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Barrett's esophagus and esophageal cancer after sleeve gastrectomy. Myth or reality?☆



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ABSTRACT

Sleeve gastrectomy has become the most performed bariatric surgery technique in the world. This bariatric technique has been related to the appearance of gastroesophageal reflux and recently with *de novo* Barrett's esophagus. It is not clear that this leads to an increased incidence of esophageal adenocarcinoma. In this review we analyze the current scientific literature to try to answer the true incidence of Barrett's esophagus and adenocarcinoma after sleeve gastrectomy, and whether these data should make us change the indications for this technique.

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Esófago de Barrett y cáncer de esófago tras gastrectomía vertical. ¿Mito o realidad?

RESUMEN

La gastrectomía vertical se ha convertido en la técnica de cirugía bariátrica más realizada en el mundo. Esta técnica bariátrica se ha relacionado con la aparición de reflujo gastroesofágico y recientemente con el esófago de Barrett *de novo*. No queda claro que esto conlleve un aumento en la incidencia de adenocarcinoma de esófago. En esta revisión analizamos la bibliografía actual para intentar responder a la verdadera incidencia de esófago de Barrett y adenocarcinoma tras la gastrectomía vertical, y si estos datos nos deben hacer cambiar las indicaciones para esta técnica.

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Vertical sleeve gastrectomy (VSG) has been the most performed bariatric surgery technique worldwide since 2015,¹ at 58.5% between 2014 and 2019.² Onset of gastro-oesophageal reflux (GOR) after VSG is a controversial topic. Following the publication by Braguetto and Csendes³ in 2016 highlighting the presence of de novo Barrett's oesophagus (BO) after VSG, there have been numerous publications addressing this issue. Alarm was raised in 2017 following the publication of the Italian study by Genco et al.⁴ where the incidence of de novo BO was 17.2% after VSG. Subsequent meta-analyses studying the occurrence of BO after VSG show incidences between 85%⁵ and 11.6%.⁶ The IFSO 2020 position statement puts the incidence at 4.6%.²

Because GOR is asymptomatic in a high percentage of patients undergoing VSG, and the consequences in the form of oesophagitis or BO are potentially serious, endoscopic follow-up is recommended, even if asymptomatic.⁷

In Spain, according to the multicentre study led by Ferrer et al.,⁸ 5 years after VSG, de novo reflux appeared in more than 50% of patients, oesophagitis in more than 30%, and in contrast to the data from the meta-analyses cited, de novo BO appeared in only .9% of patients.

When comparing different bariatric techniques, GOR symptoms worsen after adjustable gastric banding (AGB) and VSG, improve in Roux-en-Y gastric bypass (RYGB), and remain unchanged in one-anastomosis gastric bypass. Erosive oesophagitis occurs in 74% of VSGs, 42% of AGB, and 22% of bypasses (Roux-en-Y and one-anastomosis). BO was found only after VSG (16.8%). This multicentre study confirms that VSG has a higher rate of GOR, oesophagitis, and BO.⁹

However, some authors suggest that the presence or absence of GOR should be established before and/or after VSG by high-resolution manometry and pH monitoring, given that the signs and symptoms of GOR are not sufficient to demonstrate it.¹⁰ This would make it possible to determine preoperatively which patients are at risk for GOR and would benefit from an antireflux technique such as RYGB.

Routine gastroscopy prior to bariatric surgery is also controversial. A meta-analysis of 6,845 patients showed changes in the initial surgical plan in 7.8% of cases. These changes included: hiatal hernia repair, delay of surgery to resolve gastritis or peptic ulcer, change of surgical technique, or endoscopic resection of suspicious lesions. In 27.5% of patients, endoscopy resulted in a change in medical treatment to eradicate *Helicobacter pylori* or initiation of proton pump inhibitors (PPIs) to treat gastritis or reflux.¹¹ This has led societies such as the American Society for Metabolic and Bariatric Surgery to recommend preoperative gastroscopy as routine from 2021.¹² The presence of BO or a large hiatal hernia would contraindicate VSG, reinforcing the recommendation for preoperative gastroscopy.¹¹

The rate of conversion from VSG to RYGB due to GOR ranges from 3.1% in the meta-analysis by Guan et al. (2019)¹³ to 4% in the meta-analysis by Yeung et al. (2020).⁵ In a recent multicentre study evaluating patients undergoing VGS at 15 years, Felsenreich et al. (2021)¹⁴ describe GOR conversion rates of 18.9%, which could be influenced by including patients operated during the learning curve of the technique. In addition to technical issues, appropriate selection of patients for VGS may reduce the incidence of postoperative GOR. The

presence of a gastric band or preoperative GOR seem to be factors that worsen post-operative GOR.¹⁴

It seems clear that post-VSG GOR is due to functional or mechanical changes such as transhiatal migration of the VGS, stenosis, and incompetence of the lower oesophageal sphincter. The onset of post-VGS GOR is higher than 30%, although with large variations between groups (22%–50%). Therefore, patients should be informed of the possibility of reconversion in .5%–7% of cases. Endoscopic follow-up every 2–3 years after VGS is also essential. The recommended conversion is to RYGB, and repair is recommended if hiatal hernia is found.¹⁵

