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Gastric Cancer After Restrictive Bariatric Surgery: A Clinical Pitfall
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Abstract
Although vertical banded gastroplasty is rarely performed at present, most bariatric surgery departments continue to follow up patients who underwent this procedure in the past few decades. In view of this, it is advisable for bariatric and general surgeons to know how to diagnose the very rare event of the development of a gastric cancer after this restrictive procedure. In this report, 2 cases of gastric cancer occurring years after vertical banded gastroplasty are presented, and clinical presentation and diagnostic difficulties are discussed.

Keywords
bariatric surgery, restrictive procedures, vertical banded gastroplasty, gastric cancer, dysphagia

Introduction
Although, to date, vertical banded gastroplasty (VBG) seems to be a nearly abandoned bariatric procedure,1 patients who underwent this intervention in the past few decades represent a nonnegligible group of subjects2 both in long-term bariatric surgery follow-up programs and in general surgery daily practice. In view of this, it is advisable for bariatric and general surgeons to know how to recognize and manage long-term complications of this procedure, including very rare events, such as the development of a gastric cancer.

The aim of the present study was to report 2 cases of gastric cancer occurring in the gastric pouch years after VBG and to discuss clinical presentation and diagnostic difficulties.

Case 1
A 50-year-old woman, with no medical history, non-smoker, underwent a laparoscopic VBG in March 2000 according to MacLean (Figure 1) for severe obesity. At the time of surgery, her weight was 100 kg and the body mass index (BMI) 41.6 kg/m². Postoperative course was uneventful and the patient had a good weight loss result, with a weight of approximately 75 kg, 2 years after surgery (BMI = 31.2 kg/m²) and no alimentary difficulties. Eight years after surgery, the patient presented with a 2-month history of vomiting, dysphagia and a 12-kg eight loss. At clinical evaluation, the abdomen was unremarkable; laboratory tests showed mild anemia (hemoglobin 11.7 g/dL). The patient underwent a water-soluble contrast that showed gastroesophageal reflux with a mild stricture of the neopylorus and an upper endoscopy, which found a severe stricture; after a 15-mm pneumatic dilation, the distal stomach showed severe hypertrophic–ulcerative gastritis, which on histological examination revealed a poorly differentiated gastric carcinoma (Figure 2).

The patient underwent a computed tomography (CT) scan of the abdomen, which confirmed a marked thickening of the gastric wall with multiple adenopathy and peritoneal carcinosis Figure 3), and a CT scan of the chest, which was negative for metastases. The patient underwent an explorative laparotomy that confirmed an advanced unresectable gastric cancer with neoplastic infiltration of the omentum, left hepatic lobe, pancreas, lymph nodes, and ascites. The patient underwent a palliative digiunostomy. Afterward, she underwent chemotherapy (5 cycles of a TCF scheme: taxoten, cisplatin, and 5FU). She developed cranial metastases treated
with 5 cycles of radiotherapy (DFT30Gy/10fr) and died 4 months after surgery due to disease progression.

**Case 2**

A 56-year-old woman underwent laparoscopic MacLean VBG for obesity in April 2005; preoperative weight and BMI were 98 kg and 43.6 kg/m², respectively. She had a good weight loss result, with 2-year follow-up weight at 70 kg (BMI = 31.1 kg/m²). Three years after surgery, the patient referred with dysphagia, emesis, and weight loss. She underwent an upper endoscopy, which revealed a neo-pylorus stricture; after a 20-mm Pneumatic dilation, the distal stomach showed an ulcerative lesion and the histology revealed a moderately differentiated carcinoma (Figure 4). The abdominal CT confirmed a gastric wall thickening (Figure 5) while the CT study of the chest was negative for metastases. The patient underwent a total gastrectomy with Roux-en-Y esophagojejunal anastomosis and D1 lymphadenectomy. The patient underwent adjuvant chemotherapy (TCF scheme: taxoten, cisplatin, and 5FU), which was stopped at the fourth cycle for severe neutropenia. To date, at 4-year follow-up, the patient is alive and free of disease.

**Discussion**

Mason first described VBG in 1982; in the original technique, the gastric pouch was simply sutured, without transection from the gastric fundus. In 1993 MacLean et al described VBG with a divided gastric pouch (Figure 1), and its laparoscopic approach was described shortly after by Hess and Hess. VBG represented the most frequently used restrictive bariatric procedure before the advent of laparoscopy, but the introduction of laparoscopic adjustable gastric banding in the early 1990s led to an increasingly extensive use of the latter and VBG has been progressively abandoned, also because of high reported long-term failure and reintervention rates.

