ABSTRACT

Introduction: Delayed massive bleeding from an ischemic ulcer is a complication after Roux-en-Y gastric bypass (RYGB). Ischemic ulcers that present with massive bleeding are rare and challenging for the gastroenterologist as well as the bariatric surgeon.

Case Description: This report reviews the case of a 63-year-old man who underwent an uncomplicated laparoscopic RYGB for morbid obesity and experienced two episodes of massive hemorrhage after the procedure, almost 1 year apart.

Conclusion: To our knowledge, there are only a few such specific cases reported. Here, we describe the treatment and outcome for such a case and present a review of the literature.

Key Words: Roux-en Y gastric bypass, Bariatric surgery, Morbid obesity, Postoperative complications, Marginal ulcer, Anastomosis.

INTRODUCTION

Obesity, defined as a body mass index (BMI) of >30 kg/m², is an increasing problem in the Western world. In the United States, the prevalence is approximately 30% in the adult population.¹ The incidence is increasing, and the World Health Organization (WHO) predicts that worldwide, in 2025, there will be 300 million obese people.²

Obesity is associated with the development of metabolic syndrome, early osteoarthritis, obstructive sleep apnea, and a high risk of cardiovascular disease.³ So far, the only treatment for morbid obesity with good long-term results is bariatric surgery. Bariatric surgery aims at inducing weight loss by reducing gastric volume, or absorption capacity of the intestines, or both of these together. Over recent decades, a wide variety of bariatric procedures have been developed, such as adjustable gastric banding, gastric sleeve, and Roux-en-Y gastric bypass (RYGB). At present, laparoscopic RYGB (LRYGB) is the gold standard. The results are superior compared with the results of gastric banding in sustained weight loss and resolution of diabetes.⁴

However, RYGB is a major operation that has a risk of severe early and late complications. Most complications occur during the procedure or directly after (eg, anastomotic leakage and bleeding); however, a few also potentially life-threatening complications can manifest years later.⁵

CASE REPORT

The patient was a 63-year-old man with a BMI of 57 kg/m². His medical history included hypertension, hyperlipidemia, insulin-dependent diabetes mellitus, and obstructive sleep apnea syndrome. Preoperative esophagogastroduodenoscopy (EGD) showed no abnormalities apart from a distinct sliding hernia diaphragmatica. In February 2009, the patient underwent an uneventful laparoscopic antecolic and antegastric RYGB with a 120-cm alimentary limb. The gastrojejunostomy was constructed using a linear Endo-GIA Universal stapler (Johnson & Johnson, Somerville, New Jersey, USA); the anterior side of the anastomosis was closed with uninterrupted Vicryl 2.0 (Ethicon Inc., Somerville, New Jersey, USA). The postoperative course was uncomplicated, and discharge followed after 3 days.
Weight loss was 75 kg in 15 months. In March 2010, he developed idiopathic atrium fibrillation de novo, which was treated with acenocoumarol. In August 2010, he underwent an uncomplicated abdominoplasty.

In May 2010, he was admitted to the hospital with severe melena and hematemesis; hemoglobin (Hb) at admission was 5.6 mmol/L. After 6 packet cells (PCs), he was hemodynamically stable. At EGD, a visible vessel was observed around the anastomosis. Hemostasis was achieved with adrenaline (7 mL) and hemoclip (Resolution Clip, Boston Scientific, Natick, Massachusetts, USA). After 9 days, the patient was discharged and prescribed 40 mg of pantoprazole once daily (twice daily for week 1), and the acenocoumarol was restarted. At discharge, Hb was 7.7 mmol/L.

Almost 1 year later, the patient was readmitted to the hospital with a second bleeding episode and Hb of 4.3 mmol/L. EGD showed a slightly restricted anastomosis and an ischemic ulcer with an adherent blood clot behind it. Close to the ulcer, an active bleeding focus was observed. Around the bleeding focus (17 mL), adrenaline was injected and hemostasis was reached. The blood clot could not be removed for the surgeon to examine the ulcer or to add additional therapy (eg, Goldprobe or hemoclip). Again, the acenocoumarol was antagonized using prothrombin complex, and IV pantoprazole was started. The patient received 5 PCs and became hemodynamically stable.

