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Single Case

Colitis-Induced Pancreatic Fistula with Pancreatic Ascites

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Keywords
Colitis · Pancreatic fistula · Pancreatic ascites

Abstract
Introduction: Although cases of severe pancreatitis causing fistula formation into the colon have been documented, the reverse process of colitis causing a pancreatic fistula remains undocumented. ***Case Presentation:*** We present the case of a 79-year-old male with severe colitis resulting in perforation and pericolonic abscess formation adjacent to the pancreas, which resulted in an internal pancreatic fistula and pancreatic ascites. After 2 paracenteses, our patient ultimately underwent endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy and pancreatic duct stent placement. The patient clinically improved and was ultimately discharged. ***Conclusion:*** Follow-up ERCP was performed 2 months after discharge and showed no contrast extravasation, illustrating closure of the previous pancreatic fistula. Ultimately, our case demonstrates that cases of severe colitis may contribute to adjacent pancreatic fistula and ascites formation.

Introduction

A pancreatic fistula is defined as an abnormal connection between the pancreatic ductal epithelium and another epithelial surface. This abnormal communication can lead to the leakage of an enzyme-rich pancreatic fluid, resulting in a nonhealing fistula [1, 2]. An internal pancreatic fistula results from disruption of the pancreatic duct, resulting in communication with the peritoneal or pleural cavities. An external pancreatic fistula occurs when the

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disrupted pancreatic duct communicates with the skin, leading to external drainage of pancreatic fluid [3]. Pancreatic fistulas can be iatrogenic or non-iatrogenic. Iatrogenic causes include operative trauma or during biopsy of a pancreatic mass, pancreatic resection, complications from endoscopic interventions, or percutaneous drainage of a pancreatic fluid collection such as in a pancreatic pseudocyst. Non-iatrogenic causes include abdominal trauma, acute pancreatitis, and chronic pancreatitis [4]. Although cases of severe pancreatitis causing fistula formation into the colon have been documented [5], the reverse process of colitis causing a pancreatic fistula remains a rare possibility with limited medical literature describing this process previously. We present the case of a 79-year-old male with severe colitis, resulting in perforation and pericolonic abscess formation adjacent to the pancreas, which we believe resulted in an internal pancreatic fistula and recurrent pancreatic ascites.

Case Presentation

A 79-year-old male with atrial fibrillation, hypertension, and previous cholecystectomy for cholelithiasis presented to the emergency department with acute-onset left flank pain radiating to the left lower quadrant of the abdomen. The patient described the abdominal pain as sharp, and onset was sudden. He denied any epigastric pain or radiation of pain straight to his back. Abdominal exam showed tenderness to palpation in the left lower quadrant without rebound or guarding. Concern for systemic infection was low as patient presented without leukocytosis, fever, hemodynamic instability, or other vital sign abnormalities. Initial CT of the abdomen and pelvis showed nonspecific colitis of the splenic flexure and proximal to mid descending colon with perforation, resulting in a 7.1 × 6 cm multi-loculated fluid collection in the left paracolic gutter. A second loculated pocket of fluid between the spleen and pancreatic tail measuring 3.9 × 2.5 cm was also present. No diverticulosis or diverticulitis was observed on this imaging. However, large volume ascites was noted at this time. The patient was admitted, and paracentesis was performed. 1.5 L of straw-color ascitic fluid was drained and found to have amylase level of 1,365, consistent with pancreatic ascites. One day after admission, a MRI/magnetic resonance cholangiopancreatography (MRCP) was performed which revealed the majority of the pancreatic parenchyma up to the distal tail to be unremarkable and without evidence of acute or chronic pancreatitis (Fig. 1). However, serum lipase was found to be 455 U/L, suggesting pancreatic irritation and subsequent acute pancreatitis. The patient was treated with IV antibiotics and image guided drainage of the left-sided collection was performed 6 days into his hospital stay. At this time, a second paracentesis was done which drained an additional 3 L of fluid. Given the rapid re-accumulation of significant ascites, a pancreatic fistula was suspected and an endoscopic retrograde cholangiopancreatography (ERCP) was performed 12 days into his hospital stay. The ERCP demonstrated a leak from the pancreatic tail (Fig. 2). Given the patient had a rapid re-accumulation of pancreatic ascites and ERCP findings, sphincterotomy with pancreatic duct stent placement was performed to prevent further re-accumulation. The patient was discharged several days later with resolution of symptoms. A follow-up ERCP was performed approximately 2 months after discharge which revealed slight irregularity of the pancreatic duct walls, thought to be stent induced, but no pancreatic duct dilation. No definite features of chronic pancreatitis were seen. No extravasation of contrast was seen during ERCP, indicating healing of the previously seen fistula (Fig. 3). The pancreatic stent was removed, and the patient was asked to return for regular follow-up. A follow-up colonoscopy demonstrated abnormally thickened colonic mucosa at the site of previous colitis approximately 50 cm from the anus. Localized erythema, granularity and congestion with no bleeding were noted and biopsies of this area revealed benign colonic mucosa with focal, acute inflammation, and focal granulation tissue formation.

