

# A case of chronic exocrine pancreatic insufficiency in a gastric bypass patient

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## Abstract

Exocrine pancreatic insufficiency (EPI) is a significant complication of bariatric surgery, but its frequency and outcomes are not well-studied. If EPI is not diagnosed, it can result in nutritional deficiencies, dehydration, and acute kidney damage. Our patient, a 47-year-old woman, underwent Roux-en-Y gastric bypass (RYGB) surgery due to morbid obesity four years ago and lost contact with outpatient follow-ups. She came to us presenting chronic diarrhea and fatigue and was diagnosed with chronic pancreatic exocrine deficiency based on a low fecal elastase level. After starting her on pancreatic enzymes, her symptoms resolved. It is crucial to maintain a high degree of suspicion in order to diagnose EPI in patients who have undergone RYGB. The fecal elastase test, which is both reliable and inexpensive, is an effective diagnostic tool for EPI; prompt treatment can alleviate symptoms within days.

**Keywords:** exocrine pancreatic insufficiency, Roux-en-Y gastric bypass, fecal elastase, pancreatic enzyme replacement therapy

## Introduction

Exocrine pancreatic insufficiency (EPI) is a common complication following Roux-en-Y gastric bypass (RYGB) surgery. Diagnosis can be confirmed via a positive fecal elastase-1 test or noticeable improvement following pancreatic enzyme replacement therapy [1]. Fecal elastase is a highly accurate diagnostic test for EPI [2]. Post-operative diarrhea is a common issue after this type of surgery, with potential causes including dumping syndrome, short bowel syndrome, small intestinal bacterial overgrowth (SIBO), malabsorption of bile acids or carbohydrates, and EPI.

EPI, which can result from RYGB due to irregular secretion of pancreatic enzymes, features additional symptoms such as fat maldigestion and steatorrhea. These occur due to procedure-related changes [3]. Therefore, EPI should always be considered as a potential cause of post-operative diarrhea following RYGB surgery.

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### Informed Consent

The authors stated that the written consent was obtained from the patient presented with images in the study.

### Conflict of Interest

No conflict of interest was declared by the authors.

### Financial Disclosure

The authors declared that this study has received no financial support.

### Published

2024 November 12

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## Case presentation

A 47-year-old female patient who previously underwent RYGB surgery for morbid obesity 4 years prior presented with complaints of loose stool, fatigue, headache, and nausea that had persisted for 2 weeks. She had not been regularly followed up on an outpatient basis since her surgery. Daily, she experienced six to eight episodes of greasy diarrhea, which worsened after consuming fatty meals. The patient denied any unintentional weight loss or instances of melena. She reported fatigue and progressive weakness, becoming tired even with minimal activity. Her appetite had significantly declined since the onset of these symptoms, and nausea limited her ability to eat and drink.

During the presentation, her vital signs were recorded as follows: temperature 35.5°C, pulse 84 beats per minute, respiratory rate 18 breaths per minute, and blood pressure 122/71 mmHg with a mean arterial pressure of 99. The physical examination was largely normal, the exception being conjunctival pallor and lower abdominal tenderness; however, there was no abdominal rigidity or guarding.

Her complete blood count (CBC) was mostly normal, apart from normocytic anemia, with a hemoglobin level of 7.7 g/dl. The liver function tests showed regular results, with a slight increase in aspartate aminotransferase (AST). Her creatinine level was high (3.6 mg/dl, compared to a usual range of 1.5–1.7 mg/dl). The bicarbonate was 7 mEq/L at admission, and the anion gap was 9. Venous blood gas (VBG) displayed a pH of 7.05 and a  $\text{pCO}_2$  of 27.

Her iron studies were standard, except for a mild decrease in serum iron to 38  $\mu\text{g/dl}$ . The total iron binding capacity was 272  $\mu\text{g/dl}$ , with transferrin saturation at 14% and ferritin at 15 ng/ml. Vitamin B12 and folate levels were standard at 598 pg/mL and 13.7 ng/mL, correspondingly. The thyroid stimulating hormone (TSH) was 1.811 mIU/L. However, her vitamin D level was critically low at 10 ng/mL.

The urinalysis indicated hazy urine, displaying positive results for both urine nitrite and leukocyte esterase, accompanied by an excess count of white blood cells (WBC) beyond measurable limits in the high-power field. The urine culture revealed pan-sensitive *E. coli*. A computed tomography (CT) scan of the chest, abdomen, and pelvis, performed without intravenous contrast, displayed no acute conditions. No abnormalities were spotted on the chest X-ray and renal ultrasound. There was an increase in stool fat, and the Fecal Pancreatic Elastase was recorded below 10  $\mu\text{g/g}$ , a reduction from 23  $\mu\text{g/g}$  during her admission 18 months previously. Evaluations for stool WBC, C difficile, stool ova, and parasites returned negative. Additionally, both stool and blood cultures showed no growth.

The esophagogastroduodenoscopy (EGD) revealed a gastric bypass with a small pouch and an intact staple line. It showed a gastrojejunal anastomosis with a healthy-looking mucosa and a standard esophagus. During the colonoscopy, the terminal ileum and colon appeared normal. Biopsies were taken from the right and left colon for histology. The colonic biopsy demonstrated an acute colitis pattern with preserved architecture of the colonic mucosa and several occurrences of cryptitis and crypt abscess. However, no features of chronicity, granulomatous inflammation, dysplasia, or malignancy were observed.

