Acute pancreatitis associated with duodenal obstruction induced by groove pancreatitis

A case report

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Abstract

Rationale: Groove pancreatitis (GP) is a rare form of chronic pancreatitis. Since GP presents with nonspecific symptoms, it can be challenging to diagnose. Duodenal obstruction is often caused by malignant diseases; however, when associated with acute pancreatitis, it is rarely induced by groove pancreatitis.

Patient’s concerns: A 56-year-old man who presented with acute pancreatitis complained of recurrent upper abdominal discomfort. His concomitant symptoms included abdominal pain, postprandial nausea, and vomiting. Contrast-enhanced computed tomography (CT) of the abdomen showed thickening of the duodenal wall. Gastrointestinal radiographs and upper gastrointestinal endoscopy showed an obstruction of the descending duodenum.

Diagnosis: The pathologic diagnosis was groove pancreatitis.

Interventions: The patient underwent gastrojejunostomy to relieve the obstruction.

Outcomes: The patient had an uneventful recovery with no complications.

Lessons: Groove pancreatitis should be considered in the differential diagnosis of patients presenting with acute pancreatitis and duodenal obstruction. These data can help to make a precise diagnosis and develop an appropriate treatment plan.

Abbreviations: CA = carbohydrate antigen, CEA = carcinoembryonic antigen, CT = computed tomography, EUS = endoscopic ultrasound, EUS-FNA = EUS-guided fine-needle aspiration, GP = groove pancreatitis, MRCP = magnetic resonance cholangiopancreatography, MRI = magnetic resonance imaging.

Keywords: acute pancreatitis, case report, duodenal obstruction, groove pancreatitis

1. Introduction

Groove pancreatitis is an uncommon form of chronic pancreatitis that affects the groove region between the pancreatic head, duodenum, and common bile duct. It often presents in middle-aged males with a history of alcohol abuse. Pathogenesis is still unclear and clinical presentation is not specific. It is a rare disease, and most of the cases tend to be diagnosed after surgery. As a consequence, the diagnosis of GP can be challenging. Imaging features such as sheet-like hypodensity in the pancreaticoduodenal groove, medial duodenal wall thickening, and cystic changes in the duodenal wall are important to suggest the diagnosis of groove pancreatitis. GP can be cured by conservative medical treatment, and surgery is reserved only for patients with persistent and severe clinical symptoms or for a definitive exclusion of malignancy. The differential diagnosis of GP includes duodenal cancer, pancreatic carcinoma, cholangiocarcinoma, or acute pancreatitis with phlegmon along the groove area. In the present case, acute pancreatitis with duodenal obstruction caused by groove pancreatitis could be easily misdiagnosed.

2. Case presentation

A 56-year-old male was admitted to the hospital with acute epigastric abdominal pain. He was diagnosed with “acute pancreatitis,” for which he received conservative treatment in the duration of one week. Three days after his discharge, he presented to our clinic with recurrent sharp abdominal pain accompanied by nausea and vomiting, which worsened following the food intake. He had a history of hypertension. The patient denied tobacco or alcohol use. Family history was not significant.
Physical examination revealed epigastric tenderness. Laboratory tests showed alanine aminotransferase 64 U/L (normal range 9–50 U/L), and gamma-glutamyl transferase 93 U/L (normal range 10–60 U/L). His serum amylase and lipase levels, aspartate aminotransferase, serum carbohydrate antigen (CA) 19-9, and carcinoembryonic antigen (CEA) levels were within normal limits. During his first hospitalization, the CT scan demonstrated enlargement of the pancreatic head, as well as infiltration of peripancreatic fat, which indicated acute pancreatitis (Fig. 1). During his second hospitalization, contrast-enhanced computed tomography showed improved swelling of the pancreatic head, gastric outlet obstruction, and thickening of the duodenal wall (Fig. 2). An upper gastrointestinal endoscopy was performed, revealing a scar formed by a duodenal bulb ulcer. Furthermore, the second duodenal portion was edematous and narrow (Fig. 3), whereas histology was negative for malignancy. Gastrointestinal radiographs showed that the descending duodenum was narrow (Fig. 4). Contrast-enhanced magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) showed thickening and luminal narrowing of the descending part of the duodenum without any obvious mass in the pancreas or duodenum (Fig. 5). The biliary system was normal. The pancreatic duct was not dilated.

