

Late Marginal Ulcers after Gastric Bypass for Morbid Obesity. Clinical and Endoscopic Findings and Response to Treatment

Attila Csendes · Jannina Torres · Ana María Burgos

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Abstract Marginal ulcer (MU) is an occasional complication after gastric bypass which can occur early or late after surgery. In this study, we evaluated the incidence, clinical presentation, and endoscopic behavior of patients with late MU. Five hundred fifty morbidly obese patients were evaluated prospectively performing an endoscopic study 1–8 years after surgery. They were submitted either to laparotomic ($n=392$) or laparoscopic ($n=158$) approach. Six patients (1%) presented late MU 12 to 84 months after surgery. Four patients had single ulcer, while two patients had multiple ulcers. All were treated with proton pump inhibitors (PPIs). Several endoscopic evaluations were performed in each patient showing healing and no recurrence of the ulcer. Late MU occurs in a small proportion (1%) of patients submitted to gastric bypass. It can be single or multiple. Medical treatment with PPIs achieves healing at a mean time of 7 months. Several endoscopic evaluations should be performed in these patients in order to demonstrate healing of the ulcer and no recurrence.

Keywords Late marginal ulcer · Gastric bypass · Morbid obesity

Marginal ulcer (MU) or anastomotic ulcer is a peptic ulcer produced at the jejunal mucosa just distal to the gastro-jejunal anastomosis after partial gastrectomy for benign diseases, such as gastric or duodenal ulcer or after surgery for morbid obesity [1]. After gastric bypass, we have reported two different behaviors regarding MU: The “early” MU, which is frequently seen in a consecutive endoscopic

study, is performed 1–2 months after surgery and the “late” MU which occurs 1 year or more after surgery. The complete clinical and endoscopic findings of early MU were reported in that publication [1]. The purpose of the present prospective study is to report the clinical and endoscopic features of a group of patients with MU late after surgery and their response to medical treatment.

Material and Methods

Patients Studied

A total of 550 patients with morbid obesity submitted to gastric bypass were evaluated in a prospective trial by clinical questionnaire and one upper endoscopy performed in all patients once 1–8 years after surgery. The mean time for performing endoscopy was 40.5 months (18–96). These patients were operated on between August 2002 and August 2007 at the Department of Surgery University Hospital, and all had a complete upper endoscopic evaluation, as described before [2]. They were submitted either to gastric bypass by laparotomic approach ($n=392$) or by laparoscopic approach ($n=158$). In the laparotomic group, a resectional gastric bypass was performed [2]. All patients gave their written informed consent to be included in this investigation. Exclusion criteria corresponded to patients who refused endoscopic evaluation [3] and those submitted to other bariatric procedures.

Clinical Questionnaire

All 550 patients were evaluated by clinical approach, asking for the presence of epigastric pain, previous or actual upper gastrointestinal hemorrhage, and vomits.

A. Csendes (✉) · J. Torres · A. M. Burgos
Department of Surgery, Clinical Hospital University of Chile,
Santos Dumont 999,
Santiago, Chile
e-mail: acsendes@redclinicauchile.cl

Endoscopic Evaluation

After an 8-h fast, upper endoscopy was performed by a video endoscope (Olympus, Japan) by one of the authors (AC). After a slight pharyngeal anesthesia and intravenous premedication with 7.5 mg midazolam and hyoscine butylbromide, the endoscope was introduced in a gentle way, avoiding the “pull and push” effect. The macroscopic aspect of the small gastric pouch, the size and aspect of the gastrojejunal anastomosis, and the aspect of the jejunal mucosa were carefully evaluated. This examination was performed at a mean of 44 months after surgery (range 12–72). In patients with MU, this examination was repeated several times.

Results

From the 550 patients evaluated, 6 patients (1%) developed late marginal ulcer and constitute the study group. The main clinical features of these patients are shown in Table 1. The proportion of gender was similar to the entire group. Actual BMI was in the adequate range late after surgery, with a% of loss of excess weight of 72%. Persistent epigastric pain was present in 83% of the patients. This pain had the typical features of peptic ulcer pain: it was localized in the epigastrium very precisely, it was persistent, and during night sleep, it awakened the patient. In the whole group of 550 patients evaluated, there were 26 patients (4.7%) who presented upper abdominal pain without the features of peptic ulcer pain and in whom the endoscopy was normal. One case had an upper gastrointestinal hemorrhage which needed also endoscopic treatment with injection of adrenaline. None of the patients had received NSAID drugs before the appearance of MU; however, four of them were heavy smokers.

The main endoscopic findings are shown in Table 2. There were two patients with two anastomotic ulcers, while four (67%) had a single ulcer. The size varied from 8 to 20 mm. No patient had *Helicobacter pylori* present at fundic mucosa. Table 3 shows the endoscopic behavior of marginal ulcer in each patient. A mean of four endoscopies were performed in this group: Two patients developed MU

Table 1 Clinical features of six patients with late marginal ulcers

Age (year)	Mean 49 (32–70)
Gender	5 women and 1 man
Actual BMI (kg/m ²)	28.5 (25–32)
Epigastric pain	5
Upper GI hemorrhage	1

Table 2 Endoscopic findings in six patients with late marginal ulcers

No. of ulcers	Single	4
	Multiple	2
Size ulcers (mm)	<10 mm	2
	11–15 mm	3
	>16 mm	1
No. of endoscopies after surgery/patient		4[4–7]

12 months after surgery, one patient 24 months after surgery, and three patients 60–84 months after surgery. All had one or more endoscopies after medical treatment with proton pump inhibitors (PPIs) for 12 months, showing healing of all ulcers at the consecutive endoscopic control at a mean time of 7 months of treatment. During each endoscopic procedure, the size of the gastric pouch was measured from the esophagogastric junction up to the anastomosis, measuring between 2 and 5 cm. In none, there was any significant increase in the size of the pouch. In an unpublished investigation, we evaluated the size of the pouch in 105 patients submitted to three or more endoscopies after surgery, showing no increase in size up to 120 months of follow-up; serum gastrin was not measured in any patient.