Whereas VBG accounted for 51% of all bariatric procedures in 1987-1989, it represented only 3% of procedures in 2002-2004 and 1.1% in 2008. Nevertheless, since a high percentage of bariatric patients underwent VBG in the past few decades, they still represent an important group of patients both in bariatric and in general surgery daily practice. In view of this, it is of critical importance to promptly diagnose and manage post-VBG complications and rare events, such as gastric cancer development. The development of gastric cancer after bariatric surgery represents a very rare event, although the actual incidence is still unknown since the available literature is in form of isolated case reports while large, epidemiological studies are lacking. With regard to VBG, only 9 cases of esophagogastric neoplasms have been previously described, all but one located in the stomach, diagnosed at a mean of 10 years after bariatric surgery, ranging 2 to 16 years (Table 1).

From a pathophysiological point of view, post-VBG gastric cancer may be related, on one hand, to stasis of food and gastric acid in the pouch causing both chronic friction and inflammation of the gastric mucosa, both prolonged contact between gastric pouch mucosa and food or other exogenous carcinogens. On the other hand, post-VBG gastric cancer may be related to the local inflammation caused by the band mesh: Negri et al reported pathological findings at the transitional zone in 31 VBG patients, with proximal and distal pouch macroscopically normal in most cases. The effects of the mesh is thought to be related not only to the foreign body itself, which cause adhesion and local reaction, but also to the internal pressure against the mucosa leading to decreased blood flow, decreased parietal cell number, and metaplastic mucosal changes, which may progress to malignancy.
It could be speculated that both the pathophysiological mechanisms described could lead to the same pathological effect, activating the so-called “inflammatory pathway” of carcinogenesis, based on an increase of pro-inflammatory effectors in the gastric microenvironment. Genetic alterations that induce malignant transformation also produce a cancer-promoting inflammatory microenvironment: Inflammation is epigenetically linked to loss of protein expression through transcriptional silencing via promoters. Accumulating evidence indicates that the “inflammation pathway” plays a role in cancer development in many sites other than the stomach, such as ovaries, liver, pancreas, lungs, endometrium, esophagus, or colon and rectum. Genetic alterations that induce malignant transformation also produce a cancer-promoting inflammatory microenvironment: Inflammation is epigenetically linked to loss of protein expression through transcriptional silencing via promoters. Accumulating evidence indicates that the “inflammation pathway” plays a role in cancer development in many sites other than the stomach, such as ovaries, liver, pancreas, lungs, endometrium, esophagus, or colon and rectum. Many studies have demonstrated a key role of cyclooxygenase-2 and prostaglandin E2 receptor signaling, which, for example, promotes the activation of epidermal growth factor receptor signaling, the NF-κB pathway (which plays a central role in the regulation of oxidative stress and cytokine expression), or several other different molecular mechanisms. Nevertheless, the potential risk of gastric neoplasm onset, although not completely understood, appears to be low, considering the low number of reported cases compared with the number of VBG performed worldwide in the past few decades. Although rare, the main concern in gastric neoplasms after VBG is the delay in diagnosis that may have catastrophic effects on patients’ prognosis. This delay is mainly related to the fact that symptoms such as dysphagia, food intolerance, gastroesophageal reflux, and emesis are frequently attributed by the patient to the side effects of surgery. Furthermore, the patient could be happy with a late weight loss thus further delaying to seek medical advice. Finally, the clinician himself or herself may attribute symptoms to VBG’s side effects. It should be thus underlined that it is of critical importance to exhaustively study all patients presenting with any new or modified post-VBG symptoms, and also in the very long-term follow-up. Furthermore, it should be borne in mind that in VBG patients, the upper gastrointestinal tract is still easily accessible for endoscopy and radiographic evaluation. While contrast upper gastrointestinal series could have an uncertain role, upper endoscopy has a critical importance in the diagnostic evaluation and should be promptly performed. During endoscopic evaluation it is moreover critically important to obtain a complete dilation of any strictures that could hamper the evaluation of the whole stomach, as occurred in both our patients. Also, in the case reported by Sweet, endoscopy found a marked stenosis of the pouch outlet with no evidence of mass or ulceration within the pouch, but once the outlet was dilated, the subsequent contrast study showed a narrowed outlet that led to surgical exploration. When an early diagnosis is obtained, standard surgical and chemotherapy protocols can be performed with no worsening in prognosis, as demonstrated by reported case 2. To obtain an early diagnosis, it is critically important to follow up all patients who underwent VBG; in our department, follow-up visits are scheduled at 1 month, 3 months, 6 months after surgery, and annually thereafter. Upper endoscopy is routinely performed before surgery and 1 to 2 years after that, while in the long-term follow-up, contrast gastrointestinal series and upper endoscopies are performed based on the patient’s symptoms, such as dysphagia, emesis, and heartburn. A main concern in bariatric surgery is the lack of long-term follow-up data, because of a very high incidence of drop-outs. Nevertheless, efforts should be made by each bariatric surgery center to follow up all patients consistently, bearing in mind that surveillance of patients after bariatric surgery should ideally be lifelong. In conclusion, we recommend long-term follow-up in all patients who underwent VBG, with periodic endoscopic surveillance and a promptly through evaluation of patients with any new or modified upper gastrointestinal symptom.
References