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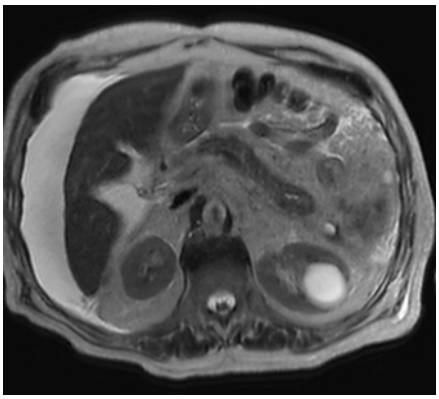

Fig. 1. MRI/MRCP demonstrating absence of pancreatic ductal dilation from any obstruction and normal pancreatic parenchyma at 1 day into hospital stay.

Biopsy findings and endoscopic evaluation of the colon suggested localized infection as the initial cause of the patient's colitis as no signs of further colon inflammation were noted to suggest inflammatory bowel disease, no signs of ischemia were found, and patient had no history of radiation treatments or prior surgical manipulation. No diverticulosis was observed throughout the colon at this time. Unfortunately, after stent removal and follow-up colonoscopy, this patient was lost to follow-up. He is currently not under our care at this time. Upon review of the case and formation of the associated manuscript, the CARE Checklist has been completed and attached as supplementary material (for all online suppl. material, see https://doi.org/10.1159/000543007).

Discussion

In severe pancreatitis, fistula formation to the peritoneum or colon may result from disruption of the pancreatic duct and leakage of digestive enzymes [6]. However, it should be noted that this process may also occur in reverse, with the initial insult coming from severe inflammation of the colon resulting in local inflammation which disrupts the integrity of the pancreatic duct. Research shows documented cases of duodenopancreatic fistula formation in patients with Crohn's disease [7]. However, this is very rare and majority of colopancreatic fistulas have been found in the setting of recurrent acute pancreatitis. Our patient's initial MRCP showed unremarkable pancreatic parenchyma up to the distal tail and no evidence of ductal dilatation or obstructing stone. The patient additionally denied any known pancreatic problems in the past, quit any alcohol consumption 7 years prior, and underwent cholecystectomy years ago making common causes of pancreatitis improbable. These findings argued against a primary pancreatic process, and suspicion was raised for pancreatic irritation due to the adjacent infectious or inflammatory process centered on the colon. We hypothesize that as this adjacent inflammation from severe colitis persisted throughout the patient's stay, erosion of the pancreas wall ensued and created the pancreatic duct leak later found on ERCP.

Conclusion

As seen in our case, patients with new-onset ascites in the setting of colitis should have peritoneal fluid analyzed for amylase and lipase. If high levels of amylase or lipase are found in the peritoneal fluid, further investigation of the pancreatic duct may be obtained with either MRCP or ERCP. Pancreatic fistula is an urgent problem that can result in fatal complications

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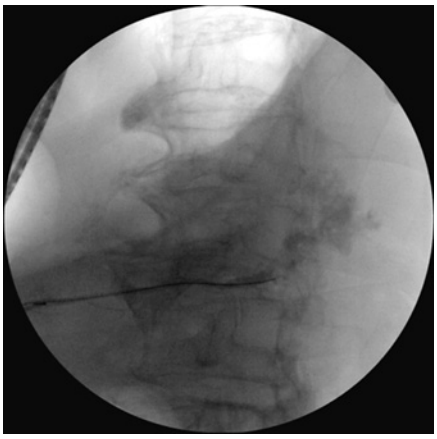

Fig. 2. Initial ERCP performed 12 days into hospital stay showing extravasation of contrast from the pancreatic tail.

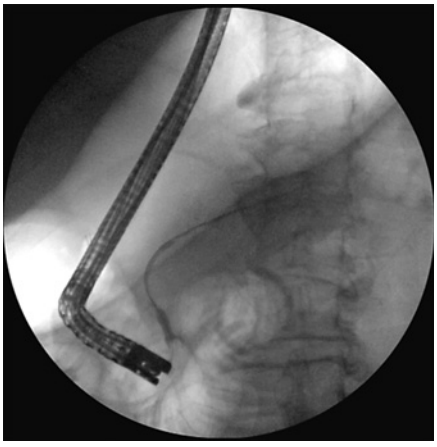

Fig. 3. Follow-up ERCP performed 2 months after hospital discharge with no extravasation of contrast indicating healing of the previously seen fistula.

such as hemorrhage or sepsis and should thus be treated as quickly as possible. Nonsurgical options include pancreatic duct stents, percutaneous drainage, and endoscopic transenteric drainage. Surgical intervention has been suggested in cases of large feculent peritoneal collection, colonic stricture, and severe pancreatico-colonic fistula symptoms [8].

Statement of Ethics

This research has complied with the guidelines for human studies and in accordance with global standards including the World Medical Association Declaration of Helsinki. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. All identifying information has been removed. This retrospective review of patient data did not require IRB ethical approval in accordance with local and institutional guidelines.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Evan Winrich, Ethan Steele, Amal Shine, and Dipendra Parajuli remained involved in conception and design, analysis and interpretation of the data, drafting of the article, and critical revision of the article for important intellectual content. Dipendra Parajuli was the senior investigator and approved the final article submission.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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