Journal of Gastroenterology and Hepatology

JGHF

doi:10.1111/jgh.16683

REVIEW ARTICLE

Groove pancreatitis: From enigma to future directions—A comprehensive review

Dushyant S. Dahiya,[*] Yash R. Shah,[†] Andrew Canakis,[‡] Charmy Parikh,[§] Saurabh Chandan,[¶] Hassam Ali,[**] Manesh K. Gangwani,[††] Bhanu S. M. Pinnam,[‡‡] Sahib Singh,[§§] Amir H. Sohail,[¶¶] Raj Patel,[***] Daryl Ramai,[†††] Mohammad Al-Haddad,[‡‡‡] Todd Baron[§§§] and Amit Rastogi[*]

[*]Division of Gastroenterology, Hepatology and Motility, The University of Kansas School of Medicine, Kansas City, Kansas, [†]Department of Internal Medicine, Trinity Health Oakland/Wayne State University, Pontiac, Michigan, [‡]Division of Gastroenterology and Hepatology, University of Maryland School of Medicine, [§§]Department of Internal Medicine, Sinai Hospital, Baltimore, Maryland, [§]Department of Internal Medicine, Carle BroMenn Medical Center, Normal, [‡‡]Department of Internal Medicine, John H. Stroger, Jr. Hospital of Cook County, Chicago, Illinois, [¶]Division of Gastroenterology and Hepatology, Creighton University School of Medicine, Omaha, Nebraska, [**]Division of Gastroenterology, Hepatology and Nutrition, East Carolina University/Brody School of Medicine, Greenville, [§§§]Division of Gastroenterology and Hepatology, University of North Carolina, Chapel Hill, North Carolina, [††]Department of Gastroenterology and Hepatology, University of Arkansas For Medical Sciences, Little Rock, Arkansas, [¶¶]Complex Surgical Oncology, Department of Surgery, University of New Mexico, Albuquerque, New Mexico, [***]Department of Gastroenterology and Hepatology, University of Utah School of Medicine, Salt Lake City, Utah, [†††]Department of Internal Medicine, St. Mary's Medical Center, Langhorne, Pennsylvania, [‡‡‡]Division of Gastroenterology and Hepatology, Indiana University School of Medicine, Indianapolis, Indiana, USA

Key words
Diagnosis, Etiology, Groove, Management, Pancreatitis, Pancreatitis, Pathogenesis.

Accepted for publication 2 July 2024.

Correspondence
Dushyant Singh Dahiya, Division of Gastroenterology, Hepatology and Motility, The University of Kansas School of Medicine, 2000 Olathe Blvd, Kansas City 66160, KS, USA.
Email: dush.dahiya@gmail.com

Declaration of conflict of interest: Dushyant Singh Dahiya: No disclosures or conflict of interest. Yash R. Shah: No disclosures or conflict of interest. Andrew Canakis: No disclosures or conflict of interest. Charmy Parikh: No disclosures or conflict of interest. Saurabh Chandan: No disclosures or conflict of interest. Hassam Ali: No disclosures or conflict of interest. Manesh Kumar Gangwani: No disclosures or conflict of interest. Bhanu Siva Mohan Pinnam: No disclosures or conflict of interest. Sahib Singh: No disclosures or conflict of interest. Amir H. Sohail: No disclosures or conflict of interest. Raj Patel: No disclosures or conflict of interest. Mohammad Al-Haddad: No disclosures or conflict of interest. Daryl Ramai: No disclosures or conflict of interest. Todd Baron: No disclosures or conflict of interest. Amit Rastogi: No disclosures or conflict of interest.
Author contributions: Conception and design: Dushyant Singh Dahiya, Yash R. Shah, Andrew Canakis, Todd Baron and Amit Rastogi.

Abstract

Groove pancreatitis (GP) is a rare and clinically distinct form of chronic pancreatitis affecting the pancreaticoduodenal groove comprising the head of the pancreas, duodenum, and the common bile duct. It is more prevalent in individuals in their 4–5th decade of life and disproportionately affects men compared with women. Excessive alcohol consumption, tobacco smoking, pancreatic ductal stones, pancreatic divisum, annular pancreas, ectopic pancreas, duodenal wall thickening, and peptic ulcers are significant risk factors implicated in the development of GP. The usual presenting symptoms include severe abdominal pain, nausea, vomiting, diarrhea, weight loss, and jaundice. Establishing a diagnosis of GP is often challenging due to significant clinical and radiological overlap with numerous benign and malignant conditions affecting the same anatomical location. This can lead to a delay in initiation of treatment leading to increasing morbidity, mortality, and complication rates. Promising research in artificial intelligence (AI) has garnered immense interest in recent years. Due to its widespread application in diagnostic imaging with a high degree of sensitivity and specificity, AI has the potential of becoming a vital tool in differentiating GP from pancreatic malignancies, thereby preventing a missed or delayed diagnosis. In this article, we provide a comprehensive review of GP, covering the etiology, pathogenesis, clinical presentation, radiological and endoscopic evaluation, management strategies, and future directions. This article also aims to increase awareness about this lesser known and often-misdiagnosed clinical entity amongst clinicians to ultimately improve patient outcomes.

Administrative support: Dushyant Singh Dahiya, Yash R. Shah, Saurabh Chandan, and Amit Rastogi. Provision, collection, and assembly of data: Dushyant Singh Dahiya, Yash R. Shah, Andrew Canakis, Charmy Parikh, Saurabh Chandan, Hassam Ali, Manesh Kuamr Gangwani, Todd Baron and Amit Rastogi. Statistical analysis: not applicable. Review of literature: all authors. Drafting the manuscript: all authors. Revision of key components of manuscript: all authors. Final approval of manuscript: all authors. Agreement to be accountable for all aspects of the work: all authors.

Ethical approval: The data utilized in this review article is available publicly without any patient identifiers. Hence, Institutional Review Board (IRB) approval was not required as per guideline put forth by our institutional IRB for review articles.

Informed consent: The data utilized in this review article are available publicly without any patient identifiers. Hence, patient consent was not required.

Financial support: No funding was received for this study.

