

A Rare Case of Perforated Gastric Remnant after Roux-en-Y Gastric Bypass

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ABSTRACT

Morbid obesity is a major health problem worldwide and the Roux-en-Y gastric bypass (RYGB) is a popular surgical treatment. A 40 year old woman, 17 years post RYGB presented with concurrent symptomatic gastric ulcers and acute pancreatitis. The diagnosis was difficult and after failed medical therapy, surgery was required. The gastric remnant is not easily accessible by endoscopy thus; detecting PUD in this excluded segment is difficult. As a result, alternative methods, such as exploratory laparotomy or pediatric endoscopy may be required to effectively diagnose the gastric remnant.

Keywords: Obesity, perforated gastric ulcer, peptic ulcer disease, roux-en-Y gastric bypass.

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I. INTRODUCTION

Morbid obesity is a major health problem worldwide, affecting nearly 5% of the population in the United States [1]. It is defined as a weight of over 100 pounds from the ideal body weight, a body mass index (BMI) of 40 or more, or a BMI of 35 or more and experiencing obesity-related health conditions. The rise in the prevalence of morbid obesity has made Roux-en-Y gastric bypass (RYGB) a popular surgery with more than half a million procedures a year worldwide to treat this condition [2]. The fate of the excluded stomach, also known as the gastric remnant can still develop various complications and pathologies [3]. The following case describes a perforated ulcer in a gastric remnant causing necrotizing pancreatitis.

II. CASE REPORT

A 40-year-old female presented in April 2021 with the complaint of sudden abdominal pain located on the left upper quadrant radiating to the epigastric area. She had a past medical history significant for morbid obesity (status post roux-en-Y gastric bypass surgery in 2004), cholecystitis

(status post cholecystectomy in 2002) and pancreatitis in 2003 that was deemed to be of unknown etiology. She was not a smoker and denied any alcohol or illicit drug use. She had a history of heavy non-steroidal anti-inflammatory drugs (NSAID) intake for chronic joint pain.

Initial physical examination included normal vital signs and was remarkable for abdominal tenderness on palpation of the left upper quadrant. Pertinent laboratory examination results were: leukocytes 12,000/mm (3), hemoglobin was 11.6 g/dL, lipase 16 U/L, triglycerides 81 mg/dL and normal alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Initial computer tomography (CT) scan of the abdomen showed confluent stranding surrounding the pancreas, extending into the perisplenic and left perinephric region that was concerning for pancreatitis. There was also significant distention of the gastric remnant, which was concerning for dehiscence of the anastomosis between the remnant and the gastric pouch.

The patient was evaluated by the Bariatric surgery team, and as per their assessment the patient's presentation was unlikely to be from dehiscence of the anastomosis of the gastric bypass, hence the running diagnosis was that of pancreatitis.

On day 3 of hospitalization, the patient developed melena and her hemoglobin dropped from 11.6 g/dL to 8.8 g/dL. She was taken for an EGD, which showed a normal appearing roux-en-y gastrojejunostomy characterized by a healthy appearing mucosa and gastrojejunal anastomosis. There were no findings to explain melena and the patient was returned to the floor for ongoing management of pancreatitis with the plan to perform a colonoscopy the next day. Overnight, the patient started complaining of worsening abdominal pain. Vital signs were remarkable for tachycardia and fever, and on a physical exam she had rebound tenderness. Laboratory studies showed leukocytosis (white blood cell count, 20,000 /mm (3)) and elevated lactic acid of 3.7 mmol/L.

A CT abdomen (Fig. 1) showed findings that were consistent with that of pancreatitis.



Fig. 1. CT scan findings consistent with that of pancreatitis with a separate fluid collection wrapping around the excluded stomach with an air-fluid level, which was concerning for a pseudocyst (arrow).