Five days later, during the same admission, the patient developed massive hematemesis and melena during a third episode of bleeding, with Hb dropping from 6.3 mmol/L to 5.0 mmol/L. Another EGD was performed, but the bleeding could not be stopped by use of adrenaline or clips. He became hemodynamically unstable, and admission at the intensive care unit was necessary. After administration of vitamin K and 13 PCs, the patient was optimized for semielective surgery. Eleven days after the first hemorrhage and stabilization, the patient underwent surgery.

At laparoscopy, adhesiolysis took place, and the marginal ulcer (MU) was resected by creation of a new smaller pouch with a 60-mm Echelon stapler (Johnson & Johnson). The proximal part of the alimentary limb, including the anastomosis, was resected, and a tensionless anastomosis was created as described above. The specimen, including the ulcer, was removed with an endobag (Figure 1), and a 27-Charrière (correct measurement) drain was left behind. Total blood loss was 300 mL, and no complications occurred during the procedure. Microscopic examination showed multifocal inflammation of the jejunum near the gastrojejunostomy, matching ischemia. No cell dysplasia or malignancy was found. His hospital stay was uncomplicated, and he was discharged in good condition after 8 days.

**DISCUSSION**

One of the late complications of RYGB is marginal ulceration. In the literature, mainly 3 synonyms are used to describe the same kind of ulcer: marginal, ischemic, and anastomotic. Below, we refer to those types of ulcers as marginal (MU). The literature reveals that developing MU after LRYGB is a relatively common complication, ranging between 0.6% and 16%.6–8 A prospective study performing routine postoperative endoscopy after 1 month and 17 months found an incidence of 4.1% MU in the first month. After 2 years, the incidence was 0.5%. Another study,9 examining only symptomatic patients, found an incidence of 6.7% based on the symptoms, but endoscopy confirmed this diagnosis in only 4% of patients.

Although typical complaints of MU, such as abdominal pain, nausea, and vomiting, are identified,8,10,11 all 7.6% of patients with MU in Garrido et al.’s study were asymptomatic.12 Other studies confirm this asymptomatic presentation. Furthermore, 28% to 61% of patients present with
massive bleeding and perforation; of those, 43% to 87% do not have any symptoms of dyspepsia or other gastrointestinal (GI) symptoms. Because of this result, it is likely that the incidence of MU is underestimated.9,12–17

Pathogenesis of the development of MU is unclear. It is thought that marginal ulceration and gastrogastric fistulas are likely a result of mucosal disruption and the digestive action of gastric secretions. It is thought that staples and other nonabsorbable materials have the tendency to migrate to the lumen of the bowel and create MU. The incidence of ulceration decreased significantly from 5.1% to 1.5% when absorbable suture material was used.12,14,17,18

An overload of gastric acid, medication, persistent Helicobacter pylori infection, pouch size, or suture material can be contributing factors.7,8,18–21 It is important that the pouch is small and limited to the cardia of the stomach, which reduces the percentage of MU to 0.01% in 1 year.22 With this technique, the parietal cell mass in the fundus is excluded, resulting in limited acid production, but tests have shown that although the acid secretion is almost none, the pH of the stomach is still low in an important part of the patients.23 A dilated pouch may predispose to late ulceration, as in our case, because of the increasing number of parietal cells after dilatation.8,20 Additionally, acid secretion is partially regulated by gastrin levels, so in a negative-feedback mechanism, acid secretion increases. Hypothetically, gastric acid plays a role in the development of MU. This is supported by the fact that a part of the marginal ulcers are curable by PPI treatment only.9,20,24,25

The contribution of H pylori to MU formation is questionable. Some studies found that infection with H pylori is a risk factor, even after eradication therapy. In the same study, 32% of ulcer beds on EGD showed remnants of suture material. Most remnants, although not significant, were of nonabsorbable materials.7 Other studies point in the opposite direction. A comparison of patient demographics (eg, H pylori seropositivity) showed seropositivity was equal in the group of patients with and without MU; occasionally, patients had no H pylori infection at all.26,27