Introduction

First described by Becker and Mischke in 1973, GP is a rare and underrecognized subtype of chronic pancreatitis.[1,2] It was initially described in German as "Rinnenpankreatitis" until Stolte *et al.* coined the term GP in 1982.[3] Numerous clinical entities such as cystic dystrophy of heterotopic pancreas, pancreatic hamartoma of duodenum, para-duodenal wall cyst, and myoadenomatosis, along with GP, have been classified under the broad umbrella term of as paraduodenal pancreatitis as they share common clinical and pathological findings.[4] Based on the predominant area of involvement, GP has been classified as follows[5]:

1. *Pure groove pancreatitis*: Inflammation involves the groove area (anatomic space between the head of pancreas, duodenum, and common bile duct [CBD]) only.
2. *Segmental groove pancreatitis*: Inflammation involves the groove area along with extension into the head of the pancreas (Fig. 1).

Although numerous risk factors have been identified for GP, it is most closely associated with alcohol and tobacco use which is more prominent in middle aged men.[6] The usual presenting symptoms include severe abdominal pain, nausea, and vomiting due to duodenal stenosis causing luminal obstruction, diarrhea, weight loss, and jaundice when there is involvement of the intrapancreatic bile duct.[6] It is challenging to diagnose GP due to lack of characteristic imaging findings although imaging modalities like computed tomography (CT), magnetic resonance imaging (MRI), or magnetic resonance cholangiopancreatography (MRCP) may be useful in making a diagnosis. Endoscopic ultrasound (EUS) has been proven to be the most sensitive modality to detect GP.[6] The treatment options for GP depend on the severity of the disease and range from conservative to endoscopic or surgical management.[6]

This comprehensive review article aims to discuss etiopathogenesis, clinical features, management strategies, and potential future advancements in the diagnosis and management of GP.

Demographics

Although a rare clinical entity, the prevalence of GP ranges up to 20%–25% in patients with chronic pancreatitis who have undergone surgical/pathological or anatomical workup.[3,7] To put that into perspective, in a recent systematic review by Ukegjini *et al.* that analyzed 649 studies from 1990 to 2022, the authors noted 1404 diagnosed cases of GP.[8] Of 1023 patients for whom gender was reported in this study, 86% were males.[8] This was consistent with previous literature, which reports that GP is predominantly seen in middle-aged men between the ages of 40 and 50 years.[4,8–11]

Risk factors and pathogenesis

Alcohol use disorder and nicotine dependence have been reported to have a strong association with GP.[6] Ukegjini *et al.* also noted that about 79% of GP patients had a history of heavy alcohol consumption and 83% were smokers.[8] Other risk factors implicated in the development of GP include pancreatic ductal stones, pancreatic divisum, annular pancreas, ectopic pancreas, duodenal wall thickening, and peptic ulcers.[12,13] Additional large studies are needed to fully understand the impact of these risk factors on GP.

Over the last few decades, numerous pathophysiological mechanisms have been proposed that may lead to the development of GP; however, the exact pathophysiology is currently unknown. The classical pathophysiological mechanism suggests that pancreatic enzymes accumulate in the duodenal wall due to ectopic pancreatic tissue leading to Brunner's gland hyperplasia, local inflammation, and fibrosis (Fig. 1).[14] This condition manifests macroscopically as duodenal wall thickening with pseudotumorous polyploid giant folds, especially in the periampullary region.[11] Intramural cystic lesions, located within the submucosa or muscularis layer, typically range from 1 to 10 cm.[11] However, it might simulate a neoplasm if large and multiple cysts invade into the adjacent groove compressing the bile duct.[11] This is also known as pancreatic heterotopia, which was first described by Potet *et al.* in 1970.[15] Differentiating GP from

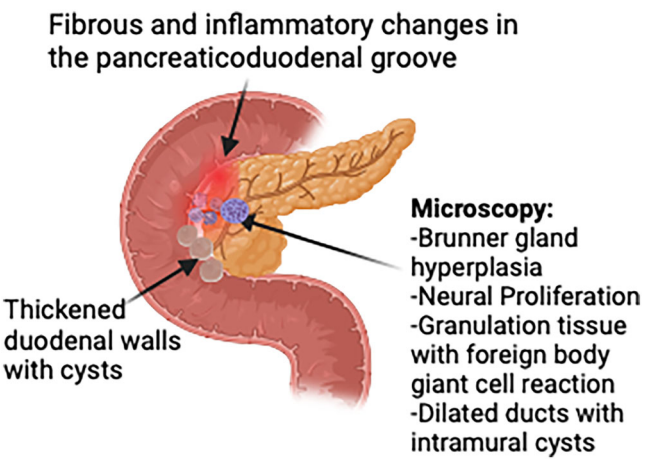

Figure 1 Pathogenesis of groove pancreatitis (created using Biorender.com).

groove carcinoma, duodenal carcinoma, and pancreas head carcinoma is crucial due to differing prognoses and management strategies.[5] Furthermore, stenosis of the second part of the duodenum may lead to gastric outlet obstruction (Fig. 1).[16]

Moreover, significant alcohol use can cause cytoplasmic lipid accumulation in acinar cells, leading to fatty degeneration, cellular necrosis, and fibrosis. Long-term alcohol abuse alters pancreatic enzyme volume and viscosity, obstructs pancreatic ducts, and forms proteinaceous plugs within the ducts.[4,5,17,18] The resulting stasis of exocrine pancreatic secretions leads to recurrent pancreatitis episodes, chronic localized inflammation, and eventually cystic dystrophy of the duodenal wall.[19]

Clinical presentation

The most common symptom of GP is upper abdominal pain, more pronounced in the right upper quadrant and epigastric region.[4] The systematic review by Ukegjini *et al*. demonstrated that abdominal pain was the most reported symptom in GP patients, followed by weight loss, nausea/vomiting, episodes of acute pancreatitis (AP), jaundice, steatorrhea, and exocrine insufficiency.[9] Asymptomatic presentation was the least common.[8] Similarly, a retrospective study on 24 patients by Bender *et al*. reported that 70.8% of the patients had abdominal pain, followed by weight loss in 41.6%, vomiting in 29.1%, and diarrhea in 20.8%, jaundice in 16.6% patients, and dysphagia in 8.3% patients with GP.[20] A retrospective study by Tarvainen *et al*. in 33 patients with confirmed GP showed similar trends with abdominal pain being the most common complaint in 87.8% followed by AP in 51.5%, nausea/vomiting 45.4%, weight loss 36.3%, and diarrhea in 12.1%.[21] Another case–control study consisting of 13 patients also reported abdominal pain, weight loss, nausea, and vomiting as the frequent presenting symptoms of GP.[19] Table 1 highlights the clinical features and their commonality in patients with GP.[20–22]

