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SPECIAL SECTION: PANCREATITIS

Groove pancreatitis: a clinical and imaging overview

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Abstract
Groove or paraduodenal pancreatitis is an uncommon fibroinflammatory form of pancreatitis involving the anatomic space of the pancreatic groove located between the C-loop of the duodenum and the head of the pancreas. Although in some patients there are distinctive clinical and imaging features of groove pancreatitis (GP), there is often significant overlap with other infiltrative processes involving the pancreatic groove such as pancreatic ductal adenocarcinoma or duodenal carcinoma. In this review, we summarize the most distinctive clinical and imaging aspects of GP and highlight some important distinguishing features that may aid in differentiating malignant lesions involving the pancreatic groove.

Keywords Pancreatitis · Groove · Carcinoma · Pancreas

Introduction

First described in the 1970s, the term groove pancreatitis refers to a localized form of chronic pancreatitis occurring in the pancreaticoduodenal space between the medial wall of the first and second portions of the duodenum and the head of the pancreas [1–9]. In some patients, there may also be associated involvement of the adjacent head of the pancreas. Multiple terms have been used to describe this rare chronic form of pancreatitis reflecting a complex pathophysiologic process involving inflammatory exudate, cyst formation, and deposition of fibrotic tissue. GP has also been referred to as paraduodenal pancreatitis, cystic dystrophy of heterotopic pancreas, myoadenomatosis, periampullary duodenal wall cyst, and pancreatic hamartoma of the duodenum [3, 4, 9].

On histology, the pathologic findings with GP may be quite variable and include dense collagenous fibrosis, cystic areas devoid of epithelium, Brunner's gland hyperplasia, and both smooth muscle hyperplasia and myoadenomatosis of the duodenal wall [6]. Not infrequently these fibroinflammatory changes are centered around the minor papilla and the opening of the duct of Santorini. Proliferation of fibrotic tissue may lead to duodenal encasement and stenosis with resultant gastric outlet obstruction. In addition, biliary obstruction may occur when there is extension to the common bile duct. When the pancreas is involved, there may be changes in chronic calcific pancreatitis [4, 8].

Pathophysiology

The precise etiology of GP is poorly understood and is likely to be multifactorial involving both structural and anatomic factors [10]. In addition, physiologic mechanisms leading to localized stasis of pancreatic duct secretion are likely to be contributing aspects as well. A final common pathway for the development of GP appears to be related to the pancreatic ductal obstruction with extravasation of activated proteolytic enzymes triggering pancreatitis and a cascade of chronic inflammation and fibrosis [10].

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

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frequent finding at pathology [10]. 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. This may be accentuated by the fact that, unlike the common bile duct with the sphincter of Oddi, there is no true sphincter mechanism for the duct of Santorini, thus rendering it more susceptible to partial obstruction. Finally, other inflammatory processes involving the proximal duodenum such as ulcerations from peptic ulcer disease have also been implicated as a cause of GP [7].

There is a strong association between GP and both alcohol and tobacco abuse [1, 3–6, 8, 9]. Chronic ethanol abuse has been shown to not only decrease the volume of pancreatic fluid but also increase in the viscosity of pancreatic secretions resulting in potential luminal obstruction of the pancreatic ducts. In addition, ethanol decreases the concentration of citrate in pancreatic juice which is a causal factor in the precipitation of crystals in pancreatic ductal secretions [1]. These crystals may then become a nidus for ductal stone formation. Brunner's gland hyperplasia has also been associated with alcohol ingestion, and when this involves the second duodenum and the area of the minor papilla, it may lead to obstruction of the accessory duct of Santorini. Finally, another mechanism for the development of pancreatic ductal stone formation is that ethanol causes protein plugs within the pancreatic ducts which can also predispose to stone formation [1].

Of note is the fact that to date there is no known association between the development of GP and either gallstone or autoimmune pancreatitis [1, 5, 6, 9].

Clinical features

Patients with GP are typically male patients in the 4th or 5th decade of life and therefore, are younger than the average patient with PDAC which peaks in the 6th decade [2–9]. In the majority of patients, there is a clear history of chronic alcohol and/or nicotine abuse and up to 50% of patients may have history of prior episodes of acute pancreatitis [11]. GP is uncommon in women and in patients under 40 years of age [1, 3–9]. The clinical presentation may mimic acute pancreatitis in other forms with a constellation of findings including severe abdominal pain, nausea, and vomiting. Not uncommonly, however, the clinical presentation has more chronic and relapsing course with repeated bouts of abdominal pain and weight loss at times mimicking PDAC. Vomiting may be a prominent feature in patients who develop duodenal stenosis and gastric outlet obstruction. In patients with involvement of the common duct and post-inflammatory strictures, jaundice may be present.

