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A 53-Year-Old Man Presenting with Pancreatic Exocrine Insufficiency 7 Years After Gastric Bypass Bariatric Surgery

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 53-year-old
Final Diagnosis: Pancreatic diseases
Symptoms: Dairrhea
Medication: —
Clinical Procedure: —
Specialty: Endocrinology and Metabolic

Objective: Unusual clinical course

Background: Pancreatic exocrine insufficiency (PEI) is a clinical condition characterized by reduced or inappropriate pancreatic enzymes and secretions. It can have a variable clinical presentation and can affect patient quality of life. PEI can be associated with pancreatic and nonpancreatic disorders. Pancreatic insufficiency is a recognized complication of bariatric surgery, but there is limited awareness. This report is of a 53-year-old man who presented with PEI 7 years after his initial bariatric surgery. Revision surgery resulted in the resolution of chronic diarrhea and progressive weight loss.

Case Report: A 53-year-old man who had gastric bypass surgery had developed pancreatic insufficiency 7 years after the surgery. This diagnosis was a challenge to make and therefore treat. A multi-modal approach and revision surgery helped resolve his symptoms.

Conclusions: Pancreatic insufficiency is a challenging complication to treat after bariatric surgery. Its management includes a multi-disciplinary approach, and such cases should be managed in dedicated bariatric units. This report has highlighted the importance of excluding PEI as a complication of bariatric surgery and its management.

Keywords: Bariatric Surgery • Exocrine Pancreatic Insufficiency • Pancreatitis, Chronic • Pancreatic Elastase

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Background

Obesity is a systemic disease that is associated with multiple comorbidities. Over the last decade, much attention has been drawn to the awareness of obesity and to the management of morbid obesity [1]. Morbid obesity is defined as body mass index (BMI) kg/m^2 >40 [2]. According to the World Health Organization's report, the prevalence of morbid obesity has doubled in the last 2 decades [3]. It is commonly associated with sequelae such as obstructive sleep apnea, hypertension, type II diabetes mellitus, and cardiovascular disease. Bariatric surgery has emerged as a solution to tackle the implications of morbid obesity, thereby reducing weight [4]. With more bariatric surgeries being performed worldwide, more long-term physiological changes after surgery have been studied and reported.

After bariatric surgery, the most commonly encountered complications include internal hernia, metabolic deficiency, gallstones, pancreatic insufficiency, and liver failure. Pancreatic exocrine insufficiency (PEI) is a less reported long-term complication after bariatric surgery. PEI is a challenging complication to diagnose and eventually treat [5].

PEI can be caused by pancreatic and non-pancreatic disorders. It is mostly a sequel to chronic pancreatitis. Due to chronic inflammation and fibrosis, there is decreased lipase secretion with PEI [6].

Alcoholism and autoimmune pancreatic conditions can also increase the risk of PEI. It can also occur due to non-pancreatic diseases such as diabetes, inflammatory bowel disease, celiac disease, and Sjogren's syndrome. In diabetes, microvascular damage can induce pancreatic fibrosis. Poor glycemic control and long-term insulin dependence can also play a part in causing pancreatic fibrosis [7].

Therefore, we are reporting a rare case of PEI in a 53-year-old male patient who developed features of PEI 7 years after his initial surgery. His case was managed in our specialized bariatric unit with a multimodal approach in treatment and revision surgery, which helped in resolving his symptoms and improving his quality of life.

Case Report

A 53-year-old man was referred to the bariatric services for morbid obesity with a BMI of 43 kg/m^2 in 2013. He had associated comorbidities that included type II diabetes mellitus and obstructive sleep apnea. After the necessary preparation and stabilization, he underwent Roux-en-Y gastric bypass (RYGB) surgery, with a biliopancreatic limb of 100 cm and a Roux limb of 80 cm. His postoperative recovery was uneventful, and he lost 70 kg in 18 months. He was discharged from the clinic and returned to full-time employment as a train driver.