Compared with patients with chronic pancreatitis, symptoms of gastric outlet obstruction were more prominent in patients with GP while diarrhea was less common.[19] Duodenal stenosis, often seen in patients with GP, can lead to longer lasting symptoms like early satiety, vomiting, and weight loss as well.[23] Clinically, if an elderly patient (≥60 years old) presents with symptoms of jaundice, groove cancer should be suspected over GP and be in the differential diagnosis.[24] The duration of symptoms of GP can fluctuate from a few weeks to years and may be debilitating in the long term, especially in older individuals.[1] It is difficult to differentiate GP from pancreatic cancer as they share common clinical findings with very similar radiological and gross pathological features.[25]

Radiological evaluation

The main imaging modalities utilized to help establish a radiological diagnosis of GP include transabdominal ultrasound, CT, MRI, and MRCP of the abdomen.[26] However, a radiological diagnosis of GP is often challenging as it shares similar radiological features with localized pancreatic groove cancer, primary duodenal neoplasms, ampullary neoplasms, and pancreatic neoplasms.[27,28] Hence, these patients may be subjected to unnecessary surgical intervention due to misdiagnosis or suspicion of malignancy.[27] It is imperative to use clinical judgment along with imaging modalities to decrease the risk of misdiagnosis and unwarranted surgical intervention in these patients.

Table 1 Frequency of clinical symptoms in patients with groove pancreatitis

Clinical features	Range of percentage
Abdominal pain	70.8%–87.8%
Weight loss	36.3%–41.6%
Vomiting	29.1%–45.4%
Diarrhea	12.1%–20.8%
Jaundice	16.6%
Dysphagia	8.3%
Acute pancreatitis	51.5%
Nausea/vomiting	45.4%

Ultrasound. Although transabdominal ultrasound is a widely available imaging modality, radiological findings of GP on ultrasound are non-specific. Hence, a diagnosis of GP is rarely established primarily on transabdominal ultrasound. The sonographic findings of GP reported in two patients that were later confirmed histopathologically by Wronski *et al*. are a hypoechoic band like area between the pancreatic head and duodenum with enlarged low echogenic pancreatic head in the first patient and hyperechoic thick duodenal walls due to hypertrophy of the submucosal layer in the second patient.[29] These sonographic findings reflect the ongoing pathologic process and should be interpreted keeping in mind the stage of the disease.[29] Hwang *et al*. reported non-homogenous mass between the pancreatic and the descending duodenum as well as swelling of the pancreatic head on ultrasonography in a patient diagnosed with GP.[30] As it is readily available, transabdominal ultrasound is an important diagnostic tool and may serve as the initial diagnostic test to not only help diagnose GP but also rule out other clinical etiologies with a similar presentation.

Computed tomography. Loss of fat plane along with an ill-defined crescentic or frank soft tissue mass between the head of the pancreas and duodenum are the classic findings of Pure GP on multidetector CT (MDCT) (Fig. 2).[11,26] The soft tissue mass often has a sheet-like curvilinear crescentic appearance, which can be best appreciated on multiplanar reformatted images.[31] Delayed enhancement of the soft tissue is often seen on multiphase imaging due to fibrosis or a thickened medial duodenal wall, and small cysts can also be appreciated within the thickened duodenal wall or the pancreaticoduodenal groove.[32]

In patients with segmental GP, the main pancreatic duct may appear dilated in the pancreatic body and tail, whereas the pancreas mostly appears normal in patients with pure GP (Fig. 3a).[11,33] Rarely, pancreatic ducts may appear dilated in pure GP due to extrinsic compression of the pancreatic head by the groove mass.[33,34] Segmental GP can be confused with pancreatic cancer due to masking of the pancreatic groove by a mass-like enlargement of the pancreatic head, making it quite difficult to radiologically differentiate the two clinical entities.[31] On CT imaging, GP can be differentiated from acute edematous pancreatitis due to absence of diffuse retroperitoneal inflammatory changes.[31] Furthermore, pancreatic calcifications, ductal dilatation, and/or ductal

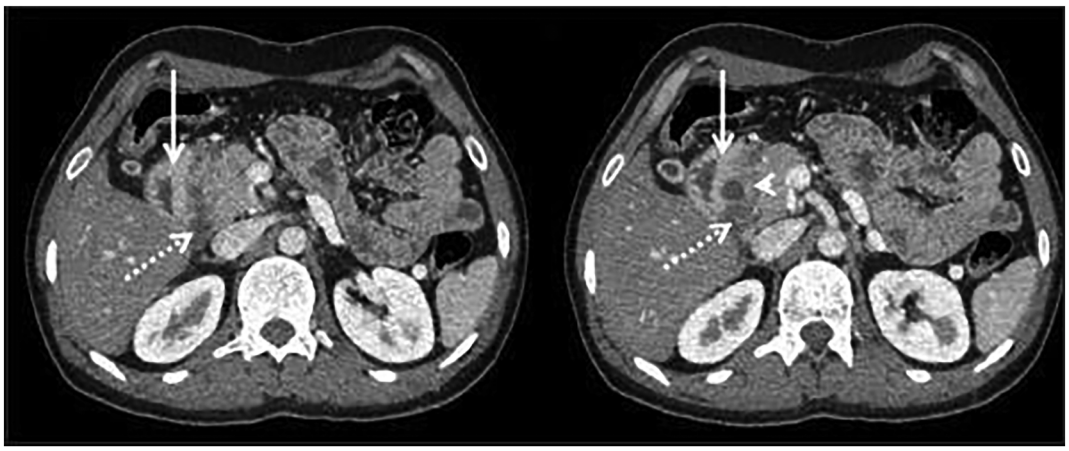

Figure 2 Axial contrast-enhanced computed tomography: duodenal wall thickening with mural hyperenhancement (arrow), paraduodenal cyst (arrowhead), and hypoenhancing soft tissue between pancreatic head and duodenum (dotted arrow).[11]