Laboratory values and biochemical markers are often nonspecific [1, 3–9]. Typically, there is slight elevation of pancreatic enzymes (lipase and amylase) as well as liver function tests with mild elevation of glutamyltransferase and alkaline phosphatase reflecting cholestasis. In addition, the bilirubin may be elevated in patients with common duct strictures. Of note is the fact that unlike patients with PDAC, tumor markers such as carbohydrate antigen (CA-19-9) and carcinoembryonic antigen (CEA) are not significantly elevated in GP [3, 4, 6, 8, 9]. Elevation of these tumor markers may be a useful finding indicating PDAC involving the pancreatic groove that mimics GP. [4–8].

Imaging features of groove pancreatitis

Contrast-enhanced CT and MRI are the primary imaging modalities used to suggest the diagnosis of GP [6, 9, 10, 12]. Both modalities are very useful to anatomically localize the fibroinflammatory process of GP to the pancreaticoduodenal space. Most patients present clinically with signs and symptoms of pancreatitis, and thus the initial study may only be a screening CT study performed during the portal venous phase. Improved imaging of the pancreas may be possible with a dedicated pancreatic protocol CT using dual energy to increase lesion conspicuity [13, 14]. This involves performing late arterial phase ("pancreatic phase") imaging after a rapid intravenous bolus of contrast. Pancreatic phase imaging may be very useful to more confidently identify a small pancreatic mass. Delayed images are often quite helpful to demonstrate retention of contrast within fibrotic tissue. One advantage of CT is its ability to detect small ductal calcifications that are characteristic of alcohol-related chronic calcific pancreatitis. In addition to routine axial and coronal images, curved planar reformations, volume-rendered imagines, and minimum intensity images are useful post-processing adjuncts to standard CT.

Due to its superior contrast resolution with T2 imaging, MRI is very useful to demonstrate the small non-epithelial duodenal or paraduodenal cysts that often occur with GP. MRCP may be useful to characterize the long smooth tapering of the common bile duct typical of GP. Endoscopic ultrasound (EUS) may also be a very valuable adjunct to CT and MR. It may not only provide high-resolution images of the head of the pancreas and pancreaticoduodenal groove, but may also be critical to obtain tissue sampling with fine needle aspiration in patients suspected of having malignant infiltration of the pancreatic groove. Finally, a recent report suggests FDG–PET–CT may potentially be useful as a diagnostic tool by demonstrating multiple areas of FDG in the paraduodenal tissues as opposed to a single confluent mass in the head of the pancreas as with PDAC [15].

One of the challenges in establishing a firm diagnosis of GP is the variability of the imaging findings. Apart from the one constant feature of a fibroinflammatory process involving the pancreaticoduodenal groove, the extent of fibrotic change, cyst formation, and duodenal or pancreatic involvement are all quite variable [6, 9, 10, 12]. A series by Zaheer et al. [16] showed involvement of the pancreatic head, pancreaticoduodenal groove and duodenum in 75% of patients with GP. Indeed, some authors have attempted to subtype various pathologic forms of GP based on (1) the location of the pathology with the pure form (groove-predominant) being less common than the segmental form (groove plus pancreatic involvement) (2) the type of pathology (cyst-forming versus solid and fibrotic) and (3) the "ill-defined" nature of the inflammatory process that contains neither fibrotic tissue or discrete cyst formation, but may result in poor enhancement of the pancreatic parenchyma with dilation of the main pancreatic duct [2, 10]. Acute interstitial edematous pancreatitis can on occasion be associated with acute pancreatic fluid collections and inflammatory exudate that extend into the pancreaticoduodenal groove (Fig. 1) [8]. Unlike true GP, however, these patients demonstrate multicompartmental fluid collections within the lesser sac and/or retroperitoneal spaces such as the anterior pararenal, perirenal, and interfascial spaces. Equally important is that these acute fluid collections secondarily involving the pancreaticoduodenal groove typically resolve within a few weeks and are not chronic findings as seen in patients with true GP [8].