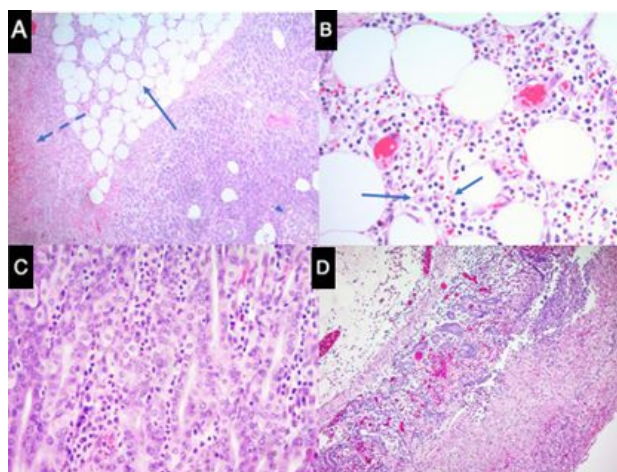


Fig. 2. (A) Sections of pancreas at 40x magnification show the usual acinar architecture with an islet of Langerhans denoted by the arrowhead. There is infiltration and destruction of the parenchyma by acute and chronic inflammatory cells, adjacent fat necrosis (solid arrow), and fibrin deposition and hemorrhage (dashed arrow), consistent with necrotizing pancreatitis. (B) At 200x magnification, the fat cells are diminished in size with loss of nuclei and infiltration by mixed inflammatory cells, including lipid-laden, or foamy, macrophages (arrows), consistent with fat necrosis. (C) Sections of the stomach show diffuse acute inflammation within the lamina propria with reactive epithelial cells. (D) There is focal ulceration, hemorrhage, and coagulative necrosis of the mucosa at the perforation site.

The patient was taken for an emergent exploratory laparotomy the gastric remnant was found to be necrotic in appearance with a large perforation along the posterior wall. Dissection into this area gave way to the fluid collection,

which was foul-smelling, bloody and purulent. The gastric remnant was filled with blood and was adherent to the pancreas which appeared necrotic. It was not clear whether the gastric remnant had perforated into the pancreas, or if the pancreatitis had led to the perforation of the stomach. The patient underwent subtotal gastrectomy, distal pancreatectomy as well as splenectomy due to concern for involvement of the splenic vessels.

Histologically the gastric mucosa showed signs of active chronic gastritis with ulcer formation near the site of perforation and the pancreas showed signs of necrotizing pancreatitis (Fig. 2).

Patient's recovery has been prolonged and has been complicated by intra-abdominal infections and leak of the gastric anastomosis sutures, which is being managed with a non-surgical approach.

III. DISCUSSION

Perforated gastric ulcers and acute pancreatitis are frequent pathologies, however the presence of the two pathologies simultaneously is unusual. The patient we describe above was found to have a perforated gastric ulcer as well as necrotizing pancreatitis. It is likely that the presence of one led to the other, however it is not clear what came first.

Enteric perforations are a rare complication of necrotizing pancreatitis and have been described in only a few case reports. Reference [4] reported necrotizing pancreatitis leading to gastric perforation. Reference [5] described a patient with acute pancreatitis and gastric perforation, which was caused by gastric erosion and rupture of a pseudoaneurysm. Reference [6] reported a case of acute pancreatitis and pseudocyst formation complicated with gastric perforation caused by ischemic mucosal injury.

The mechanism we initially proposed was that the chronic history of NSAID use inevitably led to the formation of gastric ulcers which perforated into the pancreatic parenchyma causing necrotizing pancreatitis. This is an extremely rare complication of gastric perforations and to date has only been reported by [7] whereby a patient with kissing gastric ulcers had a posterior perforation that was a suspected cause of acute pancreatitis. However, upon reviewing the pathology of the gastric mucosa, it appeared as if the gastric perforation was caused by the pancreatitis. The wall of the stomach near the perforation was atrophic which is compatible with chronic ischemic changes. The mucosa, in contrast, has diffuse acute inflammatory cells with focal areas of necrosis near the perforation which is suggestive of acute ischemic necrosis (Fig. 2). Since the wall has more chronic changes than the mucosa, it likely seems to be an outside-in process.

IV. CONCLUSION

Although RYGB is an effective treatment for weight loss in morbidly obese patients, it is important to be aware of the possible complications associated with the procedure. Specifically, the gastric remnant still remains susceptible to ulcer formation and subsequent perforation, which can be life-threatening. As the gastric remnant is not easily

accessible by endoscopy, detecting PUD in this excluded segment is difficult. As a result, alternative methods, such as exploratory laparotomy or pediatric endoscopy, may be required to effectively diagnose and treat PUD affecting the gastric remnant.

CONFLICT OF INTEREST

Authors declare that they do not have any conflict of interest.

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