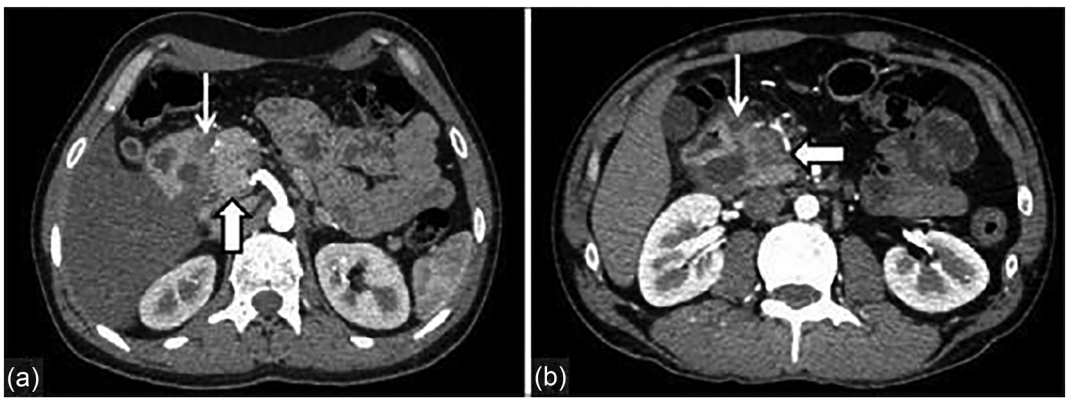

Figure 3 (a) Pure form: normal appearing pancreatic head (thick arrow), inflammation confined to groove region (thin arrow); (b) Segmental form: Involvement of head and hypoenhancing lesion (thick arrow), predominantly cystic form of disease (thin arrow).[11]

beading/irregularity due to the progressive narrowing and fibrosis of the pancreatic duct and parenchyma seen in chronic pancreatitis may help differentiate it from GP.[31]

MRI. A retrospective study conducted by Kalb *et al*. to assess the performance of MRI in distinguishing GP from pancreatic carcinoma showed that MRI is 87.2% accurate and has a negative predictive value of 92.2% for diagnosing GP.[35] MRI has a sensitivity of 88.2% and specificity of 86.7% in the diagnosis of GP, with a negative predictive value of 78.9% and a positive predictive value of 92.9%.[35] Hence, MRI is probably the most important imaging modalities in establishing a diagnosis of GP. Radiological features of GP on MRI closely resemble those seen on CT. The presence of a sheet-like mass between the head of pancreas and duodenum, which appears hypointense to pancreatic parenchyma on T1-weighted images and hypointense, isointense, or hyperintense on T2-weighted images, is the most characteristic finding of GP on MRI (Fig. 4).[11,36] The variation could be due to the time of onset of disease as subacute disease appears brighter due to edematous changes while fibrosis in chronic disease states leads to a low intensity signal.[36] Hence, these changes in signal intensity can be helpful in understanding and assessing the chronicity of the disease as well.

The involvement of medial duodenal wall (duodenal wall thickening) can be seen in both pure and segmental GP on MRI.[31,37] Multiple T2 hyperintense cysts may also be visualized in the duodenal wall and pancreaticoduodenal groove.[31,37] Furthermore, hyperplasia of Brunner's gland, encasement of the CBD, and cicatrization and stenosis of the duodenal wall seen in patients with GP can be useful in differentiating it from pancreatic carcinoma.[37]

In segmental GP, the main pancreatic duct usually has a mild, regular, and progressive pattern of narrowing in the head of the pancreas.[36] In GP patients with diffuse chronic inflammatory changes, higher degree of the duct of Wirsung narrowing along with secondary duct ectasia can be seen on MRI.[37] Additionally, "banana-shaped" gallbladder is an ancillary finding seen in GP patients on MRI due to narrowing of ampulla and stricturing of distal CBD (Fig. 5).[11,31]

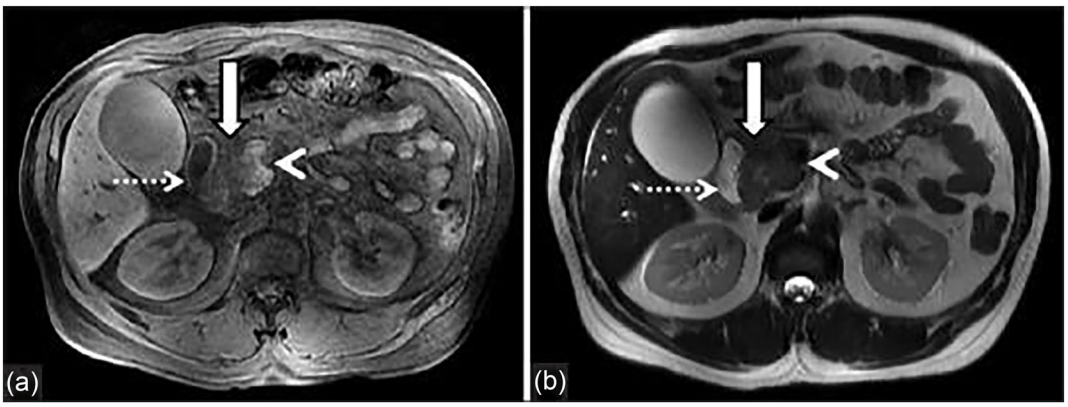

Figure 4 Magnetic resonance imaging: (a) hypointense signal on T1-weighted image; (b) isointense signal on T2-weighted image; mass (arrow) in the groove sandwiched between pancreatic head (arrow head) and descending duodenum (dotted arrow).[11]

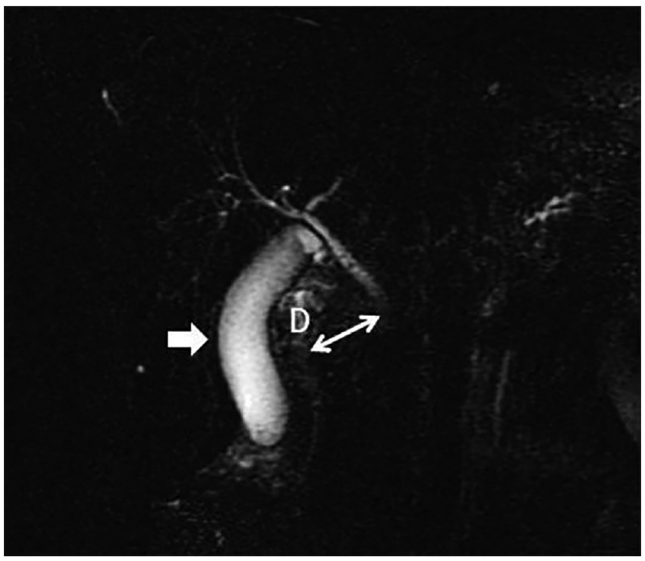

Figure 5 Magnetic resonance cholangiopancreatography: excessive widening of space between descending duodenum (D) and bile duct (thin arrow). Ectatic and banana-shaped gallbladder (thick arrow).[11]

MRCP. With recent advancements in radiological imaging over the last few decades, MRCP has gained immense popularity and evolved into an important diagnostic tool for numerous pancreaticobiliary disorders. The study by El-Nekidy *et al*. consisting of 16 GP patients demonstrated that MCRP could establish a relationship between the T2 hyperintense cysts in the duodenal wall and pancreaticoduodenal groove, CBD, and pancreatic ducts.[38] MRCP can help assess the caliber of the distal CBD and dilatation of the main pancreatic duct seen in patients with segmental GP.[26] However, additional studies are needed on a larger cohort of GP patients to identify more unique radiological differentiators of GP on MRCP from other similar clinical entities.