The "sheet-like" mass of fibrotic tissue associated with GP often demonstrates a crescentic configuration on coronal views conforming to the pancreaticoduodenal groove [12]. With multiphasic contrast images, this fibrotic tissue characteristically shows delayed contrast enhancement (Fig. 2) [6, 9, 10, 12]. Intrinsic involvement of the duodenum is variable and may demonstrate mural thickening of the medial wall, intramural cyst formation, or fibrotic stenosis causing gastric outlet syndrome (Figs. 3, 4, 5) secondary to Brunner's gland hyperplasia, edema, spindle cell proliferation, and duodenal wall cysts on histology. Similarly, the common bile duct may become involved in the fibroinflammatory process associated with GP producing biliary strictures and jaundice (Fig. 2). On imaging the biliary strictures from GP are typically long smooth areas of gradual tapering compared to the abrupt, focal strictures associated with PDAC. When GP involves the pancreas, there may be dilation of the main pancreatic duct as well as the accessory duct of Santorini. Ductal obstruction may occur not only as the result of fibrotic strictures but also intraductal calculi.

Finally, vascular structures within or bordering the C-loop such as the gastroduodenal artery and its continuation as the anterior superior pancreaticoduodenal artery may become entrapped by the fibroinflammatory tissue associated with GP. Thus, "vascular encasement" is not useful as a key differentiating feature to distinguish GP from PDAC [9].

Differentiating GP from PDAC

In selected patients, a combination of clinical, laboratory, and imaging findings may strongly suggest the diagnosis of GP. It should be emphasized however that compared to PDAC, GP is relatively rare and the clinical and radiologic literature characterizing this entity is largely composed of small, single institution, retrospective studies. The true

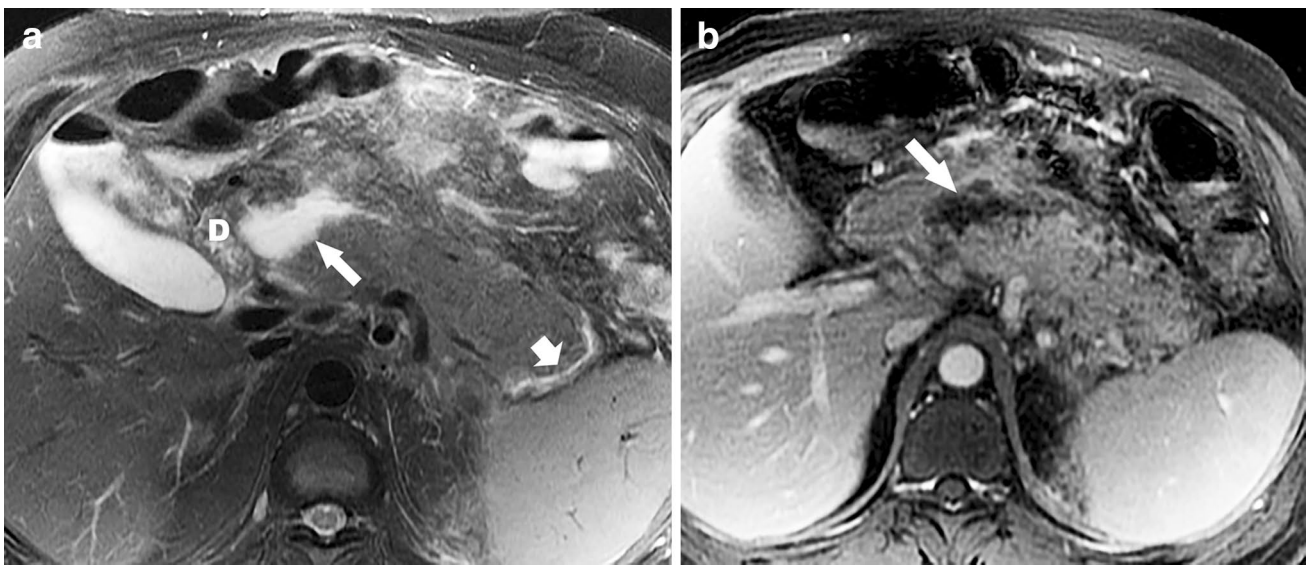

Fig. 1 Sixty-four-year-old female with acute edematous pancreatitis. Axial T2-weighted MRI shows acute fluid collection in the pancreaticoduodenal groove (long arrow) as well as acute fluid collection in the anterior pararenal space adjacent to the tail of the pancreas (short arrow)