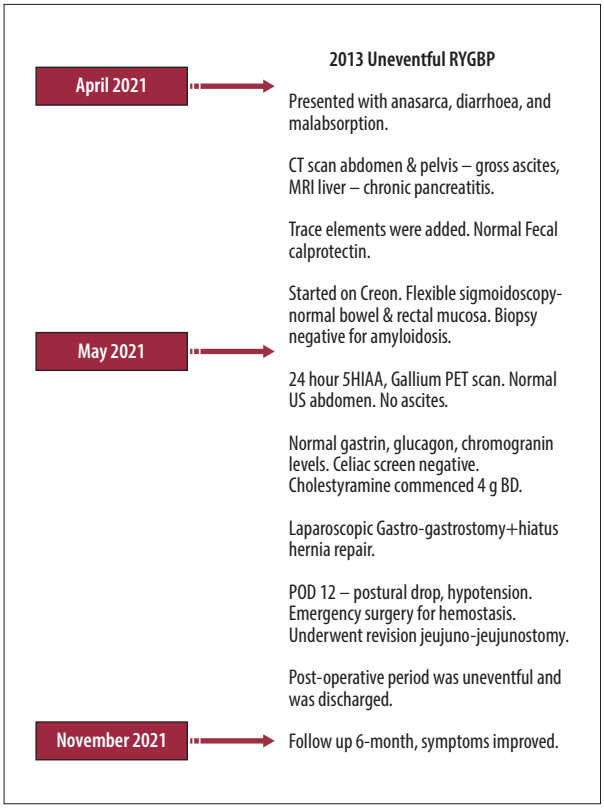

Figure 1. Timeline of investigations and procedures. RYGBP – Roux-en-Y gastric bypass; CT – computed tomography; MRI – magnetic resonant imaging; US – ultrasound; PET – positron emission tomography; POD – post operative day.

He presented after 7 years with unexplained intractable diarrhea, malnourishment, abdominal ascites, and anasarca. He was initially treated in another hospital, with no bariatric input. He was then transferred to our hospital after 8 months from the start of diarrhea. During his presentation, he was severely cachectic and unable to mobilize, requiring a wheelchair, and had a BMI of 17 kg/m^2. He had pressure ulcers on his heels. His blood tests revealed anemia and hypo-albuminemia. His renal functions were normal, but he had trace elements deficiency. His fecal elastase level was zero.

He underwent extensive investigations to establish the cause of his symptoms. Gastroscopy revealed gastric erosions with evidence of previous surgery. His colonoscopy and colonic biopsy results were normal (**Figure 1** explains the timeline of investigations and procedures).

A computed tomography (CT) scan of the chest, abdomen, and pelvis revealed ascites, with bilateral pleural effusion and

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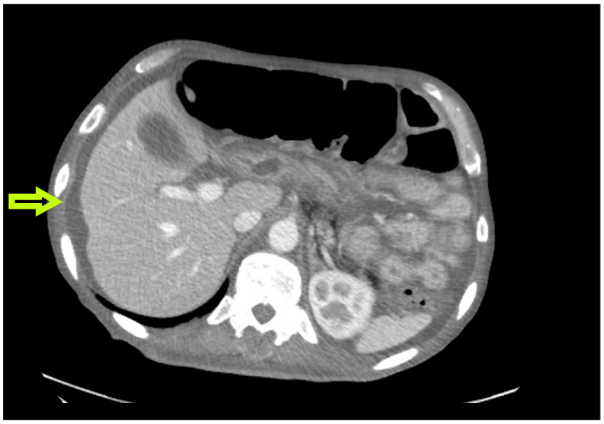

Figure 2. Computed tomography of abdomen and pelvis, showing ascites (yellow arrow).