Given the high number of VGS performed worldwide, and based on some publications,^{4–6} we may be creating a serious problem with an unprecedented incidence rate of BO. The risk of BO in the general population is only 1%–2%,¹⁶ therefore, if estimates are correct, by performing VGS we could be creating a population with an up to 6-fold risk for developing BO. If BO progresses to oesophageal adenocarcinoma (OAC), there would be a limitation in reconstruction, requiring coloplasty, a more complex technique with greater morbidity than gastric interposition.¹⁷ However, although these concerns are logical, they remain unsubstantiated hypotheses in the literature, particularly the onset of OAC after VGS, which is limited to isolated cases.¹⁸ Some studies even suggest a reduction in the rate of OAC after bariatric surgery, but given that the incidence rate of OAC is low, these analyses should be viewed with caution as the incidence may vary over time.¹⁹ The time factor is important as the risk of developing OAC is approximately .33% per year for patients without dysplasia and .19% per year for short-segment BO.²⁰ Therefore, there remains a risk of a substantial delay between index surgery (VGS) and the development of cancer.

When analysing the prevalence of cancer in obese patients, the increase compared to non-obese patients is striking. In a retrospective study on a database of more than 15 million US patients, morbidly obese patients who did not undergo surgery, those who underwent VGS and those who underwent BPG surgery in the period 2010–2018 with a follow-up of 5 years were distributed homogeneously according to their comorbidities into 3 groups with a 1:1:1 ratio. The non-operated group developed cancer of any type more frequently (4.61 vs. 3.47 vs. 3.62%; $p < .0005$) and the same was true for obesity-related cancer (4.82 vs. 3.48 vs. 3.52%; $p < .0005$). In those who did not undergo surgery, the increased incidence of colon, lung, liver, ovarian, and womb cancer is noteworthy. No significant differences were observed in stomach or oesophageal cancer, among others. Among the operated patients, the risk of any type of cancer was reduced by 25.7% (VGS) and 22.2% (GBP), and of obesity-related cancer by 29% and 28%, respectively.²¹

When specifically studying the incidence of oesophageal or gastric cancer after bariatric surgery, in a retrospective, multicentre, observational study of patients operated on between 1985 and 2020 (OGMOS study),²² the interval between bariatric surgery and cancer diagnosis was 5.9 ± 4.1 years after VGS, compared to 9.4 ± 7.1 years after RYGB, 10.5 ± 5.7 years after adjustable gastric banding, and 2.0 ± 1.4 years after one-anastomosis GB. Tumour location was equally distributed in the oesophagus, oesophago-gastric junction, or stomach

after VGS or RYGB. Adenocarcinomas accounted for 82.9%. One third of the patients had metastases at diagnosis. Because this is a retrospective study and the total number of patients who underwent surgery is not known, the incidence rate of oesophago-gastric cancer in this population cannot be calculated.

If the presence of GOR and BO has been associated with an increased incidence of OAC in the general population, it would seem logical to assume that an increase in GOR and BO after VGS would be associated with an increase in OAC. Let us examine what is true about this hypothesis. Based on a database of 48,991 bariatric surgery patients from New York State (USA) between 1995 and 2010, only 21 patients had OAC (.043%). They found no differences between surgical techniques. However, they did find an increase in OAC in the bariatric surgery population compared to the general population (.0028%).²³

The Italian group, led by Genco et al. published a series of 3 patients who developed OAC after VGS. They were diagnosed 27.3 ± 7.6 months after the surgery.²⁴ Although the number of cases is low, the short time between VGS and OAC is noteworthy. A recent review article found 31 cases of OAC after RYGB or VGS. The common feature is the presentation as dysphagia and affecting the distal oesophagus or oesophago-gastric junction.²⁵

A Canadian study analysed a population of patients undergoing reflux-prone surgery (VGS and duodenal switch), another population theoretically protected against GOR (RYGB), and a population of obese non-operated patients (control). With a mean follow-up of 7.6 years, they found 8 cases of OAC after bariatric surgery, with no significant differences between surgical groups. The risk of OAC was higher for the VGS group compared to the control, but the difference disappeared after adjusting for confounding factors.²⁶

In our experience, following published studies warning of the increased incidence of BO after VGS, we performed a gastroscopy on all patients undergoing VGS and found 33% of patients with oesophagitis and one patient with high-grade dysplasia on a BO that required endoscopic mucosal resection and radiofrequency ablation of the BO.²⁷

Conclusions

The incidence of BO after VGS, a widely performed surgery worldwide, and its potential for malignancy, have opened up the debate regarding the indication and follow-up of VGS. The rapid transformation to BO after VGS compared to that occurring in non-operated patients with GORD suggests that the aggression of the reflex is more potent. Determining the malignancy potential of post-VGS BO is critical for decision making because obese patients undergoing surgery are young and the potential GOR onset may be long. Until this is clarified, it seems prudent to limit the indications for this technique. We should be stricter in selection, avoiding those with clinical or endoscopic findings of GOR or BO. It is essential to perform an endoscopic study prior to surgery, and long-term endoscopic follow-up.

Conflict of interests

The authors have no conflict of interests to declare.

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