Original article

Effect of Roux-en-Y gastric bypass in obese patients with Barrett’s esophagus: attempts to eliminate duodenogastric reflux

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Abstract

Background: To assess the effect of Roux-en-Y gastric bypass (RYGB) at a tertiary referral Center of Excellence for bariatric surgery on the length and presence of dysplasia in morbidly obese patients with Barrett’s esophagus (BE). Esophageal reflux of gastroduodenal contents (acid, bile) contributes to the development of BE and progression in the dysplasia-carcinoma sequence. Obese patients have a high prevalence of gastroesophageal reflux and might be at an increased risk of developing BE and esophageal adenocarcinoma. The effect of eliminating duodenogastroesophageal reflux on BE is not known.

Methods: We performed a retrospective review of all patients with pre-existing, biopsy-proven, long-segment (>3 cm) BE undergoing RYGB at our institution. Only patients with >1 year of endoscopic, biopsy-controlled follow-up (mean 34 mo) were included.

Results: Five patients (3 men and 2 women) were identified. The mean ± standard error of the mean preoperative length of BE was 6 ± 2 cm; 2 patients had low-grade dysplasia and 1 indeterminate dysplasia. At the postoperative follow-up (>1 yr) examinations, the length of BE had decreased in 4 patients; the overall length was 2 ± 1 cm; and only 1 patient had dysplasia. All patients experienced a decrease in the length of BE (n = 4), complete disappearance of BE (n = 2), or improvement in the degree of dysplasia (n = 3). The body mass index had decreased from 43 ± 4 kg/m² to 33 ± 3 kg/m², and all experienced subjective improvement in reflux symptoms postoperatively. RYGB resulted in complete or partial regression of BE in 4 of 5 patients and improvement in reflux symptoms in all.

Conclusion: Our results suggest that RYGB might be the procedure of choice in morbidly obese patients with BE requiring surgical treatment for gastroesophageal reflux disease. (Surg Obes Relat Dis 2008;4:1–5.) © 2008 American Society for Metabolic and Bariatric Surgery. All rights reserved.

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Barrett’s esophagus (BE) is thought to occur secondary to chronic reflux of acid and/or bile into the distal esophagus [1]. A cell line of Barrett’s adenocarcinoma exposed repeatedly to pulses of acidified bile in vitro demonstrated decreased rates of apoptosis and increased rates of proliferation compared with pulsed acid or bile alone [2]. One of the most highly studied and reliable animal models of BE involves the induction of acid bile reflux into the distal esophagus of rats by creating a proximal loop esophagojejunostomy [3]. This model induces the changes of BE in 75% of rats by 20 weeks and adenocarcinoma in about 50% of the rats by 28 weeks.

Symptomatic gastroesophageal reflux disease has been strongly associated with morbid obesity [4–10]. Population-based studies have demonstrated an increase in the risk of reflux symptoms with increasing body mass index (BMI)
Clinical symptoms of gastroesophageal reflux disease are well-known risk factors for the development of BE [7,11]. Increased BMI and symptoms of gastroesophageal reflux disease could potentially contribute to high rates of BE among the morbidly obese compared with the nonobese, although supportive evidence is lacking [12,13]. Roux-en-Y gastric bypass (RYGB) is known to decrease both the symptoms and the use of medications directed at gastroesophageal reflux, as well as to eliminate or decrease dramatically the amount of acid present in the proximal gastric pouch [10,14–16]. RYGB diverts bile and duodenal contents away from the distal esophagus completely, thereby removing both the stimuli (acid and bile) implicated in the development of BE and esophageal adenocarcinoma. The effect of effectively diverting these substances from the distal esophagus on the presence, length, and associated dysplasia of a segment of BE is not well defined. Others have suggested that an effective antireflux procedure could lead to regression of BE [17]. Therefore, the aim of our study was to assess the changes in the length and degree of dysplasia in patients with long-segment BE who had undergone vertical, disconnected RYGB [18].

Methods

The institutional review board of Mayo Clinic Rochester approved this study in accordance with the U.S. department of Health and Human Services guidelines. We performed a retrospective chart review of all patients undergoing RYGB at our institution with a preoperative diagnosis of long-segment BE who had ≥1 year of complete endoscopic follow-up. All patients at our institution with a histopathologic diagnosis of BE from 1985 to the present were identified and compared with our comprehensive institutional bariatric surgery database, started in 1985, to identify those patients with BE who had subsequently undergone our technique of RYGB. The inclusion criteria included histopathologic evidence of intestinal metaplasia with goblet cells of ≥3 cm length (BE) diagnosed at our institution and surveillance endoscopy for ≥1 year postoperatively with appropriate biopsies (4 quadrant biopsies at 2-cm intervals) according to our protocol for patients with BE. The exclusion criteria included suspected BE not confirmed by pathologic examination, biopsies demonstrating intestinal metaplasia of the cardia only, diagnoses by outside pathologic examinations, and no postoperative endoscopy with biopsy.

Of about 1500 patients with records in our database, 11 met the preoperative inclusion criteria. However, 6 of these patients did not undergo postoperative endoscopy at our institution, leaving 5 patients for this study. The patient records were reviewed for demographics, weight, preoperative endoscopic findings, medical history, and follow-up information. All values are reported as the mean ± standard error of the mean, unless otherwise noted. The mean duration of endoscopic follow-up was 34 ± 7 months.

The operative RYGB procedure included gastric partitioning to create a small (≤10 cm³), vertical disconnected proximal pouch of cardia in all patients, with a Roux limb of 150–240 cm in length in 4 patients and a malabsorptive, very, very long-limb RYGB [19] with a common channel of 100 cm in 1 patient with a BMI >50 kg/m².