Endoscopic retrograde cholangiopancreatography. The role of endoscopic retrograde cholangiopancreatography (ERCP) is limited in the evaluation of patients with suspicion of GP due to possible duodenal stenosis in these cases; however, it can demonstrate smooth CBD stenosis with normal main pancreatic duct if cannulation is successful.[31,39] In a case report by Gupta *et al*., ERCP showed soft tissue swelling and a mass in the periampullary region which was later diagnosed as GP on pathological examination.[40] Another case of GP reported by Sanada *et al*. demonstrated smooth stenosis of the main pancreatic duct with pooling of contrast in the pancreatic head suggestive of a cyst along with obstruction/stenosis of the duct of Santorini on ERCP.[41]

EUS. EUS is one of the most sensitive diagnostic modalities utilized in the evaluation of pancreaticobiliary disorders. It can be instrumental in the evaluation of both pure and segmental GP.[26] EUS can accurately identify and assess the location of the cysts in the muscularis propria of duodenal wall, which appear as enlargements around the minor papilla area without communication with pancreatic ducts.[6,11,42] A heterogeneous pattern of pancreatic parenchyma, which is less hypoechoic compared with that of a carcinoma, and the absence of vascular involvement can also be seen (Fig. 6).[11,42]

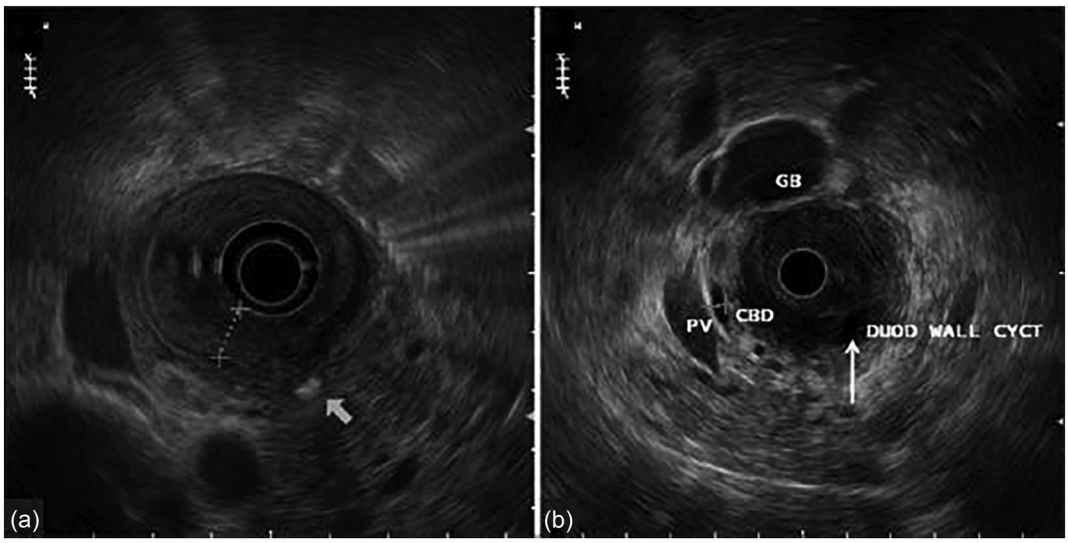

Figure 6 Endoscopic ultrasound: (a) medial duodenal wall thickening, focal pancreatic calcification (thick arrow); (b) intraparietal cysts within duodenum (thin arrow).

EUS enables visualization of the surrounding lymph nodes, which do not achieve the same size and pattern of involvement as that seen in pancreatic cancer. Furthermore, it enables therapeutic endoscopists to perform fine-needle aspiration/biopsy (FNA/FNB) of the lesion and surrounding lymph nodes.[43,44] Although a negative result on FNA/FNB does not completely rule out malignancy, it favors a more benign etiology in the appropriate clinical setting.[43,44] Hence, EUS is the modality of choice which can be used to differentiate GP from pancreatic ductal adenocarcinoma (PDAC) as it allows FNA to establish a cytohistological diagnosis early in 90% of the cases.[45] Despite being an excellent diagnostic tool, EUS has its own set of limitations. Although it is very helpful in localizing the disease and evaluating the surface involved, oftentimes it cannot adequately differentiate inflammation from infiltration and neoplasm.[26] A summary of sensitivity, specificity, positive predictive value, and negative predictive value is discussed in Table 2.[26,35,46–48]

Differential diagnosis

Several conditions involving the pancreaticoduodenal groove can mimic GP and should be considered in the differential diagnosis as discussed below:

PDAC. PDAC is one of the most important considerations and differentiation from GP, especially segmental GP, can be extremely challenging due to similar radiological and clinical features.[9] Nonetheless, it is imperative to differentiate the two clinical entities as PDAC is the fourth leading cause of cancer-related deaths in the US, while GP follows a relatively benign course.[49] Several features can help differentiate GP from PDAC. Clinically, PDAC may have overt jaundice and elevated tumor markers on presentation.[28] Radiologically, from a ductal architecture standpoint, there can be an abrupt cutoff and irregular dilatation of the main pancreatic duct due to invasion and obstruction by the cancer, whereas GP may have smooth progressive narrowing of the main pancreatic duct.[32,37] GP and PDAC have similar findings on CT and MRI in terms of a hypointense mass on T1-weighted imaging and isointense to hyperintense on T2-weighted imaging with delayed progressive enhancement with contrast; however, enhancement can be patchy and heterogenous in GP while it can be homogeneous and hypointense in PDAC.[37,50]

Histologically, GP is characterized by periarterial fibrosis which affects the small arterioles but does not involve any other major vessel.[37] On the other hand, PDAC can infiltrate major vessels including the gastroduodenal artery and the retroperitoneal space. Furthermore, although both GP and PDAC have luminal stenosis secondary to duodenal wall thickening, the presence of cystic changes is highly suggestive of GP.[32,37]