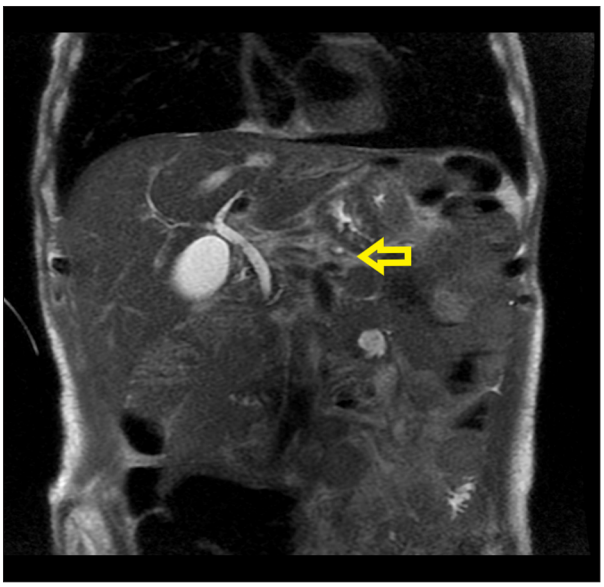

Figure 3. Magnetic resonance image of liver, showing changes suggestive of chronic pancreatitis (yellow arrow).

subcutaneous edema (**Figure 2**). An abdominal magnetic resonance imaging (MRI) scan revealed features consistent with chronic pancreatitis (**Figure 3**).

He was started on total parenteral nutrition for his nutrition replenishment and correction of deficient trace elements. His albumin level improved to 23 g/L with the improvement of his abdominal ascites. Nephrotic syndrome was excluded, and his echocardiogram was normal.

Thorough discussions were carried out with the multi-disciplinary team, the patient, and his family. There was no guarantee that a revision surgery would be successful. He was started on high-dose pancreatic supplements and was treated with rifaximin for clinically suspected bacterial overgrowth. Celiac disease, amyloidosis, inflammatory bowel disease, and malignancy were excluded. His symptoms persisted, and the possibility of home parenteral nutrition was being considered.

We eventually proceeded with the revision of RYGB into normal anatomy in a 2-stage step. The first stage was laparoscopic gastro-gastrostomy with hiatus hernia repair; however, 12 days after surgery, he developed hematemesis due to bleeding from the anastomosis site, which was corrected laparoscopically.

Two weeks later, we proceeded with a jejuno-jejunostomy, and his basic gastrointestinal anatomy was restored to normal with the restoration of the full length of the gastrointestinal tract.

His recovery was satisfactory, his diarrhea improved, and he was discharged home with pancreas enzyme replacement therapy.

At a 6-month follow-up review, his BMI had increased to 33 kg/m^2. His blood parameters were back to normal, his fecal elastase was still low at 26 µg/g. His quality of life had improved and his diarrhea had resolved.

Discussion

PEI is a serious consequence of bariatric surgery. It results in malfunctioning of the pancreas, which can present with nonspecific gastrointestinal symptoms, including malabsorption, steatorrhea, weight loss, abdominal pain, and diarrhea [8]. Owing to the nonspecific clinical presentation, it has been observed that conclusive PEI is not a straightforward diagnosis [9].

Pancreatic societies worldwide have accepted the loss of pancreatic parenchyma as the cause of PEI and acknowledge bariatric surgery as a potential cause, along with pancreatic disorders, diabetes, and inflammatory bowel disease [10].

Over the years, RYGB surgery has become one of the most commonly performed bariatric procedures worldwide. It involves creating a small gastric pouch that is drained into the jejunum with no contact between nutrients and the pylorus and duodenum. The length of the common limb plays an important role in the development of serious implications, like malabsorption and nutrient deficiency [11].

It is believed that after RYGB, the integrity of the functioning of the pancreas is affected, eventually resulting in compromised pancreatic exocrine function [12]. PEI occurs due to decreased ductal function, with a decrease in the concentration of pancreatic enzymes. It is reported that overt symptoms of PEI appear when pancreatic lipase levels fall below <10% of normal levels [13].

There is adequate literature to suggest that after RYGB, gut hormone and peptide levels are affected. Gastrin levels decrease

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after RYGB, resulting in changes such as chronic gastritis and atrophic gastritis [14].