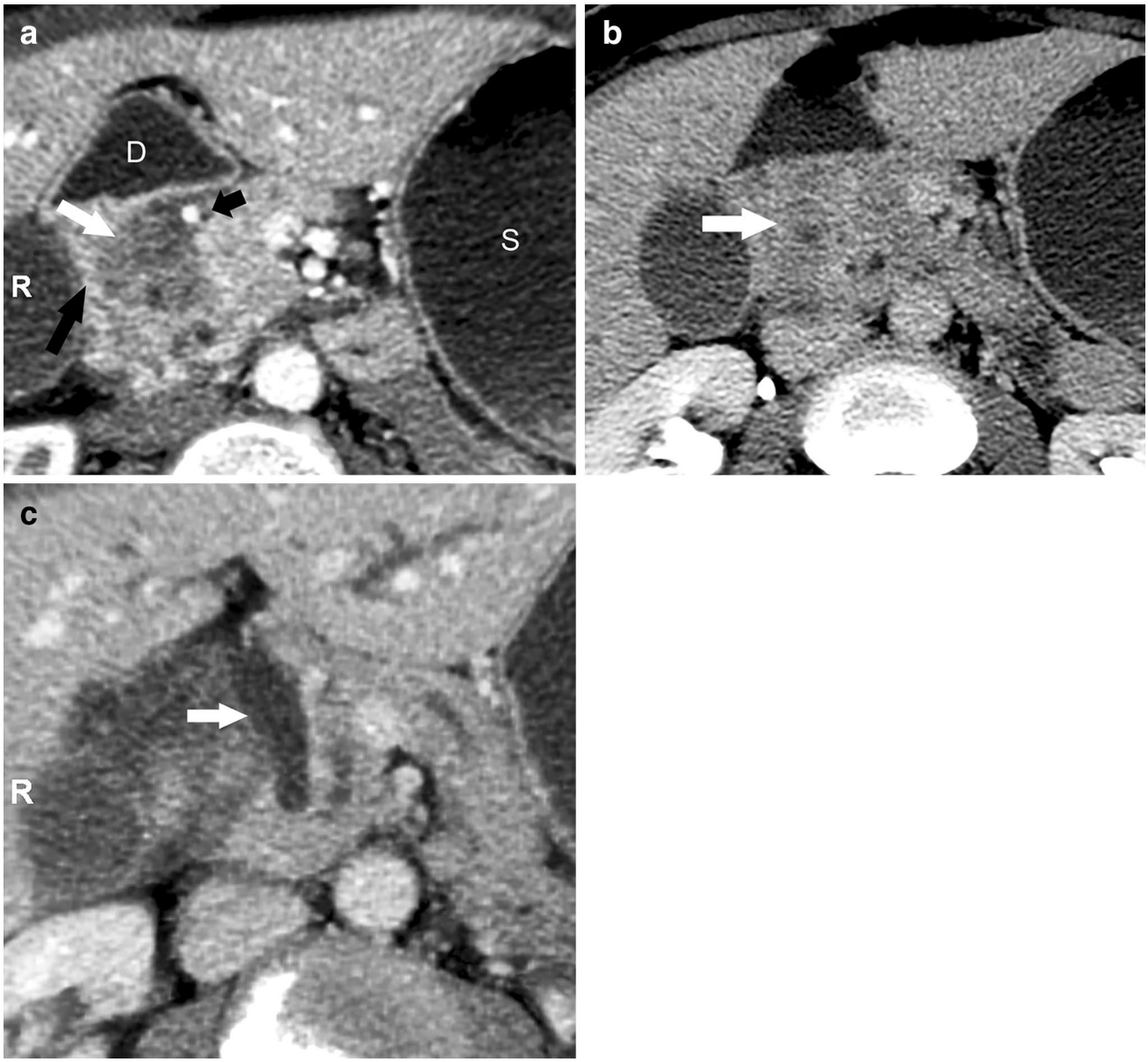

Fig. 2 Fifty-one-year-old alcoholic male with groove pancreatitis and fibrotic tissue in the pancreaticoduodenal groove showing delayed retention of contrast. **a** Axial CT in the portal venous phase demonstrates low attenuation tissue within pancreaticoduodenal groove (white arrow) that is seen to encase the gastroduodenal artery (short black arrow). D = duodenum. S = stomach. Note the marked compression of the duodenal lumen by the fibroinflammatory mass in the pancreaticoduodenal groove (long black arrow). **b** Axial three-minute delay scan shows delayed retention of contrast within the fibrotic tissue. Fibrotic stenosis of the duodenum resulted in gastric outlet obstruction necessitating surgery. **c** Note the long smooth tapering of the common bile duct from a biliary stricture (arrow)

incidence of GP is unknown and it has been reported to be only 2% of pancreatic resections for chronic pancreatitis [17]. Unlike patients with PDAC, the majority of patients with GP are treated conservatively with bowel rest, analgesics, pancreatic enzymes, and cessation of alcohol and tobacco. The clinical consequences of misdiagnosis of PDAC are quite profound and due to the frequent overlap of the clinical, laboratory, and imaging findings a healthy skepticism of the diagnosis must always be considered. When positive for PDAC, tissue sampling with EUS may be definitive, but when negative one must always consider the possibility of a sampling error. The most classic presentation of GP involves a male in his 40s or 50s with a long history of alcohol and tobacco abuse developing severe abdominal pain and vomiting. Laboratory values demonstrate mild elevation of pancreatic and liver enzymes, but tumor markers are notably within normal limits [3, 4, 6–9]. There has been an overall decrease in the number of pancreaticoduodenectomies