Duodenal adenocarcinoma. Duodenal adenocarcinoma is a malignant lesion arising from the duodenal epithelium. Due to its distinct anatomical location, it may be a challenge to differentiate from GP. Duodenal adenocarcinoma may present as a mass-like lesion circumferentially involving the duodenum with or without nodal disease, or it may have an infiltrative component involving adjacent fat planes and encasing vascular structures.[51] Accurate distinction between a duodenal mass and an inflammatory process centered in the pancreaticoduodenal groove can be made by focusing on coronal multiplanar reformats on radiological imaging.[31] A few studies on CT and MRI perfusion have also shown increased mean blood flow and higher blood volume in inflammation compared with carcinoma, and a lower mean apparent diffusion coefficient on diffusion-weighted imaging in malignancy.[52,53] Furthermore, if the duodenal adenocarcinoma arises close to the ampulla, a "double duct" sign can be seen on CT scan as it can obstruct the biliary and pancreatic ducts.[51] In cases where traditional radiological distinction between GP and duodenal adenocarcinoma is difficult, EUS with FNB may serve as a vital diagnostic modality.[31]

Table 2 Summary of clinical performance of various imagining modalities for work-up of pancreatitis

Imaging modality	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Ultrasound[46]	38%–100%	34%–100%	—	—
Computed tomography[46,47]	58%–100%	59%–100%	81.1%	100%
Magnetic resonance imaging[35]	88.2%	86.7%	78.9%	92.9%
Magnetic resonance cholangiopancreatography[35]	85%	96%	—	—
Endoscopic ultrasound[48]	91%–100%	85%–100%	92%–100%	92%–95%
EUS FNA/FNB[26]	90%	100%	100%	93%

Abbreviations: EUS, endoscopic ultrasound; FNA, fine-needle aspiration; FNB, fine-needle biopsy.

Gastrointestinal stromal tumors. Gastrointestinal stromal tumors (GISTs) are one of the most common mesenchymal tumors arising from the gastrointestinal tract. It has been reported that about 3%–5% GIST arises from the duodenum.[54] On MRI, GIST can alter signal intensity based on the presence or absence of tumor necrosis, hemorrhage, and cystic degeneration.[55] Furthermore, solid components are T1 hypointense and T2 hyperintense on MRI, and contrast enhancement is usually uniform in small tumors, whereas it is heterogeneous in larger tumors.[55] GIST arising from the medial wall of duodenum may appear hypodense and become difficult to differentiate from GP.[28,31] Hence, it is an important differential to consider. Similar to duodenal adenocarcinoma, EUS may also serve as an important diagnostic tool in differentiating GIST from GP.

Intraductal papillary mucinous neoplasm. Intraductal papillary mucinous neoplasms (IPMNs) are intraductal mucin producing tumors with papillary projections that lead to the cystic dilatation of pancreatic ducts or its side branches.[4,56,57] When seen in the head of the pancreas, they may mimic GP on radiological imaging. On endoscopic evaluation, gaping papilla or "fish-mouth" papillae may be visualized due to the production of copious amounts of mucin from the main pancreatic duct in patients with IPMNs.[28] As cystic chances of IPMNs mimic GP on traditional radiological imaging, EUS-FNA can be useful in differentiating between two clinical entities. Furthermore, carcinoembryonic antigen levels of the intralesional contents are also higher in patients with IPMN as compared with GP.[28]

Benign neoplasms. Benign neoplasms in the ampulla and the periampullary region are rare and represent $< 10\%$ of all neoplasms in this anatomical location.[58] Adenomas are the most common benign lesions, but they have some risk of malignant conversion with time.[58] Ampullary adenomas are commonly seen in patients with familial hereditary polyposis and are often discovered incidentally on endoscopy.[59] However, they can present with signs or symptoms of biliary or pancreatic duct obstruction based on location.[59] Contrast-enhanced CT and MRI can be useful in detecting aggressive features like ulceration, heterogeneity, vascular invasion, and metastasis seen in carcinomas whereas adenomas normally appear as smooth, marginated, and enhancing mass in the periampullary region, making it difficult to differentiate adenomas from GP.[60] Hence, in these cases, traditional imaging, coupled with EUS and clinical judgment may be required to help establish a diagnosis.

Acute edematous pancreatitis. It is important to differentiate conventional acute edematous pancreatitis from GP if the pancreaticoduodenal groove is involved. AP is caused by the premature activation of digestive enzymes in the pancreatic acinar cells leading to inflammation and subsequent autodigestion of the pancreas.[61] On imaging, AP usually involves a substantial portion of the pancreatic parenchyma with inflammation extending to the pararenal spaces and peripancreatic fluid.[31] However, GP has minimal retroperitoneal inflammation, and the involvement of pancreas is limited to the pancreatic head and the pancreaticoduodenal groove.[31] Another important differentiating feature is the lipase levels, which are normal to borderline high in GP and more than two times the upper limit of normal in patients with AP. Furthermore, AP usually resolves on follow-up imaging while imaging findings of GP often persist.[62] A summary of the differential diagnosis to consider for GP is outlined in Table 3.[25,31,50,56,63–73]

Management

The management of GP can be divided into three main categories as described below.

Conservative management. Conservative management is recommended initially if the clinical, radiological, and histopathological evidence is suggestive of GP.[62] Conservative management includes sufficient bowel rest, quitting alcohol and tobacco use, utilization of proton pump inhibitor therapy, avoidance of fatty food and adequate, yet aggressive, pain management.[25,62] Acetaminophen is the recommended first line medication for pain control. However, neuromodulators such as gabapentin, pregabalin, and tricyclic antidepressants are potential alternatives.[74] Additional therapeutic options include antispasmodics and muscle relaxants such as hyoscyamine and baclofen, respectively.[74] Some studies have also demonstrated the effectiveness of somatostatin analogs such as octreotide in the management of GP.[75] However, a systematic review by Ukegjini *et al*. refuted this literature as the authors showed that the utilization of analgesics and octreotide was not very effective in management of GP as complete pain relief was not achieved.[8] If all above-mentioned pain management options fail to provide symptomatic relief, then narcotics can be considered, keeping in mind that they should be used cautiously when addressing pain in individuals with chronic pancreatitis.[72] Neurostimulation techniques such as spinal cord stimulation and transcranial magnetic stimulation have shown potential; however,

