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Case report

Acute gastric remnant dilation after laparoscopic Roux-en-Y gastric bypass operation in long-standing type I diabetic patient: case report and literature review

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Acute gastric remnant dilation is a rare postoperative complication of laparoscopic Roux-en-Y gastric bypass (LRYGB) occurring in 0–0.8% of cases [1–7]. It is usually associated with jejunojejunostomy obstruction or gastric remnant ulcer formation. The typical presenting symptoms include left upper quadrant abdominal pain, hiccups, and persistent tachycardia. It is managed by gastrostomy tube decompression, and relief of distal obstruction. Delayed diagnosis may lead to gastric remnant perforation, organ failure, sepsis, and death.

We encountered 1 case of acute gastric remnant dilation in a young patient with type 1 diabetes mellitus after an uneventful laparoscopic LRYGB. This complication was not associated with concomitant jejunojejunostomy stricture or distal bowel obstruction. The patient denied having any autonomic neuropathy or symptoms of gastroparesis preoperatively. However, she was later found to have severe hypomotility of the gastric remnant.

To our knowledge, this is the first reported case of isolated gastric remnant dilation after LRYGB in the English literature.

Case Report

The patient was a 34-year-old woman with a body mass index of 38 kg/m^2, who presented for bariatric surgery after failing numerous weight loss programs. Her past medical history was significant for type 1 diabetes, hypothyroidism, degenerative joint disease, and two cesarean sections. Her medications consisted of insulin and levothyroxine.

After appropriate preoperative evaluation and education, the patient underwent uneventful antecolic/antegastric LRYGB. A 30 cm^3 gastric pouch was created. Gastrojejunostomy and jejunojejunostomy anastomoses were performed using linear staplers. The alimentary and biliopancreatic limbs were measured to 80 and 30 cm, respectively, and the mesenteric defects were not closed.

The patient had an unremarkable postoperative course and was discharged home on postoperative day 3. Two days after discharge, the patient presented to the emergency room with watery diarrhea, nausea, and vomiting. She denied any abdominal pain, respiratory distress, fever, hematemesis, hiccups, or abnormal blood sugar values at home. On evaluation, she appeared to be dehydrated and tachycardic with benign abdominal examination findings. The initial laboratory results are outlined in Table 1. To confirm that her clinical presentation was not due to gastroenteritis or antibiotic-induced colitis, contrast-enhanced computed tomography of the abdomen was performed, revealing biliopancreatic limb dilation extending to the second part of the duodenum (Figs. 1 and 2).

Because the patient continued to deteriorate clinically, with worsening metabolic acidosis despite aggressive resuscitation and medical management, we decided to perform re-exploration rather than pursuing less aggressive measures such as placing a percutaneous gastrostomy tube under radiologic guidance. The differential diagnoses included internal hernia, acute gastric remnant dilation, anastomotic leak, and even ischemic bowel.

In the operating room, an arterial line was placed, and the initial blood gas results revealed a pH of 7.0, with a base deficit of 22. Systematic laparoscopic exploration revealed intact gastrojejunostomy and jejunojejunostomy anastomo-

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Table 1
Laboratory values

White blood cell count	16,840/mm^3
Sodium	130 mmol/L
Hemoglobin	14.6 g/100 mL
Potassium	5.9 mEq/L
Hematocrit	43%
Chloride	106 mEq/L
Platelets	547,000 μL
Bicarbonate	8 mmol/L
Blood urea nitrogen	7 mg/dL
Creatinine	1.1 mg/dL
Glucose	330 mg/dL

ses. No evidence of an anastomotic stricture was found. The gastric remnant and duodenum proximal to the ligament of Treitz were dilated. The alimentary limb, common channel, and distal small bowel were all nondilated and intact. No extraluminal succus entericus to suggest perforation nor any evidence of an internal hernia was found. A 20F gastrostomy tube was placed in the gastric remnant, and a copious amount of gray fluid was suctioned out.