Treatment of MU with PPIs is sufficient in most cases.9,20,28 Treatment of ulcer disease exists in PPIs for 6 months and, if necessary, is combined with H pylori eradication therapy. Endoscopy confirmed the healing properties of PPIs in late MU. In a group of 550 patients, 6 presented with late MU and were treated for at least 12 months. After 7 months, healing was complete in all patients.20 Any anticoagulation therapy should be antagonized and nonsteroidal anti-inflammatory drugs should be stopped. Patients who smoke tobacco should be motivated to quit. Another study showed that 4% in a group of 547 patients developed MU. All patients responded to 8 weeks of high-dose oral PPIs and received low-dose maintenance therapy.9 Subsequent to that study, the investigators recommended prophylactic PPI treatment after RYGB postoperatively. This resulted in a significantly lower incidence of MU. None of the 73 patients who received PPI treatment developed symptoms of MU. However, the effect of prophylactic PPI usage is questionable because of the wide incidence of MU with and without PPIs. In different published studies, administration and/or recommendation differs from 30 days and 2 years to lifelong. Currently, administration of PPI after RYGB as prophylactic therapy for 6 months is standard protocol in our facility.30

The surgical approach to MU is another option, especially for those MU that are resistant to medical treatment. However, revisional bariatric surgery is technically demanding and has been associated with high morbidity and mortality rates in acute situations.31–35 The mortality rate for an emergency operation to treat upper GI bleeding is still 10% to 30%. When done electively, it is <2%.35,54 Therefore, a semi-elective operation in a stable situation is preferred. This circumstance makes the recognition of ulcers with a risk of bleeding a crucial part of treatment. Operations for intractable MU are very successful in nonsmokers; 87% remained free of MU after revision. No data are available as specified for smokers.31 In addition to its success in treating MU, surgery gives the opportunity to correct any pouch dilatation or remove foreign material. Most data reflect the laparotomic technique, which is known for its greater complication rate, including leakage, wound infections, higher intraoperative blood loss, and a higher mortality rate.36,35,55 The fear of not being able to perform laparoscopic revisions after open procedures seems to be illegitimate. Currently, laparoscopic revisions are more successful, even after open gastric bypass.36 The treatment aim is resection of the ulcer. Ulcers and less-vital tissue should not be included in the new anastomosis.35,55,56

CONCLUSION

We present a case of a 63-year-old man who underwent LRYGB surgery for morbid obesity and developed repetitive delayed episodes of massive hemorrhage resulting from an ischemic ulcer. He was treated successfully with semi-elective revisional surgery.

Factors related to a lower incidence of MU include a small pouch of only the cardia of the stomach, no tension on anastomosis, the use of absorbable suture material, and
prescribing PPIs for at least 30 days after the operation. However, no consistent evidence of duration of administration exists. In the literature, this differs from 1 month to lifelong. Symptoms may vary from no symptoms to massive bleeding.

In most cases, treatment of the ulceration is successful with PPIs. However, in 32% of cases, reoperation is required. Reoperation is a difficult approach that is necessary only for patients who present with chronic anemia, gastrogastric fistulae, acute life-threatening upper GI bleeding, gastric perforation, and occurrence of nonhealing ulcers despite maximum pharmaceutical treatment.

A surgical resection is performed with excision of the gastrojejunostomy, including the ulcerated areas. After revisional surgery, subsequent therapy with PPIs is required.

It can be concluded that as a result of the relatively limited number of studies with a wide variety in the incidence of MU, no robust conclusion can be drawn. This overview of the literature published about MU and RYGB raises more questions than answers, especially concerning the role and effect of PPIs and the determination of patients who are more at risk than others to develop MU.

A major limitation of recent studies is that all but a few focus on and consequently test symptomatic patients. However, the chances of this complication in asymptomatic patients are not clear.

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