Table 3 Differential diagnosis in patients with groove pancreatitis

Diagnosis	Clinical features	Histopathological features	Radiological features
Groove pancreatitis	Severe postprandial upper abdominal pain, nausea, weight loss[25]	Irregular cysts with proteinaceous material, myofibroblast proliferation with occasional atypical mitotic figures, Brunner gland hyperplasia[25]	Ill-defined fat stranding and inflammatory changes in the pancreaticoduodenal groove. Sheet-like curvilinear crescentic shape. Increased delayed heterogeneous enhancement due to fibrosis[31]
Pancreatic ductal adenocarcinoma	Asymptomatic in early stages, dull nonspecific abdominal pain, jaundice, pancreatic exocrine insufficiency (steatorrhea, weight loss, bloating)[63]	Infiltrating glandular duct like structures, loss of SMAD4 protein in 55% patients, desmopressin reaction[64]	Lack of internal cystic changes, retroperitoneal infiltration and vascular encasement, homogeneously hypodense enhancement[31,50]
Duodenal Adenocarcinoma	Late symptoms, abdominal pain, nausea, weight loss, vomiting, and fatigue[65]	Tubular/cribriform glands lined by columnar neoplastic cells, simple cuboidal/columnar cells glands with pleomorphic nuclei in pancreaticobiliary type, MUCC1, MUC2, CDX 2 positive[65,66]	Focal thickening of medical duodenal wall or mass like thickening of duodenum, "double duct sign" in lesions close to ampulla, infiltration of adjacent fat planes and encasement of vasculature[31,51]
Gastrointestinal stromal tumors	Gastrointestinal bleed, anemia, abdominal distension, and pain[67]	Spindle cells arranged in short fascicles or whirls, epithelioid cells arranged in nested or diffuse pattern, KIT + ve[67,68]	Uniform contrast enhancement, evidence of necrosis/hemorrhage/cystic degeneration[31,55]
Intraductal papillary mucinous neoplasm	Asymptomatic or abdominal pain, back pain, nausea, vomiting, jaundice[69]	Mucin producing cells with varying degrees of dysplasia, tall columnar cells in intestinal type, complex thin branching papillae and cuboidal cells with prominent nuclei in pancreaticobiliary type. MUC1/MUC2/CEA/CA 19–9/CK19, CK-17 + ve[70]	Distension or main pancreatic duct or multi-ocular grape like cystic appearance due to cystic dilatation of pancreatic ducts, "fish mouth" papillae on endoscopic ultrasound[28,70]
Acute edematous pancreatitis	Moderate to severe epigastric abdominal pain, nausea, vomiting[71]	Edematous and acute inflammatory cells with limited fat necrosis, marked hemorrhage and coagulative necrosis with intravascular thrombi in acute necrotizing forms[71]	Pancreatic enlargement and decreased parenchymal echogenicity due to interstitial edema. Inflammation extending to pararenal space and peripancreatic fluid collection with resolution on follow up imaging[31,72]

they are rarely used in conventional treatment due to a lack of evidence supporting their use.[76]

Endoscopic management. The evidence on endoscopic management of GP is limited and should be directed towards pancreatic duct strictures with upstream dilation and symptomatic pseudocysts. ERCP with pancreatic duct stenting has become a successful treatment strategy in patients with pancreatic duct stricture.[77] Studies have already demonstrated excellent clinical outcomes of endoscopic intervention via ERCP for these strictures.[78,79] A retrospective study by Chantarojanasiri *et al*. showed successful outcomes of endoscopic transpapillary stenting and dilatation in six out of seven patients specifically with GP.[80] Endoscopic cystoduodenostomy, which is used to drain pancreatic fluid collections, has proven to be a successful treatment option for these individuals with symptomatic collections.[81] Another study by Arvanitakis *et al*. reported 94.1% survival rate and 70.7% clinical success in patients with paraduodenal pancreatitis undergoing various endoscopic interventions including cystenterostomy, pancreatic/biliary duct drainage, and/or duodenal dilatation.[82] Endoscopic intervention can also be employed for pain management in patients with inadequate pain control with conservative strategies. EUS-guided celiac plexus blocks, which involve the injection of a steroid and a long-acting local anesthetic to the celiac ganglion, are a potential option for pain relief in these patients.[83] However, additional large studies are still needed to fully investigate the efficacy of celiac plexus blocks in these patients.

Surgical management. Surgery remains the therapeutic option for patients who do not respond to conservative management, as well as for patients who have ambiguous clinical diagnoses including lesions that are highly suspicious of malignancy, or leading to refractory obstruction of pancreatic duct, bile duct, or the duodenum.[10,84] Pancreaticoduodenectomy, often known as Whipple's surgery, is the preferred surgical technique.[10,84] In some cases, pancreatectomy with duodenal preservation, notably the pylorus-preserving Whipple technique, has resulted in satisfactory outcomes. It can, however, be difficult in cases of severe fibrosis enveloping the duodenum.[31,85] Other strategies used for individuals with significant duodenal stenosis or who are ineligible for pancreatectomy include duodenal resection while preserving the pancreas.[86,87] In the year 2017, a ground-breaking surgical technique called the groove resection of pancreatic head procedure was introduced which involves selective resection of the pancreatic groove area. A large portion of the pancreatic head, main pancreatic duct, CBD, and duodenum are all preserved using this procedure. This method, which has demonstrated feasibility and effectiveness, presents a possible alternative for surgical management of GP in instances without duodenal stenosis.[88]

There is an ongoing debate on the comparative effectiveness of endoscopic versus surgical intervention in the management of

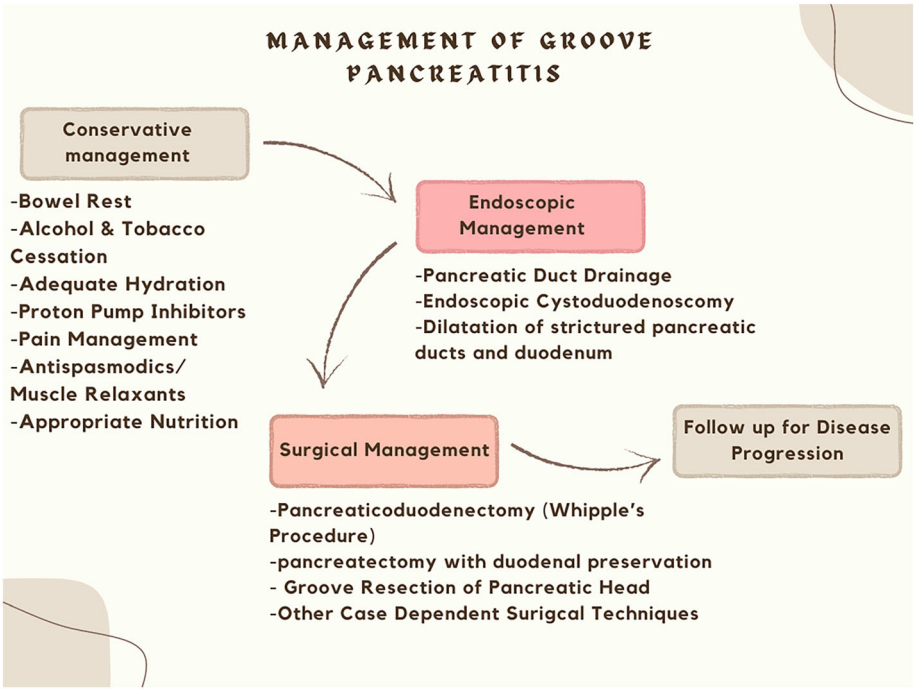

Figure 7 Outline of the management of groove pancreatitis.

