Catastrophic Bleeding From a Marginal Ulcer After Gastric Bypass

Shafik Sidani, MD, Ehab Akkary, MD, Robert Bell, MD, MA

ABSTRACT
Marginal ulceration at the gastrojejunal anastomosis is a common complication following Roux-Y gastric bypass (RYGB). Hemodynamically significant hemorrhagic marginal ulcers are usually treated either endoscopically or surgically. We describe a unique case of life-threatening hemorrhagic marginal ulcer eroding into the main splenic artery. This condition was initially managed with angiographic embolization, followed by surgical intervention.

Key Words: Marginal ulcer, Gastric bypass, Embolization, Morbid obesity.

INTRODUCTION
It is well established that Roux-Y gastric bypass (RYGB) causes significant weight loss and aids resolution of obesity related comorbidities.1 The surgery is also associated with a low incidence of early and late complications, including anastomotic leak, pulmonary embolism, small bowel obstruction, anastomotic stricture, gastrointestinal hemorrhage, and marginal ulceration (MU).2 Although a relatively minor complication, MU commonly occurs during the early or late postoperative period. The reported incidence of MU after RYGB ranges between 0.6% and 16%, and is more frequently diagnosed in smokers than nonsmokers.3,4 The true incidence of MU is difficult to assess, because many patients are asymptomatic. Management of MU varies from medical treatment with proton pump inhibitors, H2-receptor blockers, and sucralfate, to endoscopy or surgery, depending on the cause and complications.5 In the case of hemorrhagic MU, endoscopic control or laparotomy is usually the standard of care.4,6 Bleeding marginal ulcers following gastrojejunostomy are usually supplied by jejunal branches of the superior mesenteric artery or gastric branches of the celiac artery.7 We present a case of life-threatening hemorrhagic MU 18 months after conducting open RYGB. The bypass eroded into the main splenic artery and was managed with angiographic embolization followed by surgery.

CASE REPORT
A 65-year-old male with a history of diabetes and hyperlipidemia underwent an open RYGB at an outside institution 18 months prior to presentation. He was not on chronic acid suppression therapy. At the emergency room, the patient presented with hematemesis and orthostatic hypotension. In the supine position, his blood pressure was 109/66 with a heart rate of 77. Orthostatic measurements were 86/54 and 89, respectively. His hematocrit was 21%. After initial fluid resuscitation and blood transfusion, the patient underwent esophagogastroduodenoscopy (EGD) that revealed large blood clots in the gastric pouch and bright red blood oozing from a marginal ulcer at the gastrojejunal (GJ) anastomosis. The patient received multiple transfusions of packed red blood cells and a pantoprazole infusion. Adequate visualization and endo-
scopic hemostasis could not be performed and repeat endoscopy was also unsuccessful. Urgent angiography did not reveal any active bleeding. Subsequently, the hematocrit stabilized, and the patient seemed to recover for the next 5 days. At that point, the patient developed an abrupt onset of severe hematemesis. EGD showed recurrent bleeding from the GJ anastomosis. Mesenteric angiography revealed gross extravasation from the mid splenic artery (Figure 1A), which was successfully embolized. Following embolization, the distal splenic artery could not be visualized, and the splenic parenchyma could not be seen due to poor collateral flow (Figure 1B). Within 8 hours of embolization, the patient developed abdominal pain, distention, and lactic acidosis. An abdominal CT scan revealed pneumoperitoneum with high attenuation ascites representing contrast and hemorrhage (Figure 2). The patient was urgently taken to the operating room for exploratory laparotomy. Approximately 2.5 liters of blood and clots were evacuated from the peritoneal cavity. With the aid of intraoperative endoscopy, a large defect was found in the gastric remnant, the gastric pouch, and the proximal Roux limb of the jejunum. The Roux limb was completely detached from the gastric pouch. Gelfoam and coils were visualized in the lesser sac, and the splenic artery was non-pulsatile. The spleen appeared dusky; hence, splenectomy was performed. The gastric remnant, gastric pouch, and Roux limb of the jejunum (that included the perforations) were resected back to viable tissue. The remaining gastric pouch and the proximal Roux limb of the jejunum were subsequently reanastomosed utilizing a circular stapling technique. Intraoperative endoscopy revealed a patent, leak-free, anastomosis. The patient was discharged on the ninth postoperative day, after a negative Gastrografin swallow evaluation. At discharge, he tolerated an oral liquid diet. During the postoperative course, he developed a left upper quadrant intraabdominal abscess requiring percutaneous drainage and intravenous antibiotics.