Cholecystokinin is secreted in the duodenal mucosa and promotes the release of pancreatic enzymes, slowing gastric emptying. Peteri et al [15] postulated that postprandial cholecystokinin concentration levels increase after RYGB as compared to the preoperative concentrations.

Some authors have postulated that altered anatomy affects motor and sensory coordination of the gastrointestinal tract and reduces the absorptive surface by diversion [16]. Factors affecting PEI include the length of the biliopancreatic limb and the transit time phase of the intestine [17]. Clinical diagnosis of PEI is a challenging task but should be considered in patients with unremitting symptoms.

Malabsorption is a sequela that happens due to an imbalance between pancreatic enzymes. It is a burden for the patient and has an association with cardiovascular issues, increased infection risks, and increased mortality and morbidity [18].

Checking fecal elastase levels is a noninvasive test that can be used for the assessment of overall pancreatic secretions. Fecal elastase levels below 200 µg/g is suggestive of moderate PEI and levels below 100 µg/g suggest severe dysfunction [19]. In severe cases, the sensitivity of the fecal elastase level is around 82% to 100% [20].

Vujasunovic et al [21] stated that using fecal elastase levels helps in diagnosing pancreatic insufficiency. He also echoed using investigations such as MRI along with blood tests to evaluate zinc, copper, selenium, and vitamin A and D levels.

There is not sufficient literature on PEI after bariatric surgery at present. In 2016, Borbely et al investigated stool samples of around 188 patients after RYGB for fecal elastase; his results showed that 19.7% had fecal elastase <200 ug/g [22].

A previous study showed fecal elastase levels are considerably low in the post-bariatric surgery population as compared to the general population [23]. Patients with malabsorption can develop serious nutritional deficiencies, especially in the fat-soluble vitamins A, D, E, and K [24].

Armbrecht et al [25] studied the effectiveness of pancreatic replacement therapy in treating a patient with PEI and concluded it had a positive effect. Past studies have shown the effect of limb length on the development of PEI [26].

One study showed the benefit of revisional RYBG was an improvement in the quality of life of patients. This showed that 1% of the patients had presented with refractory diarrhea and fluid retention resistance to pancreatic replacement therapy. Revising the length of the limb improved the symptoms that presented after the primary surgery, and hence, improved patient quality of life [27].

This is the third case of PEI after gastric bypass in our 22 years of experience in our unit. This diagnosis should be considered, and we amended our protocol to include fecal elastase in the list of investigations of diarrhea after bariatric surgery.

A similar case was reported in Sweden, where 50-year-old woman had developed unretractable diarrhea and abdominal pain 10 years after laparoscopic gastric bypass for morbid obesity. Her immediate postoperative period was unremarkable. She underwent multiple gastroscopies, diagnostic laparoscopy, and imaging scans (CT, MRI), but nothing was conclusive. She was eventually started on pancreatic enzyme replacement therapy and her symptoms improved gradually [28].

The multi-disciplinary approach in a bariatric unit for treatment in such cases is vital for the well-being of patients. PEI can occur after bariatric surgery. It most commonly presents as a long-term complication and therefore is difficult to diagnose and treat because it is often overlooked. Considering it as a diagnosis and performing adequate investigations helps to treat it in a timely manner.

We still do not know the exact cause of this complication. Many factors have been reported, including humoral factors that suppress pancreatic function, bacterial overgrowth in the biliopancreatic limb that deactivated pancreatic enzymes, and autodigestion of the pancreatic enzymes while en route. These all are theories that we discussed as a team and which may provide ideas for further research.

Conclusions

PEI should be considered in patients presenting with malabsorption and unexplained diarrhea after bariatric surgery. Our case illustrates that a delayed presentation can occur, but timely diagnosis and a multi-disciplinary approach can improve the final outcome. These cases should be managed in a dedicated bariatric unit and future research is required. We report a patient with severe symptoms that responded to revision surgery and pancreatic enzyme replacement therapy.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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