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Online case report

Gastrointestinal bleeding from the excluded stomach: a proposed algorithmic approach to management

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The Roux-en-Y gastric bypass (RYGB) is the gold standard for surgical treatment of obesity and its related co-morbidities [1]. Outcomes after RYGB are associated with sustained weight loss and a decrease in long-term mortality associated with resolution of co-morbidities [2,3]. Although the benefits of RYGB are substantial, the short- and long-term risks are anastomotic leak (3%), deep vein thrombosis or pulmonary embolism (3%), anastomotic strictures (5%), ulcers (2%), early and late gastrointestinal (GI) bleeding (3%), incisional hernias, and small bowel obstruction (2%) [4,5].

GI hemorrhage occurs at a frequency of 1.93% after laparoscopic RYGB in the early postoperative period and may originate from the excluded stomach, the gastrojejunostomy, or the jejunojejunostomy staple lines [5]. Conversely, late postoperative GI hemorrhage from the excluded stomach or duodenum is quite uncommon and can be potentially fatal [6]. This specific location of GI bleeding poses both diagnostic and therapeutic difficulties and limitations due to the inability to identify the source of bleeding.

We report a case of a 61-year-old male, who underwent RYGB 10 years before his presentation to the emergency room with a melena. The treatment for this scenario requires complex clinical thought and algorithms because this is a rare and potentially fatal complication.

Case presentation and management

This is a case of a 61-year-old male patient, status postRYGB 10 years ago for morbid obesity. preRYGB weight was 445 pounds (201 kg), and current weight is 279 pounds (127 kg). He reported a 3-year history of recurrent GI bleeding. He was not taking a proton pump inhibitor or histamine blocker for prevention of ulcer disease. The patient denied tobacco, recreational drug, or nonsteroidal anti-inflammatory drug use and admitted to an occasional glass of red wine. The patient was admitted to a local hospital with progressive weakness, shortness of breath, colicky epigastric pain, and intermittent episodes of melena for 4 days. The patient's Helicobacter pylori status was unknown. His labs showed evidence of anemia (Hb = 7.0 mg/dL), and a coagulation profile within normal limits. The patient underwent an esophagogastroduodenoscopy (EGD), a colonoscopy, and a tagged red blood cell scan, in an attempt to localize the GI bleeding. These attempts were unsuccessful in establishing a source. The patient received a total of 26 units of packed red blood cells (PRBCs) at the outside hospital before being transferred to our institution for further management of his case.

Upon arrival, the patient presented with the same symptoms and showed signs of hemodynamic instability (heart rate = 135, systolic blood pressure = 80–105). Laboratory studies showed persistence of his anemia (Hb = 6.7 mg/dL) despite 3 more units of PRBCs. The patient then underwent massive resuscitation measures with intravenous fluids and blood transfusions and was started on a pantoprazole (80 mg in .9% normal saline [NS] at 100 mL/hr) and octreotide drip (25 mcg/hr). An EGD was performed,

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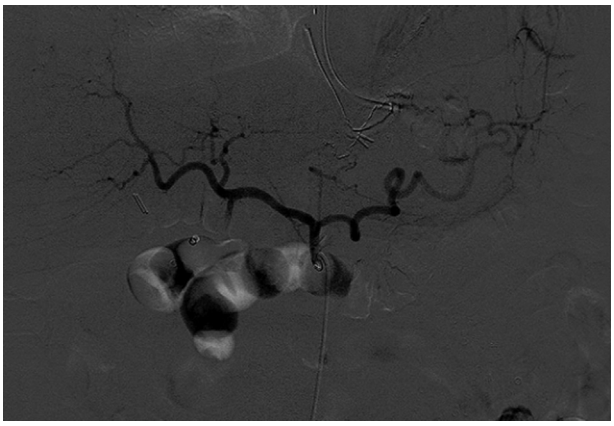

Fig. 1. Celiac angiography showing a stump at the level of the gastroduodenal artery.

