which possibly leads to an array of esophageal lesions, evolving from differing degrees of erosive esophagitis, to Barrett's esophagus (BE) with or without dysplasia, occasionally resulting in esophageal adenocarcinoma (EAC).

Nevertheless, controversies regarding the effects of SG on postoperative GERD still exist at present. Available data in the literature have generated differing outcomes in this regard, with several studies affirming an increase of postoperative GERD, while others demonstrate its reduction in post-SG patients [6]. However, several investigators have demonstrated that SG leads to esophageal mucosal damage in an elevated percentage of cases, configuring a clinical condition of BE in a proportion as high as 15–18.8% [7–9].

In consideration of the fact that BE is indeed a precancerous lesion which may progress to EAC, awareness should be raised regarding such a potential scenario after this SG. We present the clinical cases of a series of patients who developed EAC after SG, substantially adding to the scarce available scientific literature on this topic, which is additionally reviewed.

Methods

Patients who were referred to our bariatric center for abnormal endoscopic gastroesophageal findings between 2012 and 2019 were retrospectively reviewed from a prospectively collected database and consecutively included in this study. At our institution, a prospective database is routinely collected during follow-up for all patients after any bariatric procedure, including those patients operated on elsewhere and being referred to our center postoperatively. All surgical procedures were performed by different bariatric surgeons in distinct hospitals in Italy. Patients were then referred to our bariatric center due to the appearance of postoperative, atypical symptoms.

Case series

Table 1 summarizes all major aspects of patients included in the present case series.

The 3 male patients comprised in this study underwent laparoscopic SG between 2012 and 2015. Age and BMI at baseline ranged from 21–54 years and 43.1–75.6 kg/m^2, respectively. Obesity-related co-morbid conditions included hypertension in 2 patients and obstructive sleep apnea, dyslipidemia, and type 2 diabetes in 1 patient. Other additional risk factors for EAC included active tobacco use in 2 patients. Preoperative endoscopic evaluations were routinely performed in all of the patients by experienced endoscopists as part of a standard preoperative bariatric workup, and were negative for reflux, hiatal hernia, erosive esophagitis, or any other esophago-gastric abnormality. Patients did not report any typical or atypical GERD symptoms and were not on proton pump inhibitors (PPIs) or antacids. The postoperative course was uneventful and no complications were registered for any of the patients.

All patients were lost to follow-up at a mean of 3.7 ± 1.4 months after surgery.

Case 1 presented with retrosternal pain and heartburn approximately 12 months from SG. The patient sought medical attention and was initially prescribed cardiological exams, which were all within normal ranges. He was subsequently started on PPIs and other antacids *pro re nata*. At 24 months after surgery, the patient had reached a BMI of 33.4 kg/m^2 and reported progressive worsening of the aforementioned symptoms. The patient finally underwent an esophagogastroduodenoscopy (EGD) upon advice of a gastroenterologist. The EGD showed a solid mass at the lower third of the esophagus and histology confirmed a poorly differentiated adenocarcinoma. The patient underwent an open subtotal esophagectomy with colonic interposition, and the final pathologic stage was pT2 N1 M1. After surgery, the patient was submitted to adjuvant chemotherapy and died 5 months postoperatively.

Case 2 did not present with any outstanding signs or symptoms apart from weight loss failure, reaching a postoperative BMI of 70.6 kg/m^2 at 22 months post SG. For this reason, the patient underwent several diagnostic exams to further investigate the lack of weight loss. The EGD described a normal-looking gastric sleeve and a supracardial pseudopolyp, which was biopsied. Histology showed a moderately differentiated adenocarcinoma. The patient underwent an open subtotal esophagectomy with Roux-en-Y esophagojejunostomy. The pathologic stage was pT3 N0 M0. The patient was started on adjuvant chemotherapy and completed the 6-month follow-up with no evidence of disease recurrence.

