Article published online: 2021-05-11

Recurrent Acute Pancreatitis following Colonoscopic Fecal Microbiota Transplantation for Ulcerative Colitis

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J Health Allied Sci NU 2021;11:207–208.

Abstract

A 37-year-old man with corticosteroid-dependent ulcerative pancolitis was taken up for colonoscopic fecal microbiota transplant (FMT). Preparation for colonoscopy was done with 118 g polyethylene glycol (PEG) in 2 L water ingested over 2 hours, followed by clear fluids. 200 g of screened donor stool, blended with water was instilled into terminal ileum; cecum; and ascending, transverse, and descending colon. Eighteen hours following ingestion of PEG and 2 hours following FMT, he complained of severe epigastric pain with radiation to back. Serum lipase was 6,756 U/L. He was managed with intravenous (IV) fluids and symptomatic treatment with discontinuation of corticosteroids and 6-MP. Ultrasound did not reveal gall bladder stones or sludge. There was no history of alcohol intake. Contrast-enhanced computed tomography scan at 48 hours showed bulky pancreas with peripancreatic stranding. He recovered over a week with normalization of lipase. Three weeks later he again reported severe epigastric pain 14 hours following ingestion of PEG, this time prior to colonoscopic FMT. Serum lipase was 1,140 U/L; the procedure was deferred and he recovered with symptomatic treatment over 3 days. Maintenance colonoscopic FMT was performed 4 times over the following 2 years with sodium phosphate preparation with no recurrence of pain. MRCP showed no evidence of chronic pancreatitis. He remains in clinical and endoscopic steroid-free, thiopurine-free remission. PEG is a rare cause of acute pancreatitis and merits consideration in appropriate clinical setting.

Keywords
- fecal microbiota transplantation
- acute pancreatitis
- polyethylene glycol

Introduction

Several randomized control trials and meta-analysis suggest that patients with ulcerative colitis (UC) may benefit from fecal microbiota transplantation (FMT).[1] We describe a patient who developed recurrent acute pancreatitis (AP) following colonoscopic FMT.

Case Report

A 37-year-old man with corticosteroid-dependent UC for 1 year was taken up for colonoscopic FMT. He was receiving prednisolone 20 mg, 6-mercaptopurine 50 mg, and 5-aminosalicylates 3.6 g daily and his Mayo score was five. Preparation for colonoscopy was done with 118 g

published online
May 11, 2021

DOI https://doi.org/10.1055/s-0041-1726691
ISSN 2582-4287

Thieme Medical and Scientific Publishers Pvt. Ltd. A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

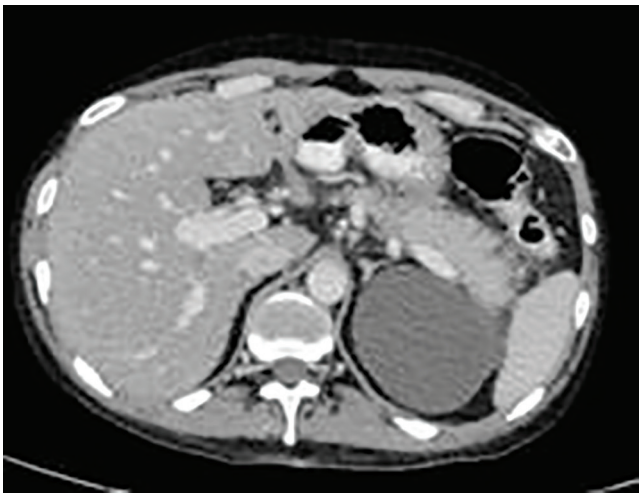

Fig. 1 Contrast-enhanced computed tomography scan showing diffuse pancreatic edema and peripancreatic fat stranding, suggestive of acute pancreatitis. Incidental large left renal cyst is also noted.

polyethylene glycol (PEG) in 2 L water over 2 hours, followed by clear fluids. Eighteen hours following ingestion of PEG and 2 hours following FMT, he complained of severe epigastric pain with radiation to back. There was marked epigastric tenderness. Serum lipase was 6,756 U/L and ultrasound showed normal gallbladder and common bile duct. There was no history of alcohol intake. He was managed with intravenous (IV) fluids, pantoprazole, and tramadol. Corticosteroids and 6-MP were discontinued. Contrast-enhanced CT scan at 48 hours showed bulky pancreas with peripancreatic stranding (►**Fig. 1**). He recovered over 1 week with normalization of lipase. Three weeks later, he again reported severe epigastric pain, 14 hours following ingestion of PEG, this time prior to colonoscopic FMT. Serum lipase was 1,140 U/L. FMT was deferred and he recovered over 3 days with symptomatic treatment. Maintenance colonoscopic FMT was performed four times over the following 2 years with oral sodium phosphate preparation, with no recurrence of pain. MRCP showed no evidence of chronic pancreatitis. He remains in clinical and endoscopic steroid-free, thiopurine-free remission on maintenance FMT protocol.

Discussion

Causes of AP in a setting of UC include gallstones and drugs like corticosteroids and thiopurines.[2] A Danish cohort study of 15,526 patients, showed four times increased risk of AP in Crohn's disease and two times higher risk in UC as compared with general population.[3] There was no history of alcohol intake or metabolic cause of pancreatitis in our patient. Pancreatitis has also reported due to PEG.[4] Postulated mechanisms include stimulation of pancreatic secretions due to gastric distension and reflux of high-pressure duodenal contents into pancreatic duct. Pancreatitis has also been described following colonoscopy, possibly due to trauma to the pancreas while negotiating splenic flexure.[5] There are no reports linking AP to FMT. The temporal relation between use of PEG and onset of AP, symptom cessation with discontinuation, recurrence after re-exposure to PEG, no recurrence of AP during subsequent FMT without use of PEG, and no evidence of chronic pancreatitis at MRCP suggest PEG-induced recurrent AP. We did not test for genetic mutations like cationic trypsinogen (PRSS1), CFTR, SPINK1, and CTRC that have been linked with AP.

Conflict of Interest
None declared.

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