The postoperative hospital course was uneventful with rapid resolution of the metabolic acidosis. On postoperative day 7, an attempt to clamp the gastrostomy tube failed, because the patient experienced recurrent nausea. A gastrograffin upper gastrointestinal study revealed severe hypomotility of the gastric remnant with a 2-hour contrast transit time out of the stomach (Figs. 3 and 4). Metoclopromide therapy was initiated, and the patient was discharged home with the gastrostomy tube in place, tolerating a liquid protein diet.

Under the guidance of her gastroenterologist, her subsequent medical therapy was changed to erythromycin and tegaserod maleate (Zelnorm). The gastrostomy tube was eventually downsized. An outside gastrograffin upper gastrointestinal study performed 4 months later by way of the gastrostomy tube demonstrated slow emptying of the contrast into the small bowel with no evidence of obstruction. An outside nuclear gastric emptying study revealed 37% emptying of gastric contents at 90 minutes. The normal reference range for this study is >50% gastric emptying at 90 minutes. The gastrostomy tube was finally removed 5 months after the second operation. The patient was asymptomatic and her medical therapy was being self tapered at her last follow-up visit. She was tolerating a normal diabetic diet and had achieved 91% excess weight loss.

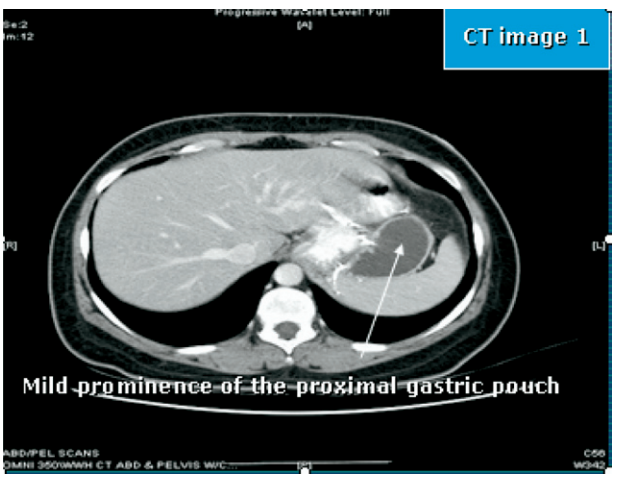

Fig. 1. Computed tomography (CT) scan showing mild prominence of proximal gastric pouch.

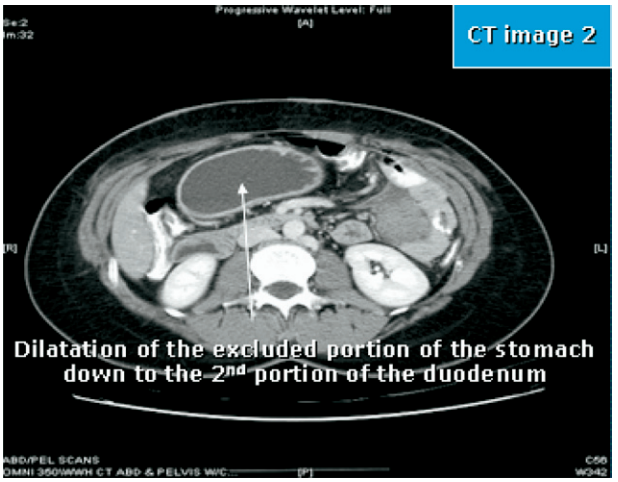

Fig. 2. Computed tomography (CT) scan showing dilation of excluded portion of stomach down to second portion of duodendom.

Discussion

Abnormal gastric emptying is a frequent complication of diabetes. Gastric emptying of solid or nutrient liquid is slow in about one half of outpatients with longstanding type 1 diabetes mellitus [8].