Table 1
Patient variables and outcomes in patients included in the case series

Case	Age, yr	Sex	Race	Baseline weight, kg	Baseline BMI, kg/m^2	Weight at follow-up, kg	BMI at follow-up, kg/m^2	Co-morbidities	Smoking habit	Endoscopic evaluation before SG	Post-SG complications	Time from SG to EAC diagnosis, mo	Presenting symptoms	Management	TNM staging	Outcome
1	54	Male	Caucasian	132	43.1	102.3	33.4	Hypertension, dyslipidemia, T2 D	Smoker	No untoward finding	None	24	Heartburn, retrosternal pain, persistent weight loss	Open subtotal esophagectomy with colonic interposition, followed by adjuvant CHT	pT2 N1 M1	Death 5 mo postoperatively
2	21	Male	Caucasian	247.8	75.6	231.4	70.6	OSA	Nonsmoker	No untoward finding	None	22	None reported	Open subtotal esophagectomy with Roux-en-Y esophagojejunostomy, followed by adjuvant CHT	pT3 N0 M0	Completed 6-mo follow-up with no evidence of disease recurrence
3	39	Male	Caucasian	148	46.3	80.2	25.0	Hypertension	Smoker	No untoward finding	None	36	Anemia, asthenia, dysphagia, heartburn	N/A	cT3 N1 M1	Death during completion of diagnostic assessment

BMI = body mass index; SG = sleeve gastrectomy; EAC = esophageal adenocarcinoma; T2D = type 2 diabetes; CHT = chemotherapy; OSA = obstructive sleep apnea; N/A = not applicable.
Patient variables include demographic data, co-morbidities, smoking habit, preoperative endoscopic findings, time from SG to EAC diagnosis, presenting symptoms, TNM stage, and EAC therapeutic management.

Case 3 displayed substantial weight loss, which showed a tendency to a continuous decrease at 3 years from SG, reaching a BMI of 25 kg/m^2. This, however, was attributed to the bariatric procedure. Due to the subsequent appearance of anemia, weakness, heartburn, and dysphagia 36 months postoperatively, the patient started a diagnostic workup on the advice of his general practitioner. The EGD showed an ulcerated mass at the level of the esophago-gastric junction, and histologic findings following biopsies confirmed a moderately differentiated EAC. The total-body computed tomography scan showed the presence of loco-regional enlarged lymph nodes and multiple liver metastases (cT3 N1 M1). The patient died during completion of the diagnostic assessment.

Literature review

A review of the English literature of all fully described cases of EAC after SG was performed using the following key words: esophageal adenocarcinoma, cancer, Barrett's esophagus, sleeve gastrectomy, bariatric surgery, esophagectomy, diagnosis, management, and surgical approach. A total of 4 articles were found and reviewed; 4 case reports regarding EAC after SG were identified as adequately describing in depth the clinical cases and were included in the review [10–13].

Demographic data, co-morbidities, preoperative endoscopic evaluation details, time from SG to diagnosis of EAC, postoperative complications, presenting symptoms, and therapeutic management approaches are summarized and reported in Table 2.

Discussion

Knowledge regarding the occurrence of esophageal malignancies after SG is extremely limited. The present case series describes the largest number of cases of EAC occurring after SG, conspicuously adding to the already reported cases in the literature.

The patients included in the study were all male patients who were diagnosed with an EAC at a mean of 27.3 ± 7.6 months (range, 22–36 mo) after SG. However, no clear timeline for the development of EAC can be determined due to the lack of adequate postoperative surveillance, loss to follow-up, and delays in subsequent endoscopies. In fact, all patients were lost to follow-up almost immediately after surgery. This, in addition to the vague or absent signs and symptoms developed by patients, might be at the basis of a delayed diagnosis. Furthermore, upper gastrointestinal symptoms, such as heartburn and dysphagia, in addition to substantial weight loss, are usually attributable to bariatric surgery itself and are often overlooked for this reason. In this case series, all patients sought medical attention through their general practitioners or other specialist categories (e.g., cardiologist, gastroenterologist) rather than seeking a bariatric surgical referral. This might have

Table 2
Literature review

Case [Ref.]	Age, yr	Sex	Baseline BMI, kg/m^2	Co-morbidities	Smoking habit	Endoscopic evaluation before SG	Post-SG complications	Time from SG to EAC diagnosis, mo	Presenting symptoms	Management	TNM Staging
1 [10]	57	Female	51.8	OSA; degenerative joint disease; COPD	Smoker	Not performed	None	4	Intolerance to solid foods	CRT; endoscopic follow-up	cT2 N1 Mx
2 [11]	44	Female	42.0	OSA; hypercholesterolemia; chronic idiopathic neutropenia	Nonsmoker	Not performed	None	30	Iron deficiency (during routine testing)	Neoadjuvant CRT; open esophago-gastrectomy followed by adjuvant CHT	pT3 N0 M0
3 [12]	48	Male	48.5	OSA, hypertension; dyslipidemia; insulin resistance	Smoker	No untoward findings	None	60	Dysphagia to solid foods	Neoadjuvant CRT; restaging PET/CT showing liver metastases, followed by a further CHT cycle	cTxN+M1
4 [13]	55	Female	42.0	Hypertension; T2 D; dyslipidemia; arthritis	Nonsmoker	Short segment BE (2 cm) without dysplasia	Gastric leak	36	Persistent vomiting	Endoscopic mucosectomy; endoscopic follow-up	pT1 N0 M0

