Journal of Pancreas

A Challenging Case of Groove Pancreatitis

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Abstract

Groove pancreatitis (GP) is a rare type of segmental chronic pancreatitis that affects the anatomical area between the pancreatic head, the duodenum, and the common bile duct, referred to as the groove area. Most patients with groove pancreatitis are males aged 40–50 years with a history of alcohol abuse. The prevalence of this condition was not determined due to rarity of cases.The clinical symptoms commonly reported were weight loss, upper abdominal pain, postprandial vomiting, and nausea due to duodenal stenosis. It is considered difficult clinically and radiographically to distinguish this form of chronic pancreatitis from other differential diagnoses of inflammatory conditions or malignancies affecting the pancreas or adjacent structures to that groove area. We report a challenging case of GP in an asymptomatic female patient during routine gastric ulcer screening. Our objective is to draw attention to this rare and atypical type of chronic pancreatitis and the importance for early detection on screening EGDs prior to its symptomatic sequelae and complications.

Case Report

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Peer-Reviewed Article

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Keywords

groove pancreatitis, pancreatic adenocarcinoma, pancreatic cancer, whipple, endoscopic ultrasound, pancreatitis

Received: May 05, 2023

Accepted: May 22, 2023

Published: September 02, 2023

Academic Editor:

Talha Bin Emran, Assistant Professor, Department of Pharmacy, BGC Trust University Bangladesh "BGC Biddyanagar", Kanchannagar-4381, Chandanaish, Chittagong, Bangladesh.

Citation:

Daniel Stenberg, Midhat, ML, Lakhani (2023) A Challenging Case of Groove Pancreatitis. Journal of Pancreas - 1(1):1-5.

Introduction

First introduced by *Becker et al.*[1] in 1973 in the German literature, groove pancreatitis (GP) is reported as a distinct form of segmental chronic pancreatitis loculated in the area between the head of the pancreas, the duodenum and the common bile duct. Compared to the non-segmental form of chronic pancreatitis, it was reported that there were no differences observed in age or sex distribution or alcohol consumption. *Stolte et al.*[2] in 1982, described GP as a chronic inflammatory process characterized by fibrous scars in the anatomical Groove area. In the early 1990s, *Becker and Mischke.*[3] classified GP into “pure” and “segmental” forms based on whether the main pancreatic duct was involved or not, and the location of the scarring. In 2009, *Triantopoulou C et al.*[4] further outlined this disease stating that it is manifested on imaging by a sheet-like mass in the groove area near the minor papilla. Other features described were thickening and cystic changes of the duodenal wall.

Due to its rarity, the incidence of GP is unknown, but it was found in about 19.5–24.4% of pancreaticoduodenectomies performed to treat chronic pancreatitis.[2,3] Clinicians ought to know or familiarize themselves with this entity due to the fact that it's very hard to distinguish it from pancreatic head adenocarcinoma radiographically and clinically, which makes a difference in management. Conservative medical measures are usually pursued for GP, while surgery is usual-

ly preserved for severe symptoms or to rule out malignancy. Pancreaticoduodenectomy using the whipple procedure or a pylorus-preserving pancreaticoduodenectomy are usually the surgical options of choice. In this article, we report a challenging case of GP in a female patient diagnosed during routine gastric ulcer screening and who was managed conservatively.

Case presentation

This is a 73-year-old woman with a past medical history significant for alcohol use and hypertension who presented for elective EGD that was scheduled as surveillance for a gastric ulcer. The patient reported no new symptoms. EGD showed a large circumferential mass-like effect with mucosal changes of the first and second parts of the duodenum, with the finding of extrinsic compression of the antrum from a possible pancreatic source. The area was friable but no active bleeding was seen. Biopsies were taken with cold forceps for histology. Liver enzymes, bilirubin, and routine blood work were unremarkable. CT scan with contrast showed marked thickening of the duodenal wall with possible underlying mass. Additionally, there were severe appearing, chronic changes in the pancreatic groove with multiple cysts of the pancreatic head and duodenal wall.