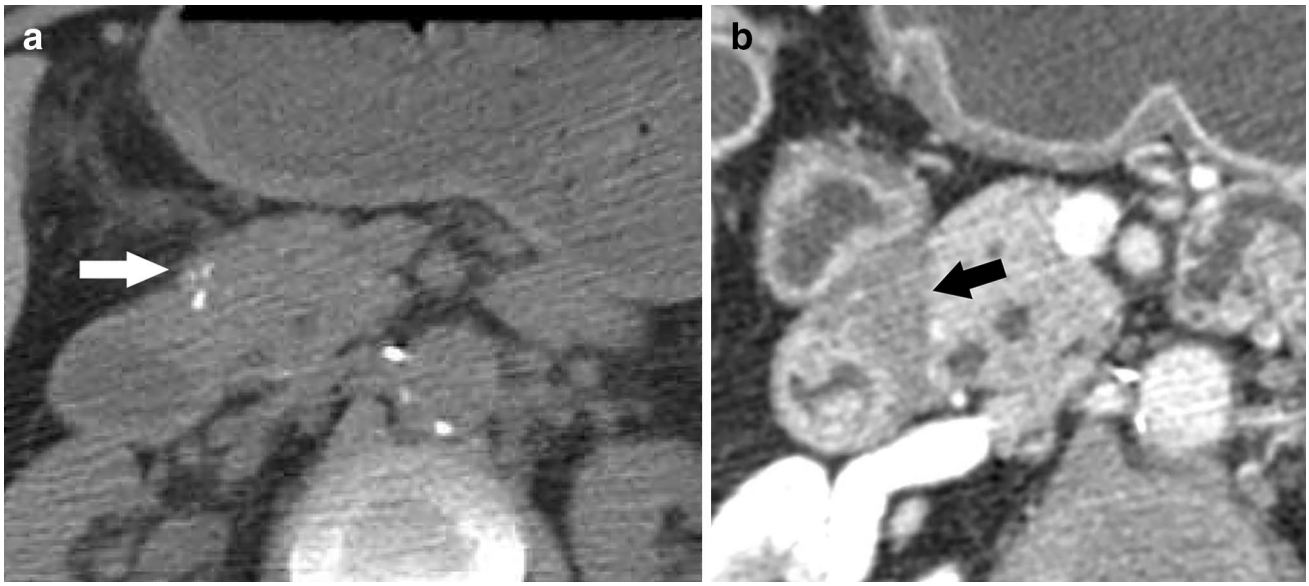

Fig. 3 Forty-nine-year-old alcoholic male with chronic calcific pancreatitis and groove pancreatitis. **a** is a non-contrast axial CT showing pancreatic calcifications (arrow). **b** is a contrast-enhanced axial CT demonstrating enhancing soft tissue in the pancreaticoduodenal groove from (arrow) groove pancreatitis

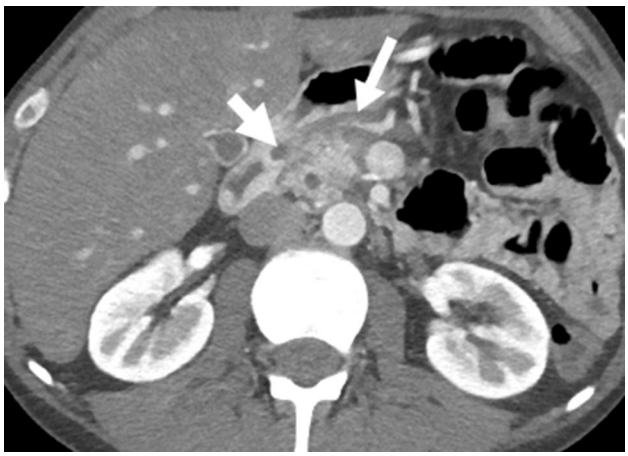

Fig. 4 Thirty-seven-year-old male with chronic calcific pancreatitis and groove pancreatitis. Axial contrast-enhanced CT image shows low attenuation tissue within the pancreaticoduodenal groove (long arrow) and an intramural duodenal cyst (short arrow)