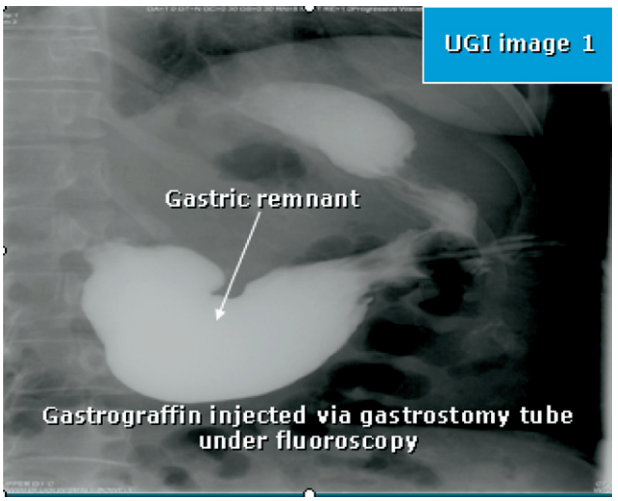

Fig. 3. Upper gastrointestinal (UGI) image showing gastrograffin injected by way of gastrostomy tube under fluoroscopic guidance.

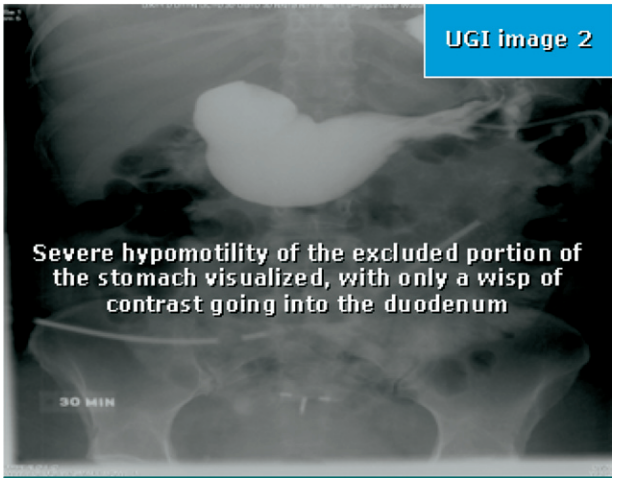

Fig. 4. Upper gastrointestinal (UGI) image showing severe hypomotility of excluded portion of stomach visualized with only wisp of contrast going into duodenum.

Gastroparesis is defined as the delayed emptying of a solid meal. The prevailing theory in diabetic gastroparesis implicates insulin deficiency and hyperglycemia in patients with diabetes, causing metabolic and vascular abnormalities that result in damage to the nerve fibers, especially the vagus nerve. The frequency of electrical waves through the stomach decreases and slows, and steady muscular contractions are lost or diminished [9]. Most of the patients with impaired gastric motility remain asymptomatic [10]. The relationship between the symptoms of gastroparesis and the rate of gastric emptying appears to be weak [10–12].

Even though our patient had long-term type 1 diabetes, she denied experiencing symptoms of autonomic neuropathy such as postural hypotension, diarrhea, vomiting, bladder paresis, or gustatory sweating [13]. It is generally thought that slow gastric emptying in patients with type 1 diabetes is related to the degree of autonomic neuropathy [14–16]. She also denied having any symptoms of gastroparesis, such as anorexia, early satiety, or postprandial abdominal fullness and discomfort. On the basis of the preoperative evaluation, which failed to reveal any symptoms of autonomic neuropathy or gastroparesis, the index of suspicion was very low that this patient had diabetic gastropathy.

Our patient's presentation was atypical, with no abdominal pain or fullness. Computed tomography of the abdomen was helpful because it confirmed our suspicion of a more severe problem than gastroenteritis or antibiotic-induced colitis. In contrast to other reported cases of gastric remnant dilation, our patient's intraoperative findings were not consistent with afferent loop obstruction or ileus [11].