An endoscopic ultrasound was performed which showed wall thickening at the duodenum apex. There was a cystic lesion beyond the mass within the duodenal wall measuring 10 mm in length. Duodenal biopsies showed findings consistent with subacute and chronic inflammation with hyperplastic surface epithelial change with features of peptic injury and focal adenomatous epithelial change, without evidence of malignant changes. An abdominal MRI was performed 6 weeks later, which showed improved inflammatory changes in the head of the pancreas, with a persistent cyst of the pancreatic head and smaller cysts in the head and uncinate process. There was near-complete resolution of the inflammatory duodenal thickening. A repeat EGD with biopsy showed no changes from prior endoscopic exams.

Discussion

Groove pancreatitis, is a rare and atypical form of segmental chronic pancreatitis first reported in 1973 by Becker in the German literature as ‘‘segmentäre pankreatitis’’ or ‘‘Rinnenpankreatitis’’.[1] In 1982, *Stolte et al.*[2] collected specimens from 123 patients who underwent surgical duodenopancreatectomy indicated for chronic pancreatitis and this form of “special” pancreatitis was found in 24.4% of cases. Duodenal stenosis was the most prominent clinical feature in the diagnosed group. In the early 1990s, *Becker and Mischke.*[3] further studied this rare form of pancreatitis, classifying it as “pure form”, which involved only the groove area, sparing the main pancreatic duct. While the “segmental form” represents the involvement of the head of the pancreas in addition to stenosis of the main pancreatic duct causing upstream dilatation.

In late 1994, *Itoh, S et al.*[4] retrospectively reviewed the Computed Tomography (CT) findings in four patients diagnosed with GP. The study included images from plain, dynamic, and high dose enhancement CT scans which were compared with histologic features examined from specimens of those patients. Cysts in the duodenal wall, and/or the groove, duodenal wall thickening, and stenosis were reported in all four patients.

In 2004, *Adsay and Zamboni* [5] suggested that this small potential space between the head of the pancreas and the 2nd part of the duodenum may have an anatomic variation of the ductal system making it vulnerable to alcoholic injury. They also suggested that the myo-adenomatoid and cystic changes involving the duodenal wall may be due to localized recurrent pancreatitis. They referred to those clinicopathological findings as Paraduodenal Pancreatitis.

In 2011, *Manzelli et al.*[6] discussed a review of literature and a miniseries of 5 cases of GP that were treated surgically in one center over a 4 year period. Certain key pathological criteria for the diagnosis of GP resulted from this review and include:1) Dilated ducts and pseudocystic changes in the duodenal wall; 2)Brunners's gland hyperplasia; 3) Fibrosis in the adjacent pancreas and soft tissue especially in the groove area. Those 5 patients reported ongoing alcohol consumption and were active tobacco users. A common feature noted on their imaging was duodenal wall thickening and cystic changes, similar to those in our patient.

Current etiologies of GP involve multiple factors that are considered a key in the disease process. For example, abuse of ethyl alcohol and smoking were found in most cases reported in literature for GP. It is believed that tissue injury resulting from both alcohol and smoking lead to pancreatic duct plug formation and eventually calcification in the long run. This will, in turn, result in obstruction of pancreatic enzymes and create an inflammatory response in the groove area which is the most susceptible area to this injury. Clinically, patients with GP are predominantly males (40-50 years) presenting with symptoms like severe epigastric pain, postprandial nausea and vomiting due to pressure effect on the duodenal wall, and weight loss. Those symptoms might be acute or chronic depending on the degree of tissue injury. The significance of our report is that our patient was female and did not have any symptoms at the time of screening.