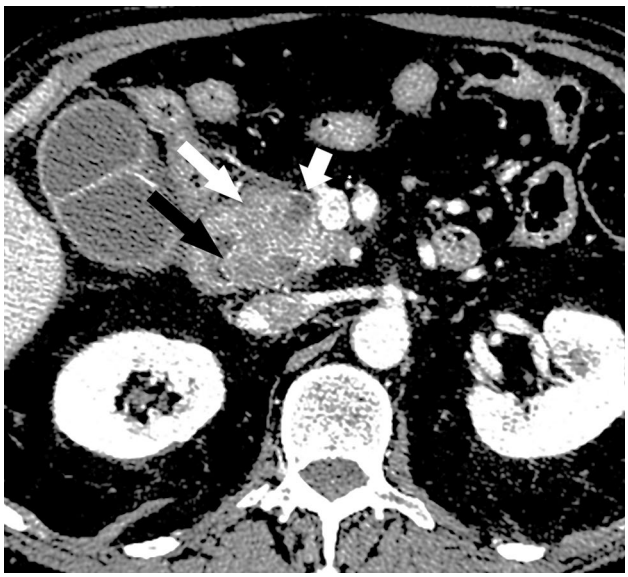

Fig. 5 Fifty-seven-year-old male with long history of smoking and groove pancreatitis. Axial CT demonstrates enhancing soft tissue in the pancreatic groove (long white arrow). Note cyst formation in the head of the pancreas (short white arrow) and duodenal wall thickening (black arrow)

performed on benign lesion due to improvement in CT and MRI/MRCP imaging [18]. Therefore, characteristic findings can make a strong argument in favor of GP. On CT and MRI, there is evidence of delayed retention of contrast within the fibrotic tissue within the pancreaticoduodenal groove that may encase the gastroduodenal artery [6, 9]. However, delayed enhancement is also demonstrated in PDAC and should be used with caution (Fig. 6). There may or may not be an associated low attenuation area within the pancreatic parenchyma. Pancreatic ductal calculi may be evident indicative of chronic calcific pancreatitis (Fig. 3). In addition, there may be long smooth tapering of the common bile duct and either intramural or paraduodenal cystic areas (Figs. 4 and 5). The medial wall of the duodenum is characteristically thickened with cystic changes (Fig. 7), but if there is extensive fibrotic tissue encasing the duodenum,