This case has demonstrated a postoperative complication that could have resulted in a catastrophic outcome had the medical and surgical interventions not been instituted in a timely manner. In morbidly obese patients, the presence of overt peritoneal signs is usually ominous, and sepsis, organ failure, and death may soon follow. We chose immediate surgical exploration rather than simple percutaneous drainage of the dilated remnant stomach by interventional radiology or some other form of conservative therapy. This case further highlights the need for extreme vigilance and a low threshold for aggressive intervention in the postoperative period after bariatric surgery. It also raises questions as to whether a need exists to preoperatively screen all patients with long-term type 1 diabetes using a gastric emptying study and whether gastrostomy tube placement should become a routine part of LRYGB surgery in those patients diagnosed with delayed gastric emptying or type 1 diabetes.

References

[1] Oliak D, Ballantyne GH, Weber P, Wasielewski A, Davies RJ, Schmidt HJ. Laparoscopic Roux-en-Y gastric bypass: defining the learning curve. Surg Endosc 2003;17:405–8.
[2] Schauer P, Ikramuddin S, Hamad G, Gourash W. The learning curve for laparoscopic Roux-en-Y gastric bypass is 100 cases. Surg Endosc 2003;17:212–15.
[3] Suter M, Giust V, Heraief E, Zysset F, Calmes JM. Laparoscopic Roux-en-Y gastric bypass: initial 2-year experience. Surg Endosc 2003;17:603–9.
[4] DeMaria EJ, Sugerman HJ, Kellum JM, Meador JG, Wolfe LG. Results of 281 consecutive total laparoscopic Roux-en-Y gastric bypasses to treat morbid obesity. Ann Surg 2002;235:640–7.
[5] Higa KD, Boone KB, Ho T, Davies OG. Laparoscopic Roux-en-Y gastric bypass for morbid obesity: technique and preliminary results of our first 400 patients. Arch Surg 2000;135:1029–33.
[6] Dresel A, Kuhn JA, Westmoreland MV, Talaasen LJ, McCarty TM. Establishing a laparoscopic gastric bypass program. Am J Surg 2002; 184:617–20.
[7] Papasavas PK, Caushaj PF, McCormick JT. Laparoscopic management of complications following laparoscopic Roux-en-Y gastric bypass for morbid obesity. Surg Endosc 2003;17:610–14.
[8] Keshavarzian A, Ilber FL, Vaeth L. Gastric emptying in patients with insulin-requiring diabetes mellitus. Am J Gastroenterol 1987;82:29–35.
[9] Feigenbaum K. Update on gastroparesis. Gastroenterol Nurs 2006; 29:239–44.
[10] Abrahamsson H. Gastrointestinal motility disorders in patients with diabetes mellitus. J Intern Med 1995;237:403–9.
[11] Migdalis L, Thomaides T, Chairopoulos C, Kalogeropoulou J, Martzara F. Changes of gastric emptying rate and gastrin levels are early indicators of autonomic neuropathy in type 2 diabetic patients. Clin Auton Res 2001;11:259–63.
[12] Horowitz M, Fraser R. Disordered gastric motor function in diabetes mellitus. Diabetologia 1994;37:543–51.
[13] Watkins PJ, Buxton-Thomas MS, Howard ER. Long-term outcome after gastrectomy for intractable diabetic gastroparesis. Diabet Med 2003;20:58–62.
[14] Merio R, Festa A, Bergmann H, et al. Slow gastric emptying in type 1 diabetes: relation to autonomic and peripheral neuropathy, blood glucose, and glycemic control. Diabetes Care Mar 1997;20:419–23.
[15] Stacher G. Impaired gastric emptying and altered intragastric meal distribution in diabetes mellitus related to autonomic neuropathy? Dig Dis Sci 2003;48:1027–34.
[16] Buysschaert M, Moulart M, Urbain JL, et al. Impaired gastric emptying in diabetic patients with cardiac autonomic neuropathy. Diabetes Care 1987;10:448–52.