Work up usually involves a detailed medical history and physical examination to rule out other causes of acute epigastric pain. As previously mentioned, GP is difficult to diagnose and often hidden under a constellation of symptoms. As such, multiple imaging modalities are utilized by the time the diagnosis is made. An abdominal ultrasound will often show a hypoechoic mass and a CT scan will reveal hypodense, poorly enhancing mass between the pancreatic head and a thickened duodenal wall. [3] MRI also will show a hypointense mass on T1-weighted imaging and on T2-weighted it shows an iso- or slightly hyperintense image. There will be delayed contrast enhancement owing to the reflection of GPs fibrous nature.[11] EUS and ERCP reveal smooth tubular stenosis of the CBD without abnormality of the main pancreatic duct, or rarely, with only slight irregularities. [12] Our patient received an EGD, CT and MRI demonstrating findings consistent with the diagnosis of GP.

The differential diagnosis includes a variety of pathologies pertaining to the duodenum, CBD and head of pancreas, such as duodenal cancer, cholangiocarcinoma or acute pancreatitis. In 1992, *Yamaguchi et al* [13] studied eight, male, Japanese patients presented with abdominal pain, with a mean age of 58 years. Radiologically, those patients were found to have duodenal strictures and biliary stenosis while some even had a mass in the pancreatic head. They all underwent a pancreatoduodenectomy for suspected pancreatic carcinoma. However, histopathological analysis later showed signs of chronic pancreatitis involving the groove between the distal common bile duct, duodenum and pancreas. This review emphasized the importance of keeping this condition in mind when suspecting pancreatic head carcinoma to avoid unwarranted surgical interventions.

Notably, however, unlike groove pancreatitis, most pancreatic adenocarcinomas do not show internal cystic changes and are much more likely to infiltrate posteriorly into the retroperitoneum and encase the vasculature (including the gastroduodenal artery). Moreover, thickening of the medial duodenal wall, a common finding with groove pancreatitis, is quite uncommon with pancreatic adenocarcinoma. Finally, some researchers have suggested that the enhancement pattern for groove pancreatitis tends to be more patchy and heterogeneous compared with pancreatic adenocarcinoma, which is usually more

homogeneously hypodense.[7] Biopsy through the duodenum is also useful for diagnosis.

Given the condition's rarity, there exists a paucity of data regarding treatment. In one retrospective chart review at an academic tertiary medical center from 2008-2017, 24 patients were identified and followed. All had MRIs performed and approximately half, 54.1%, had chronic pancreatitis. The other most common symptoms were biliary dilatation, 25%, and pancreatic duct dilatation, 20.8%. At the outset, 7 patients had ERCP, 11 opted for surgical resection, and the remaining 6 had medical management with anti-emetic therapy. None of the ERCP cohort experienced resolution while 63.6% of initial surgical patients experienced resolution. Medical management had only a 16.6% success rate. This study concluded that surgical intervention had a higher success rate but also was associated with higher morbidity in the form of peri-operative infections. [10]

Pancreatoduodenectomy using the Whipple procedure or a pylorus-preserving pancreatoduodenectomy is a rational treatment for symptomatic groove pancreatitis and is often required due to the severity of clinical symptoms as well as to rule out pancreatic head carcinoma. On gross pathology the specimen confirms the pathology in showing an abundance of a whitish firm mass in the groove area stenosing the terminal CBD. [1,2,13]

Conclusion

Groove pancreatitis is truly a unique entity with an unknown prevalence. As imaging and other clinical investigatory tools continue to improve, the incidence of GP appears to rising. Heralded by a distinct constellation of pathologic findings, it is crucial to screen symptomatic or at risk populations. Management can vary wildly and as Bender et al discovered, medical management was successful 16.6% of the time. Providers need to be cognizant of this disease in symptomatic and asymptomatic patients, as in our case, since appropriate screening can lead to potentially life-altering interventions.

Acknowledgments

The authors have no personal acknowledgments to state, and received no assistance in preparing the manuscript.

Funding

The authors have no funding to disclose behind this manuscript.

Data Availability

Data sharing is not applicable to this article as no datasets were generated or analyzed

during the current report.

Potential Conflicts of Interest

Daniel Stenberg: Has no conflicts of interest to declare

Midhat Lakhani: Has no conflicts of interest to declare

Patient Consent Statement

Patient consent was obtained and is available at the request of the publisher. The article has had all patient identifiers removed.

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