Discussion

The results of the present study suggest that if a prospective consecutive endoscopic evaluation is performed in a group of patients submitted to gastric bypass, a low incidence of marginal ulcer is demonstrated 1 year or more after surgery. This is what we denominate “late marginal ulcer” [1], in contrast to “early marginal ulcer”, which occurs 1 to 10 months after surgery [4–6]. We make this distinction because it is highly probable that the etiology of both ulcers is different. In patients with early MU, a combination of different factors has been mentioned: use of non-absorbable versus absorbable sutures [7–9], local tissue injury-related factors such as use of electrocautery, some degree of ischemia, inflammatory reaction, etc. [1, 8]. The majority of publications refer to these early marginal ulcers, as we pointed out previously [1].

The late MU, which is the purpose of the present investigation, probably has a different etiology, similar to what occurred in patients with peptic ulcer submitted to partial or subtotal gastrectomy many years ago. In these patients, excess acid contact with the jejunum has been considered the main mechanism for ulceration [8, 10, 11]. The usual gastric pouch size with a total capacity less than 30 ml produces almost no acid, as has been demonstrated

Table 3 Evolution of marginal ulcer later after gastric bypass

Endoscopy after surgery							
	Preoperative Finding	I Control	II Control	III Control	IV Control	V Control	HP Postoperative
Case 1	Normal HP (-)	12 months Ulcer	18 months Scar	36 months Normal	48 months Normal	84 m Normal	(-)
Case 2	Normal HP (-)	12 months Normal	72 m Ulcer	84 m Scar	110 m Normal		(-)
Case 3	Normal	12 months Normal	84 m Bleeding ulcer	96 m Scar			?
Case 4	Normal HP (-)	12 months Normal	24 m Ulcer	72 m Normal			(-)
Case 5	Reflux HP (-)	6 months Normal	12 m Normal	24 m Normal	60 m Ulcer	84 m Normal	(-)
Case 6	Hiatal hernia HP (-)	12 months Ulcer	24 m Normal	84 m Normal			(-)

HP (-): Absence of *H. pylori* infection.

previously [12, 13]. Therefore, an increase in parietal cell mass due to dilatation of the pouch, as suggested by Sapala et al. is one possibility [10]. Other factors such as intact vagal innervation or the appearance of gastrinoma should be considered. In recent studies, the excessive use of NSAID [11] has been mentioned, as well as the infection of HP [14, 15].

It is difficult to compare our prospective endoscopic trial with any other publication, because all reports have (a) retrospective studies, (b) small group of patients, (c) endoscopic studies only in symptomatic patients. The careful review of several publications has shown that the majority of MU were detected early after surgery and very few mention the true late ulcers. Rasmussen et al. [15] performed endoscopy in only 36% of the operated patients finding two MUs 1 year after surgery. Sapala [10], in a 3-year prospective study, mention one patient (0.6%) with MU 3 years after surgery with a gastric pouch capacity of 60 ml. Huang et al. [3] performed endoscopy in 47 symptomatic patients finding three MU more than 1 year after surgery. Cappella [7] and Vasquez [9] perform only 10 months follow-up. Some of these late MU can be very aggressive, producing perforation or severe bleeding, situation which needs emergency surgery [16, 17]. In these patients, the use of NSAID and heavy smoking were mentioned as probable inducing factors. We have performed two previous studies concerning endoscopic evaluation of the small gastric pouch 1–2 years after surgery. In the first study [18], we evaluated 227 patients 2 years after gastric bypass, finding one patient (0.4%) with anastomotic ulcer. In the second study [1], upper endoscopy was performed in 315 patients 1–2 years after

surgery, finding one case (0.3%) with MU. The present study includes a much larger number of patients as well as a later follow-up to 96 months after surgery, finding 1% of late MU after surgery. These patients were submitted to several consecutive endoscopic evaluations. The ideal method for screening patients to detect MU would be routine EGD for all postoperative patients as Rasmussen pointed out [15], and this is exactly what we performed in this investigation. The etiology of this late MU is difficult to determine. The most logical explanation would be an increase in the residual parietal cell mass, and besides, all our patients had both vagal trunks intact. This greater parietal cell mass could be due to an increase in the size of the pouch as time goes by or to the creation of a larger pouch during the initial surgery. However, all of these patients did not show an increase in the size of the pouch according to our endoscopic measurements. We did not evaluate the presence of a Zollinger-Ellison syndrome in none of these patients, because their clinical evolution was not compatible with such syndrome. There were two other important features among these patients: one is related to the fact that MU appeared after resectional gastric bypass as well as after classic bypass, eliminating in this way the hypothesis of a retained antrum of the excluded stomach and a consequent hypergastrinemia which could provoke a higher gastric output. The other special feature is the slowness of the ulcer healing after intensive PPI treatment, taking near 7 months to heal completely after treatment.

Conflicts of Interest None.

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