BMI = body mass index; SG = sleeve gastrectomy; EAC = esophageal adenocarcinoma; OSA = obstructive sleep apnea; COPD = chronic obstructive pulmonary disease; CRT = chemoradiation therapy; CHT = chemotherapy; PET/CT = positron emission tomography/computed tomography; T2D = type 2 diabetes; BE = Barrett's esophagus.

The review includes all described cases of EAC after SG (based on data from references 10–13). The table summarizes demographic data, co-morbidities, smoking habit, preoperative endoscopic findings, time from SG to EAC diagnosis, presenting symptoms, TNM stage, and EAC therapeutic management.

caused the execution of several futile or nonspecific investigations. The deferral of diagnostic workups in our group of patients led to them receiving diagnoses in advanced stages of the disease—namely, Stage IVB in 2 patients and Stage III in 1 patient—thus inevitably increasing the mortality rate.

Known major risk factors for both BE and EAC development in the included patients were male gender, obesity, and tobacco use. In fact, smoking causes a 5-fold increase in the development of EAC, and this risk is additionally raised when coexisting in conjunction with obesity [14]. Furthermore, the appearance of EAC at a considerably young age (i.e., 21 yr), as in Case 2, might be in part associated with genetic factors involved in the pathogenesis of BE, possibly accelerating the carcinogenic progression. The presence of the aforementioned risk factors must be acknowledged, as they augment the chance of developing EAC beyond the role of SG itself.

Inadequate surgical techniques may also lead to the genesis or aggravation of reflux postoperatively. Technical errors can result in kinking, twisting, narrowing, or stenosis at the level of the gastric sleeve, leading to the formation of a high-pressure zone, recognized as a risk factor not only for gastric leaks but also for the appearance or worsening of GERD [15].

The 4 reported cases in the scientific literature comprised 3 females and 1 male, with a mean age of 51 ± 6.1 years and a baseline BMI of 46.1 ± 4.9 kg/m^2, who developed an EAC at a mean of 32.5 ± 23 months (range, 4–60 mo) after SG. It is worth noting that only 2 patients had a preoperative EGD before SG. Case 1 [10] developed an EAC only 4 months postoperatively, which was likely to have already been present before SG and could have been detected if an endoscopic evaluation had been performed before surgery. Of the 2 patients who had a preoperative EGD, 1 patient (Case 3) [12] was found to have no esophago-gastric abnormality, while 1 patient (Case 4) [13] was diagnosed with a short-segment BE without dysplasia (Table 2). Despite the diagnosis of BE, a multidisciplinary team decided to proceed with performing a SG, which was complicated by a gastric leak requiring surgical reintervention. Approximately 3 years from surgery, the patient developed a supra-cardial, 2 cm, pedunculated polyp, which was removed by endoscopic mucosectomy. The final pathology report showed a well-differentiated adenocarcinoma arising on Barrett's metaplasia. This case report further emphasizes not only the importance of performing a preoperative EGD to detect any mucosal lesion, but also that BE should be an essential part of an informed consent discussion with the patient regarding procedure choice, due to its innate risk of malignant evolution. The most recent position statement (2017) on SG by the American Society for Metabolic and Bariatric Surgery acknowledges that opinion favors the preferential use of RYGB as the

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procedure of choice in patients with BE. However, the statement also notes that there is no consensus among authors that BE is an absolute contraindication to SG [16]. This may be due to differences in outcome reporting and the lack of long-term endoscopic follow-ups.

The role of perioperative endoscopy in patients undergoing bariatric surgery also remains unclear. Clinical practice guidelines endorsed by the International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) recommend the execution of preoperative EGD in patients with clinically significant upper gastrointestinal symptoms, and possibly in all patients eligible for SG [17,18]. In contrast, due to the scarce data available regarding the incidence of upper gastrointestinal malignancies, a postoperative EGD is recommended at 1 year after bariatric surgery and then every 2–3 years after SG or 1-anastomosis gastric bypass to allow for an early diagnosis of BE or other upper gastrointestinal cancers [18].