Results

Five patients (3 men, 2 women), aged 59 ± 3 years, with a confirmed preoperative diagnosis of long-segment BE and who had undergone postoperative endoscopy, had undergone RYGB at our institution. Their preoperative weight and BMI was 122 ± 8 kg and 43 ± 4 kg/m², respectively. The mean preoperative BE length was 6 cm (range 3–14); 2 patients had low-grade dysplasia and 1 had indeterminate dysplasia.

Esophageal symptoms were present in 3 patients preoperatively—2 had clinically significant reflux symptoms, and 1 complained of dysphagia. The patient with dysphagia had previously undergone vertical banded gastroplasty. Another patient had previously undergone loop gastric bypass. No patient had previously undergone an ineffective antireflux procedure (Nissen fundoplication). Perioperative complications occurred in 1 patient who developed transient renal failure not requiring dialysis. The average duration of stay was 7 ± 1 days.

The patient who had undergone loop gastric bypass about 20 years previously involving a horizontal complete gastric partitioning with a loop gastrojejunostomy, presented to our institution with symptoms of reflux, severe bile reflux esophagitis, and 14-cm BE length, with low-grade dysplasia. After conversion of her loop gastric bypass to RYGB, with a 240-cm Roux limb, she underwent photodynamic therapy to treat the BE at 10 months postoperatively. The combination of both interventions decreased her BE length from 14 cm to 5 cm at 27 months postoperatively; however, the esophageal biopsy findings showed persistent low-grade dysplasia at her last follow-up visit.

At an average of 34 ± 7 months (range 19–61) postoperatively, endoscopic examination revealed a mean BE length of 2 cm (range 0–5; Table 1). One patient had persistent low-grade dysplasia (as described above); the rest had no evidence of dysplasia. Two patients had complete regression of their BE, and all 5 had either partial regression in segment length and/or improvement in the degree of dysplasia.

Discussion

Esophageal adenocarcinoma has been correlated with obesity in several association studies [12,13]. RYGB has been shown to improve reflux and reflux symptoms in obese patients and to decrease or eliminate the amount of bile and acid present in the distal esophagus, theoretically eliminat-
ing the stimulus for the progression of BE to adenocarcinoma [10,14,16]. The aim of our study was to describe the effect of RYGB on the length and epithelial changes in obese patients with established, long-segment BE. During a mean follow-up of 34 ± 7 months, 4 of 5 patients had complete or partial regression of BE, 2 of 3 with dysplasia at baseline had no dysplasia at follow-up, and all had a decrease in their BMI.

The rate of complete regression of long-segment BE after RYGB in our series (2 of 5 patients) is consistent with that reported by Csendes et al. [20]. In their series, 2 of 6 patients with long-segment BE had complete regression at a mean follow-up of 39 months. With a mean follow-up of 34 months, they found complete regression of short-segment BE in 3 of 6 patients. As in our series, no patient in their series experienced BE progression, and most patients experienced a decrease in reflux symptoms. Similarly, Cobey and Oelschlager [21] described an obese patient who experienced regression of short-segment BE after RYGB with resolution of reflux symptoms. To the best of our knowledge, these are the only 2 reports in the English language addressing the effect of RYGB on BE.

In contrast to the study by Csendes et al. [20], we restricted our study to those patients with long-segment BE who underwent RYGB. We excluded those patients with only intestinal metaplasia of the cardia, because the significance of this condition is not known or well understood [22]. We also excluded patients with short-segment BE in an attempt to minimize sampling error during subsequent endoscopies and to include a group with a greater risk of potential sequelae of their BE.

In addition to the small sample size, another potential limitation of this descriptive study in assessing the degree of dysplasia was the potential for sampling error. To limit this possibility, we only included patients for whom an adequate number of biopsies had been collected at surveillance according to our institutional protocol for biopsies in patients with BE. We could not control for medications that might theoretically affect the degree of dysplasia, such as the use of nonsteroidal anti-inflammatory drugs or proton pump inhibitors.

Although the present study was small, the results are suggestive and highlight several points in the treatment of obese patients with BE. Although no patient in either the study by Csendes et al. [20] or the present study progressed to high-grade dysplasia or adenocarcinoma, 50–70% of patients had persistent BE at their last follow-up examination. These patients and their physicians should therefore be encouraged to participate in an ongoing surveillance program. The lack of resolution of the Barrett’s changes in a percentage of patients undergoing RYGB reinforces the need for additional research on BE and the underlying pathophysiology, as well as other therapies. Because RYGB prevents any appreciable acid/peptic/bile reflux, the maintenance of BE must provide some clues to its biologic nature. Also, our results of partial or total regression could have important implications in other patient populations.

Conclusion

In our study, RYGB resulted in dramatic improvement in reflux symptoms and, our limited experience suggests, mod-
est rates of regression of BE in obese patients, as well as helping to reverse the co-morbidity of severe obesity. The findings of the present study, combined with those from the study by Csendes et al. [20], raises the question of whether RYGB should be considered for morbidly obese patients with BE requiring surgical treatment for gastroesophageal reflux disease.

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Disclosures

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

References


Editorial comment

Comments on: Effect of Roux-en-Y gastric bypass in obese patients with Barrett’s esophagus: attempts to eliminate duodenogastric reflux

This is the second study documenting complete or partial regression of long-segment Barrett’s esophagus after Roux-en-Y gastric bypass, making it a very important contribution. Controversy also continues as to whether antireflux procedures can lead to regression of Barrett’s esophagus. This study provides further credence to this hypothesis. Although several studies have been published that have shown a decrease in gastroesophageal reflux disease symptoms after bariatric surgery, none have detailed the preoperative and postoperative objective data regarding the 24-hour esophageal pH,