but it did not reveal the source of bleeding; however, the exam was limited by the inability to access the biliopancreatic limb and the excluded stomach. The patient continued bleeding, and his hemoglobin level dropped again from 10.2 mg/dL to 7.3 mg/dL. He was again aggressively resuscitated and interventional radiology (IR) was involved. IR made multiple attempts to identify the source of active bleeding. These attempts failed to identify the source. However, on careful review of the angiography, a short gastroduodenal artery stump (Fig. 1) was considered suspicious by our team. The patient was reevaluated by IR during an active episode of melena, during which celiac angiography was again performed. During this exam, both the gastroduodenal artery and the pancreaticoduodenal (collateral flow) arteries arising from the superior mesenteric artery showed active bleeding and were coiled successfully (Fig. 2). To verify the source of bleeding had indeed been addressed, serial hemoglobin and a repeat balloon EGD were undertaken. The repeat EGD was successful in intubating the biliopancreatic limb; however, we were unable to reach to the duodenum and exclude stomach. Although we could not visualize the bleeding ulcer, we did not see any fresh blood in the biliopancreatic limb. The patient's hemoglobin remained stable throughout the remaining hospital stay, and the patient was discharged home after 6 days. Of note, the patient was treated empirically for H. pylori until serum antibody/antigen studies returned as negative.

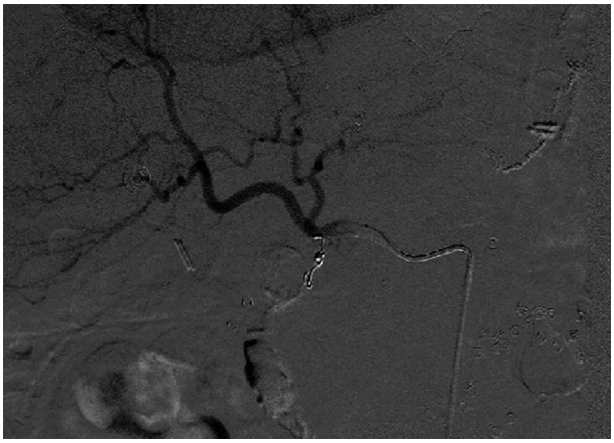

Fig. 2. Coiling of the gastroduodenal artery.

Discussion

The most common cause of upper GI bleeding in the late postoperative period in patients who have undergone RYGB is marginal ulceration, and the incidence is between .6% and 16% [7]. Other rare causes, such as in this case, are due to peptic ulceration in the excluded structures [6,8–10]. Studies conducted on animals and humans have shown decreased acid production within the new gastric pouch, but there was maintenance of acid secretion in the excluded bowel and gastric remnant favoring the development of ulcers [11–13].

Some studies, such as those conducted by Mehran et al., have addressed the issue of acute early postoperative bleeding in patients who have undergone RYGB [14,15]. Guidelines for acute bleeding from the excluded stomach and duodenum in the late postoperative period have not been established yet. We implemented this algorithm (Fig. 3), and we feel that more research should be done to further validate and solidify its outcomes. In stable patients, a full history taking and physical examination should be performed, with emphasis on risk factors such as tobacco use, nonsteroidal anti-inflammatory drug use, alcohol consumption, and H. pylori infection.

For hemodynamically unstable and/or anemic patients, resuscitation with intravenous fluids and PRBCs should be initiated and an experienced gastroenterology and interventional radiology team should be involved immediately in the patient's care. Simultaneously, a proton pump inhibitor drip should be started with the possible addition of a vasopressin or octreotide drip to shunt blood away from the somatic blood flow [15–17]. EGD by an experienced gastroenterologist should be done to identify the source of the hemorrhage. If the source is identified, proceeding with endoscopic hemostatic techniques, including epinephrine injection, sclerotherapy, cauterization, or mechanical tamponade, should be performed [18]. If the source cannot be identified, the bleeding cannot be stopped, or the bleeding recurs, the patient should be resuscitated and taken to the angiography suite with an interventional radiologist, to identify the bleeding vessel, followed by embolization [19,20].

According to a review by Loffroy et al., the clinical success rate of transarterial embolization of an acute nonvariceal upper gastrointestinal hemorrhage ranges between 63% and 97% [19]. If the source was neither localized nor controlled at the time of angiography because the source was not actively bleeding, techniques can be implemented to encourage active bleeding during the radiological