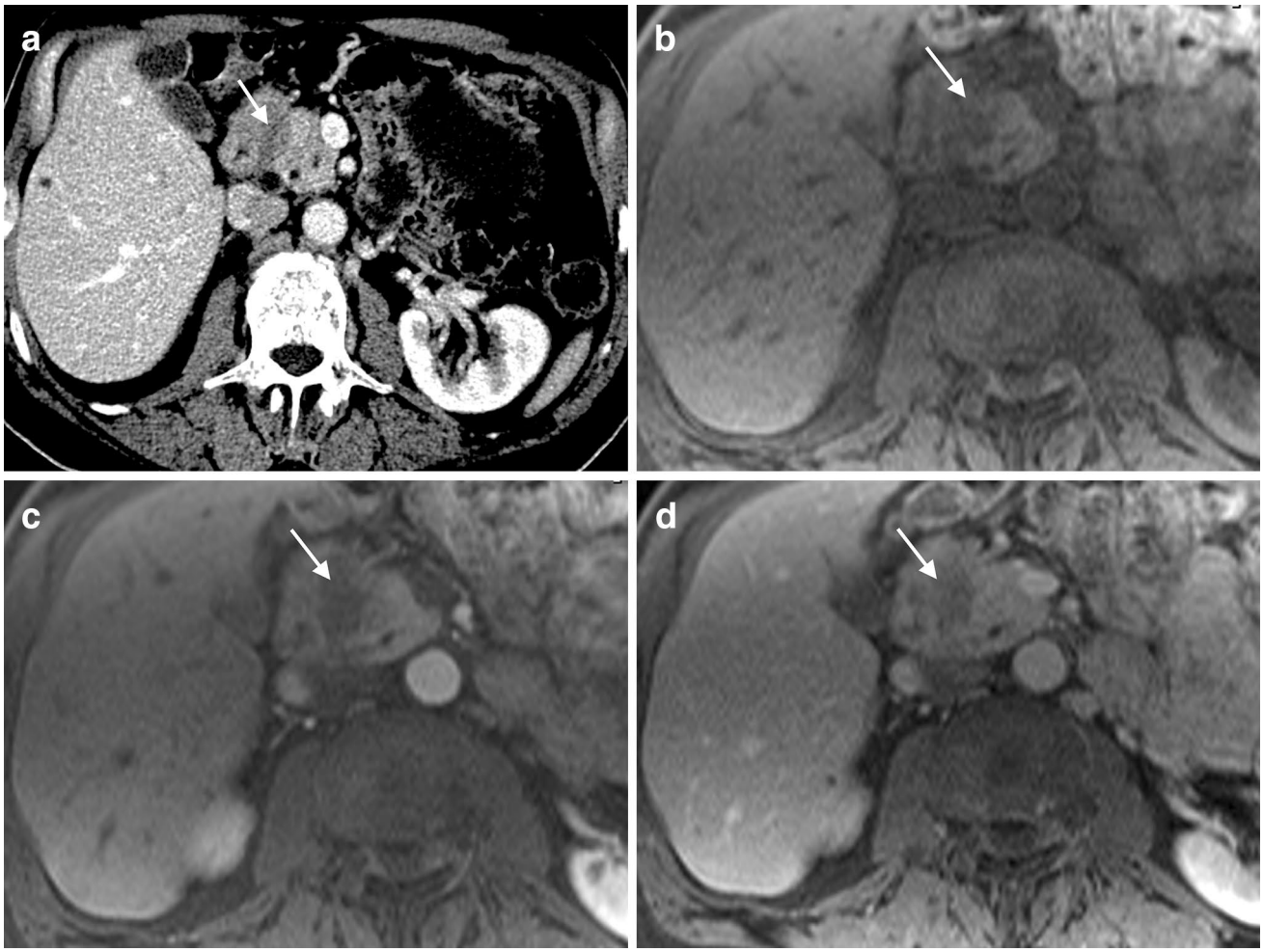

Fig. 6 Seventy-four-year-old man with abdominal pain. Axial contrast-enhanced venous phase CT **a** demonstrates soft tissue in the pancreaticoduodenal groove (arrow). There is no evidence of pancreatic duct enlargement or parenchymal atrophy. The mass demonstrates progressive enhancement on the post contrast MRI (arrows; **b** pre-contrast; **c** arterial phase; **d** venous phase). EUS guided FNA demonstrated malignant cells and pancreaticoduodenectomy was performed. Final pathology showed pancreatic ductal adenocarcinoma

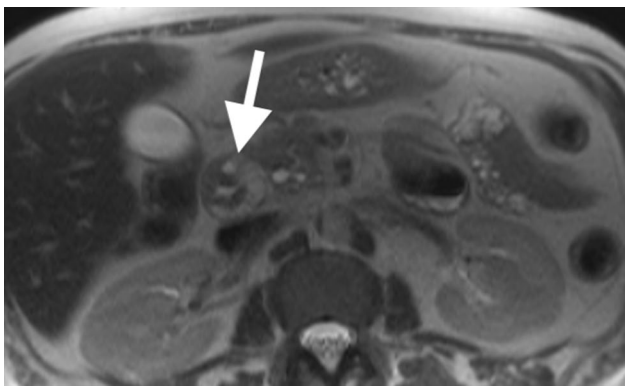

Fig. 7 Sixty-five-year old man with history of alcohol abuse and chronic abdominal pain. T2-weighted image demonstrated thickening of the duodenal wall with cystic changes which are of fluid signal (arrow). Findings are highly suggestive of GP and with a low clinical suspicion for PDAC, the patient was managed conservatively with a presumptive diagnosis of GP

there may be gastric dilatation from gastric outlet obstruction. There may or may not be dilation of the main pancreatic duct.

In contrast, the classic patient with PDAC extending to the pancreaticoduodenal groove is either a male or female in their 60s or 70s who might present with either abdominal pain or painless jaundice [3, 4, 6–9]. In addition to elevation of liver enzymes, tumor markers such as CA 19-9 are significantly elevated. With imaging, there may be a discrete hypoenhancing mass in the head of the pancreas that invades the second duodenum and extends into the pancreaticoduodenal groove to encase the gastroduodenal artery (Fig. 5). In some patients, however, it may be difficult to identify a discrete pancreatic mass separate and distinct from the soft tissue infiltrating the pancreatic groove (Fig. 8). In these patients, endoscopic biopsy is essential to confirm the diagnosis.