Whether SG induces an improvement or worsening of GERD is rather controversial [6]. However, several studies have demonstrated that SG is able to induce an increase of a biliary-type reflux into the distal esophagus [7,19–21]. Some authors confirmed that the continuous acid and/or biliary insult to the esophageal mucosa leads to a structural modification of the characteristic cells normally lining the esophagus, which are replaced by intestinal-type columnar cells, configuring the so-called histologic alteration of intestinal metaplasia. Kauer et al. [22,23] noted in patients who had not undergone bariatric surgery that a refluxate with a pH range of 4–7 into the esophagus—likely to be of duodenal derivation—could be causally responsible for the development of BE. In fact, those patients with BE were actually found to have the greatest esophageal bilirubin exposure during spectrophotometry when compared with those who only had erosive esophagitis (EE) or who had no esophageal lesions at all.

The composition of refluxate after SG is mainly mixed or biliary, which brings the intragastric pH to higher levels than those seen in patients who have not had bariatric surgery [7,19,20,24]. This could not only explain the absence or scarcity of symptoms and the relative ineffectiveness of PPI treatment in such patients, but also the greater risk of developing EE and BE. Furthermore, certain authors have hypothesized that this condition might progress to EAC even more rapidly after a bariatric operation [7,25], due to the greater exposure of the distal esophagus to biliary content. Indeed, EAC is amongst the cancer types that most commonly occurs after SG [26]. Overall, in the 7 reported cases, the diagnosis of EAC was made at a mean of 30.3 ± 17.1 months postoperatively, which seems extremely early after surgery.

The prevalence of BE in the general population ranges from 1.6%–2% [9,26,27], while its prevalence after SG may be as high as 18.8% [9]. It is difficult to know the true prevalence of BE after SG, due to the lack of standardization of both short- and long-term postoperative endoscopic surveillance. In addition, most patients are still lost to follow-up long term.

Even though only a small percentage of patients affected by BE eventually develops an EAC, when projected on the large and ever-growing proportion of SGs performed worldwide, a great number of esophageal malignancies should perhaps be inevitably envisioned in the not-too-distant future. The 2019 IFSO Global Registry reported that the approximate number of worldwide SG procedures was 305,242 in the calendar years 2015–2018 [28]. If we consider the risk these patients have of developing BE after SG to be 8%, as calculated by the latest systematic review and meta-analysis [29], we might have to deal with an estimated 24,419 post-SG individuals who could possibly develop BE. Of these patients, .3–.6% [30] have a risk of evolving to EAC, possibly translating to approximately 110 patients over 3 years who might need oncologic attention.

Furthermore, patients who are lost to follow-up after SG have a greater risk of being diagnosed with BE at later stages: for instance, when dysplasia has already developed. These patients might lose the opportunity to receive minimally invasive endoluminal approaches for the treatment of such esophageal mucosal lesions, and often required actual surgery, which may possibly lead to greater morbidity and mortality rates, as highlighted in the present case series.

Although the prevalence of BE in SG patients lost to follow-up is likely to be superimposable to the reported data in literature, what these patients are lacking is the chance of undergoing protocols of secondary prevention to identify any esophageal malignancy at its earliest stages.

In fact, the importance of postoperative endoscopic follow-ups should be stressed to all patients before surgery to perhaps increase their compliance with surveillance. Follow-ups are crucial in prevention or early diagnosis. Furthermore, endoscopic surveillance is necessary for epidemiologic data collection, so as to properly and fully comprehend the extent and incidence rate of such oncologic complications.

Conclusions

Although the rate and probability of progression from BE to EAC is still not well defined, assuming that the rising popularity and execution of SG leads to a growth in the BE incidence, then the preoperative identification and stratification of cancer risk factors in this subset of patients is required. Thus, when considering SG as the designated bariatric procedure, its favorable outcomes in terms of weight reduction and cardio-metabolic effects should be weighed alongside the risks of developing GERD and BE and the low but existing potential of malignant evolution.

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It is clear that there is poor correlation between reflux symptoms and the presence of esophageal pathology [7,31]. While the evidence-based literature is not definitive, it is becoming increasingly clear that systematic surveillance, including some plan for endoscopic postoperative assessments, in all SG patients is necessary. To this regard, clinical follow-ups are also essential to allow for prevention and early diagnosis and for epidemiologic data collection purposes.

Scientific societies should encourage the further investigation of this issue in the context of large, population-based studies, perhaps providing international online registries, to precisely determine the extent of the problem and to possibly better comprehend its pathogenesis, management, and outcomes.

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Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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