Given her lab results and medical history, she was initially diagnosed with acute kidney injury (AKI) superimposed on chronic kidney disease (CKD), normal anion gap metabolic acidosis, chronic diarrhea, urinary tract infection (UTI), and iron deficiency anemia.

The primary cause of the patient's symptoms was EPI, which led to diarrhea. This was verified by lab results, leading to the initiation of a pancrelipase (Creon) treatment. The patient was advised to eat small, frequent meals and to take pancreatic lipase with each meal and snack. A dosage of pancrelipase 24,000 Units was prescribed: two tablets three times a day with meals and one tablet two times a day with snacks. Following two days of this treatment, the patient noted a substantial improvement in their condition. There were no subsequent episodes of diarrhea, nausea, vomiting, or abdominal pain.

The presumed cause of AKI in CKD was chronic diarrhea, which led to acute tubular necrosis and acute interstitial nephritis. After proper hydration and cessation of her diarrhea and Naproxen use, her creatinine levels improved to 1.5 mg/dl.

The assumption was that the normal anion gap metabolic acidosis resulted from chronic diarrhea and renal tubular acidosis. Following sufficient diarrhea control and sodium bicarbonate treatment, the bicarbonate and VBG pH became normal at 19 mEq/l and 7.28, respectively. Outpatient sodium bicarbonate treatment was maintained.

She began taking ferrous sulfate for iron deficiency anemia and calcium carbonate and ergocalciferol for vitamin D deficiency. Multivitamins containing folic acid and thiamine mononitrate were also resumed as nutritional supplements following her gastric bypass.

After 5 days of inpatient therapy and four days of pancrelipase therapy, she was discharged. She was advised to follow up at an outpatient gastroenterology clinic. Her symptom resolution remained consistent after 6 months.

## Discussion

Adults with a body mass index (BMI) of 35  $\text{kg/m}^2$  or higher are suggested to undergo bariatric surgery, whether they have obesity-related comorbidities or not. This procedure is also recommended for diabetic patients with a BMI of at least 30  $\text{kg/m}^2$ . Bariatric surgery's benefits on weight loss, comorbidity improvement, cancer risk reduction, and long-term mortality are well-documented [4]. However, this surgery can cause complications like postprandial hypoglycemia, abdominal pain, anastomotic stenosis, deficiencies of iron, vitamins B12, folic acid and D, calcium deficiency, loss of bone density, and kidney stones [5]. Moreover, EPI is a common complication occurring in up to 41.6% of patients after such a procedure and is more prevalent in RYGB compared to sleeve gastrectomy [1].

In the given case, a patient who underwent RYGB 4 years ago due to morbid obesity (BMI  $\geq 40 \text{ kg/m}^2$ ) started showing signs of potential EPI, like diarrhea and steatorrhea, for 2 weeks. This prolonged diarrhea led to an AKI and normal anion gap metabolic acidosis. Iron deficiency and vitamin D deficiency can occur in up to 49% of patients who have undergone RYGB [6,7]. Our patient, who stopped taking her nutritional supplements, also showed deficiencies in iron and vitamin D levels.

Fecal elastase concentration testing is a cost-effective and straightforward method for diagnosing moderate to severe EPI. The patient's low levels of fecal elastase and significant improvement with pancreatic enzyme replacement therapy confirmed the EPI diagnosis. Prompt treatment with pancreatic enzyme replacement therapy led to a swift resolution of symptoms.

### Conclusion

EPI is a key complication of RYGB surgery. This condition may cause symptoms such as diarrhea and steatorrhea. If post-operative patients experience diarrhea, a high suspicion of this condition is necessary. Fecal elastase, an affordable and reliable diagnostic test, is used to confirm EPI. Based on the clinical symptoms and/or low fecal elastase levels, pancreatic enzyme replacement therapy can be initiated to alleviate symptoms.

### References

1. Moore HN, Chirco AR, Plescia T, Ahmed S, Jachniewicz B, Rajasekar G, et al. Exocrine pancreatic insufficiency after bariatric surgery: a bariatric surgery center of excellence experience. *Surg Endosc.* 2023 Feb;37(2):1466-75.
2. Löser CHR, Möllgaard A, Fölsch UR. Faecal elastase 1: a novel, highly sensitive, and specific tubeless pancreatic function test. *Gut.* 1996;39(4):580-6.
3. Capurso G, Traini M, Picicchi M, Signoretti M, Arcidiacono PG. Exocrine pancreatic insufficiency: prevalence, diagnosis, and management. *Clin Exp Gastroenterol.* 2019;12:129.
4. Eisenberg D, Shikora SA, Aarts E, Aminian A, Angrisani L, Cohen RV, et al. American Society of Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) Indications for Metabolic and Bariatric Surgery. *Obes Surg.* 2023 Jan;33(1):3-14.
5. Abdeen G, le Roux C. Mechanism Underlying the Weight Loss and Complications of Roux-en-Y Gastric Bypass. Review. *Obes Surg.* 2016;26:410-21.
6. Halverson JD. Micronutrient deficiencies after gastric bypass for morbid obesity. *Am Surg.* 1986;52(11):594.
7. Brodin RE, LaMarca LB, Kenler HA, Cody RP. Malabsorptive gastric bypass in patients with superobesity. *J Gastrointest Surg.* 2002;6:195-205.

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