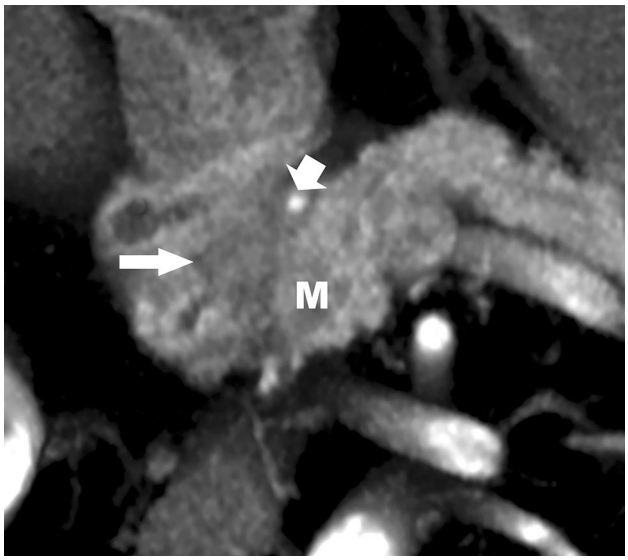

Fig. 8 Sixty-six-year-old female with pancreatic carcinoma invading pancreatic groove. Axial volume-rendered CT shows small hypodense pancreatic head mass (M) invading pancreatic groove and the duodenal wall (long white arrow). Note also the encasement of the gastroduodenal artery by the infiltrating carcinoma (short white arrow)

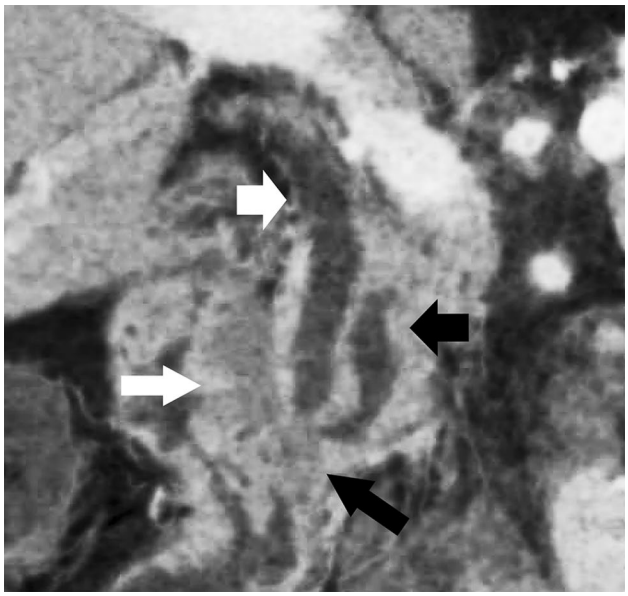

Fig. 9 Seventy-two-year-old male presenting with painless jaundice and "double duct sign" from small pancreatic carcinoma. Coronal minimum intensity projection image demonstrates small pancreatic mass (long black arrow) invading the pancreatic groove (long white arrow) and obstructing both the common bile duct (short white arrow) and the pancreatic duct (short black arrow)

Within the pancreas, in patients with PDAC involving the pancreatic groove, there is typically a "double duct" sign with a short focal stricture of the common bile duct by the mass as well as upstream dilation of the main pancreatic duct (Fig. 9). There may be distal atrophy in the body and tail of the pancreas, but pancreatic calcifications are conspicuously absent. There usually are no paraduodenal cysts.

Not surprisingly, many patients do not readily fall into these two distinct clinical categories. In an individual patient, there is frequently overlap between the clinical and imaging findings of GP and PDAC. In addition to CT and MRI, EUS with guided biopsy may be useful when there are confusing imaging findings [5]. Surgery with pancreaticoduodenectomy can provide symptomatic relief both in patients with unremitting pain from GP and when gastric outlet obstruction fails to respond to conservative treatment [17].

Conclusion

GP is an uncommon localized form of chronic pancreatitis. While in some patients, it may present with distinctive clinical and imaging features such as sheet-like hypodensity in the pancreaticoduodenal groove, medial duodenal wall thickening, and cystic changes in the duodenal wall, in others, there may be significant overlap with PDAC invading the pancreaticoduodenal groove. A healthy skepticism must be maintained about the specificity of establishing GP in order to avoid misdiagnosis.

Compliance with ethical standards

Disclosures BNP—research support (GE and Siemens); speaker's bureau (GE). RBJ, EC, AZ—no relevant disclosures.

Ethical approval The authors had control of the data and the information submitted for publication. Bhavik N. Patel received research support from GE Healthcare and is on its speaker's bureau. He also received an institutional grant from Siemens Healthcare. All other authors are not employees of or consultants for the industry and had control of inclusion of any data and information that might present a conflict of interest. There was no industry support specifically for this study.

Research involving human and animal rights This study did not involve human and animal subjects, and therefore, an Institutional Review Board approval and informed consent were not required.

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