**DISCUSSION**

Marginal ulceration at the gastrojejunual anastomosis is the most common cause of early and late upper GI bleeding following RYGB. Although gastrin levels are decreased following gastric bypass, vagal stimulation is preserved, as is the gastric pouch's ability to produce acid. Unlike the
duodenum, the jejunum’s ability to buffer acid via bicarbonate secretion is poor, further contributing to the risk of gastrojejunal marginal ulcers. The risk of postoperative MU is also increased by factors such as H. pylori infection, nonsteroidal anti-inflammatory drugs (NSAID), antiacoagulation, and smoking. Technical factors that increase the risk of MU formation include a large gastric pouch, non-absorbable sutures, and tissue ischemia at the staple line. Patients suffering from MU following RYGP usually present with epigastric pain and occasional nausea and emesis. Clinically significant upper GI hemorrhage is not usually observed. The diagnosis of MU is performed endoscopically. Uncomplicated MU is generally managed endoscopically or surgically, after initiating intravenous formulations of proton pump inhibitor therapy. Preventive low-dose PPI or H2-blocker therapy is a common practice following RYGB. Triple therapy is usually started in case of H. pylori infection. Cessation of smoking is mandatory and NSAIDS should be discontinued, if possible. Hemodynamically significant upper GI bleed secondary to MU is managed endoscopically or surgically, after initiating intravenous formulations of proton pump inhibitor therapy. At our institution, an 80-mg intravenous bolus of pantoprazole followed by an infusion at a rate of 8mg/hr is used. Endoscopic options include heater probe coaptive coagulation, bipolar probe coaptive coagulation, chemical sclerosant, epinephrine injection, laser therapy, and hemostatic clip placement. Surgical management is usually required if endoscopic therapy fails to adequately control the bleeding. Angiographic interventions are generally considered for bleeding secondary to traditional peptic ulcer disease, especially when patients are at high risk for surgery. There are no reports in the literature describing angiographic intervention for the control of bleeding marginal ulcers. In a series of 18 patients, Ljungdahl et al reported successful management of massive gastric and duodenal bleeding with transcatheter selective arterial embolization in 17 of those patients. All cases involved bleeding from the left gastric or gastroduodenal arteries or their branches. No case involved the splenic artery, nor was marginal ulceration a cause of the bleeding in any of the reported cases. When needed, surgical management can be performed using the open or laparoscopic technique and involves excision of the gastrojejunalostomy, including the ulcerated areas and construction of a new gastrojejunalostomy. During the procedure, the bleeding vessels must be meticulously identified and ligated. The current case, reports a novel approach to the management of clinically significant hemorrhagic MU, involving the splenic artery. Pre-operative control of bleeding is a key step given the amount of inflammation present in patients with MU. Attention should be paid to collateral circulation causing hemorrhage following embolization of a visceral artery, requiring trial of different embolic agents for adequate bleeding control or immediate surgery.

**CONCLUSION**

We describe a unique life-threatening complication of marginal ulceration following RYGB. We also propose a novel approach to its management. Massive upper GI bleeding, due to marginal ulcer eroding into the main splenic artery, can be controlled preoperatively with angiographic selective embolization to allow successful surgical repair. This strategy should also be considered in patients with hemodynamic instability.

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