Since it is not possible to exclude pancreas or duodenal carcinoma, surgery is used for diagnosis as well as for treatment. Gastrojejunostomy was performed to relieve the obstruction. During the surgery, extensive scarring and widening of the pancreaticoduodenal groove were found, whereas no mass or enlarged lymph nodes were seen in the pancreas or duodenum. The pathology of the groove area was negative for malignancy. The histopathologic examination revealed collagen fiber hyperplasia with Masson trichrome stain (Fig. 6) and myofibroblastic hyperplasia.
proliferation in the groove area (Fig. 7), which was compatible with groove pancreatitis.

Postoperative recovery was uneventful. Our patient recovered well, having no symptoms after 3 years of follow-up.

3. Discussion
Groove pancreatitis was first reported by Becker in 1973. GP is a distinct subset of chronic pancreatitis, which involves the duodenal wall in the vicinity of the minor duodenal papilla, the adjacent pancreatic parenchyma, and the potential space (groove) between them. Becker and Mischke further classified groove pancreatitis into two forms: pure, which involves the groove area only, with preservation of the pancreatic parenchyma and main pancreatic duct and segmental, which involves both the groove and the head of the pancreas with stenosis of the pancreatic duct causing upstream dilatation. These forms accounted for 8.9% and 15.5%, respectively, of 123 pancreaticoduodenectomies performed in patients with chronic pancreatitis. The pathogenesis of groove pancreatitis remains unclear, but anatomical or functional obstruction of the minor papilla is one of the considered issues. Several factors, such as chronic alcohol abuse, smoking, peptic ulcers, gastric resection, true duodenal-wall cysts, and pancreatic heterotopia in the duodenal wall, have been identified as potentially related to this condition. One proposed theory about the etiology of GP is that it is due to either the primary or secondary obstruction of the accessory duct of Santorini and the minor papilla. Indeed, cystic dilatation of the accessory duct (“Santorinicele”) is a
frequent finding at pathology. Heterotopic pancreatic tissue as well as Brunner’s gland hyperplasia that infiltrates the wall of the second duodenum may lead to partial obstruction of the minor papilla as the duct of Santorini. Finally, other inflammatory processes involving the proximal duodenum such as ulcerations from peptic ulcer disease have also been implicated as a cause of GP. Our patient had a duodenal bulb ulcer with no history of alcohol abuse.

The clinical manifestations of groove pancreatitis include upper abdominal pain, weight loss, postprandial vomiting, and nausea due to duodenal stenosis, while jaundice rarely occurs. Serum pancreatic and hepatic enzyme levels are sometimes slightly elevated. Tumor markers, such as CEA and carbohydrate antigen (CA) 19-9, are usually within normal limits.

Thickening and scarring of the duodenal wall close to the minor papilla usually cause stenosis of the second portion of the duodenum. Cystic changes in the thickened duodenal wall are distinctive, and typically contain clear fluid, necrotic and granular material, or stones. Becker and Mischke have found duodenal-wall cysts in 49% of patients with groove pancreatitis. The key histologic criteria include dilated ducts and pseudocystic changes in the duodenal wall; duodenal submucosal fibrosis extending to the adjacent soft tissue in the groove area and pancreas; variable Brunner gland hyperplasia forming a thick layer with surrounding smooth muscle and myofibroblastic proliferation. At histology the most common finding in GP is duodenal Brunner’s gland hyperplasia of the duodenal mucosa which contributes to the thickening of the duodenal wall. Microscopic examination reveals that heterotopic pancreatic tissue occurs in both the submucosa or muscularis propria of the duodenal wall.

Characteristic imaging findings on CT include reactive parietal thickening along the medial aspect of the descending duodenum. The thickened duodenal wall often shows prominent enhancement and, depending upon the degree of duodenal thickening/scarring and consequential luminal compromise, upstream gastric dilatation may be found. Additional findings include cystic-like lesions in the duodenal wall or in the groove area. A sheet-like mass between the head of the pancreas and the duodenum associated with duodenal wall thickening is the most characteristic finding on magnetic resonance imaging. When the duodenum of patients with groove pancreatitis is too narrow to perform gastrointestinal fibroscopy, magnetic resonance cholangiopancreatography is a useful diagnostic option.

Endoscopic ultrasound (EUS) is also effective in diagnosing groove pancreatitis. Yet, EUS makes insertion impossible due to duodenal stenosis, and the accuracy of EUS is dependent on operator and experience. The potentialities of the EUS are multiple, as it can also detect thickening and stenosis of the second duodenum part with intramural cysts, smooth stenosis of the common bile duct; and in the segmental form, heterogeneous hypoechoic mass, enlargement of the pancreatic head, with calcifications or pseudocyst and dilatation of the main pancreatic duct. EUS-guided fine-needle aspiration is a valuable approach for excluding pancreatic cancer. If the diagnosis of GP is clear, patients may be conservatively, endoscopically, or surgically treated. Conservative treatment options include stop smoking/alcohol consumption, recovery of pancreatic function, and analgesics. Yet, such treatments tend to be only temporarily effective. Relapsing episodes of pancreatitis may not be prevented in cases with an anatomic disturbance of pancreatic juice outflow.
Balakrishnan V, Chattin S, Radhakrishnan L, et al. Groove pancreatitis: a case report and review of literature. JOP 2007;8:592–7.

Becker V. [Proceedings: Fundamental morphological aspects of acute and chronic pancreatitis (author’s transl)]. Langenbecks Arch Chir 1973;334:317–22.

Arora A, Dev A, Mukund A, et al. Paraduodenal pancreatitis. Clin Radiol 2014;69:299–306.

Becker V, Mischke U. Groove pancreatitis. Int J Pancreatol 1991;10:173–82.

Stolte M, Weiss W, Volkholz H, et al. A special form of segmental pancreatitis: “groove pancreatitis”. Hepatogastroenterology 1982;29:198–208.

Arora A, Rajesh S, Mukund A, et al. Clinicoradiological appraisal of ‘paraduodenal pancreatitis’: pancreatitis outside the pancreas!. Indian J Radiol Imaging 2015;25:303–14.

Muraki T, Kim GE, Reid MD, et al. Paraduodenal pancreatitis: imaging and pathologic correlation of 47 cases elucidates distinct subtypes and the factors involved in its etiopathogenesis. Am J Surg Pathol 2017;41:1347–63.

Oza VM, Skeans JM, Muscarella P, et al. Groove pancreatitis, a masquerading yet distinct clinicopathological entity: analysis of risk factors and differentiation. Pancreas 2015;44:901–8.

Tezuka K, Makino T, Hirai I, et al. Groove pancreatitis. Dig Surg 2010;27:149–52.

DeSouza K, Nodit L. Groove pancreatitis: a brief review of a diagnostic challenge. Arch Pathol Lab Med 2015;139:417–21.

Triantopoulou C, Dervenis C, Giannakou N, et al. Groove pancreatitis: a diagnostic challenge. Eur Radiol 2009;19:1736–43.

Addeo G, Beccani D, Cozzi D, et al. Groove pancreatitis: a challenging imaging diagnosis. Gland Surg 2019;8(suppl 3):S178–87.

Levenick JM, Gordon SR, Sutton JE, et al. A comprehensive, case-based review of groove pancreatitis. Pancreas 2009;38:e169–175.

Toshihide A, Hidetiko Y, Satoshi I. A case of groove pancreatitis resisted conservative therapy and demanded a pylorus preserving pancreateoduodenectomy. J Surg 2001;46:35–9.

Laugier R, Grandval P. Does paraduodenal pancreatitis systematically need surgery? Endoscopy 2014;46:388–90.

Wang J, Barbuskaite D, Tozzi M, et al. Proton pump inhibitors inhibit pancreatic secretion: role of gastric and non-gastric H+/K+-ATPases. PLoS One 2015;10:e0126432.

Latham J, Sanjay P, Watt DG, et al. Groove pancreatitis: a case series and review of the literature. Scott Med J 2013;58:28–31.

Winter JM, Cameron JL, Campbell KA, et al. 1423 pancreaticoduodenectomies for pancreatic cancer: a single-institution experience. J Gastrointest Surg 2006;10:1199–210. discussion 1210-1191.

Lekkerkerker SJ, Nio CY, Issa Y, et al. Clinical outcomes and prevalence of cancer in patients with possible groove pancreatitis. J Gastroenterol Hepatol 2016;31:1895–900.

Egorov VI, Vankovich AN, Petrov RV, et al. Pancreas-preserving approach to “paraduodenal pancreatitis” treatment: why, when, and how? Experience of treatment of 62 patients with duodenal dystrophy. Biomed Res Int 2014;2014:185265.