Table 4 Management options for groove pancreatitis

Management	Advantages	Limitations
Conservative management		
Bowel rest, quitting alcohol and tobacco, proton pump inhibitors, avoiding fatty food, pain management	- Non-invasive - Initial approach for symptom management - Utilizes widely available medications and lifestyle modifications	- Limited effectiveness for severe cases - Potential need for narcotics - Mixed evidence on efficacy of some treatments (e.g., octreotide)
Acetaminophen (first line), gabapentin, pregabalin, tricyclic antidepressants, antispasmodics, muscle relaxants, octreotide	- Offers various pain management options - Can provide symptomatic relief - Neurostimulation techniques show potential	- Neuromodulators and narcotics require cautious use - Limited evidence for neurostimulation
Endoscopic management		
ERCP with pancreatic duct stenting, endoscopic cystoduodenostomy, EUS-guided celiac plexus blocks	- Effective for pancreatic duct strictures - Minimally invasive	- Limited evidence and studies - Potential need for repeat procedures - Efficacy of celiac plexus blocks not fully established
Surgical management		
Pancreaticoduodenectomy (Whipple's surgery), pylorus-preserving Whipple technique, duodenal resection, groove resection of pancreatic head	- Effective for severe or refractory cases - Addresses obstruction and suspicious lesions - Various techniques available depending on severity and location	- Invasive with higher risk of complications - Requires skilled surgical expertise

Abbreviation: ERCP, endoscopic retrograde cholangiopancreatography; EUS, endoscopic ultrasound.

chronic pancreatitis. Three randomized controlled trials have shown surgical intervention to be far more effective compared with endoscopic intervention for chronic pancreatitis.[89–91] However, there is a lack of data comparing the safety and effectiveness of these two treatment modalities specifically for patients with GP. Although medical management is safe and effective for GP, there should be close follow up to assess disease progression due to an inability to exclude malignancy with a high degree of accuracy in these patients. The management strategy for patients with GP is highlighted in Figure 7. A summary of advantages and limitations of various management options for GP is discussed in Table 4.

Future directions and the role of artificial intelligence in differentiating malignant pancreatic lesions from non-malignant lesions

The application of artificial intelligence (AI) is rapidly emerging in imaging-based techniques, particularly in the field of radiology and gastroenterology. Endoscopy and cross-sectional imaging are widely utilized in gastroenterology for the evaluation and management of various diseases. These modalities sometimes contain far more visual information than the human eye can distinguish.[92,93] The majority of research on AI in pancreas is based on the concept of deep learning, in which the algorithms are designed automatically to identify the patterns in data based on multiple examples instead of being explicitly programmed, and radiomics that involves the conversion of imaging data into high-dimensional mineable features that can aid in characterization of the inherent spatial heterogeneity of neoplasm and other non-neoplastic lesions.[93,94] The diagnosis and management of complex pancreaticobiliary disease relies heavily on imaging. Numerous algorithms have been developed to aid in the differentiation of various pathologies like pancreatic cancer, autoimmune pancreatitis, AP, pancreatic cystic lesions, and biliary strictures by identifying and assessing significant landmarks like ampulla on ERCP, bile duct, pancreas, and portal influence on EUS.[95] Although there are no specific studies that have studied the role of AI in diagnosis of GP, there are significant data available on chronic pancreatitis. Similar principles of AI can be applied for GP in differentiating it from other entities like pancreatic carcinoma, pancreatic ductal adenocarcinoma, benign pancreatic cysts, and the normal pancreas. Hence, AI is sure to make an impact in diagnosing GP early and easily in the near future, thereby preventing unnecessary interventions.

Conclusions

Despite its distinct clinical profile, establishing a diagnosis of GP remains challenging. GP disproportionately affects males and is commonly linked to excessive alcohol consumption and tobacco use. Symptoms often include abdominal pain, nausea, vomiting, diarrhea, weight loss, and jaundice. Classical diagnostic imaging with transabdominal ultrasound and CT scan may demonstrate non-specific radiological features leading to a missed or delayed diagnosis. MRI and MRCP provide better quality imaging with distinct radiological features. However, EUS is necessary in these patients as it offers a higher degree of diagnostic accuracy. Management of GP is primarily divided into three broad categories, namely, conservative, endoscopic, and surgical. Conservative management typically includes bowel rest, proton pump inhibitors, and aggressive pain management. EUS-guided celiac plexus blocks may help with inadequate pain control, while ERCP with pancreatic duct stenting is indicated for duct strictures. Surgical intervention, potentially including a pylorus-preserving Whipple procedure or the novel "groove resection of pancreatic head," is considered for patients who do not respond to conservative treatment. Future developments in AI-based diagnostic imaging models promise improved differentiation of GP from similar conditions, particularly malignancies. Until then, a combination of clinical suspicion, expert judgment, and advanced imaging techniques remains essential for diagnosing GP.

References

1 Gábos G, Nicolau C, Martin A, Moșteanu O. Groove pancreatitis—tumor-like lesion of the pancreas. *Diagnostics* 2023; **13**: 866.
2 Teo J, Suthananthan A, Pereira R, Bettington M, Slater K. Could it be groove pancreatitis? A frequently misdiagnosed condition with a surgical solution. *ANZ J. Surg.* 2022; **92**: 2167–73.
3 Stolte M, Weiss W, Volkholz H, Rösch W. A special form of segmental pancreatitis: "groove pancreatitis". *Hepatogastroenterology* 1982; **29**: 198–208.
4 Adsay NV, Zamboni G. Paraduodenal pancreatitis: a clinico-pathologically distinct entity unifying "cystic dystrophy of heterotopic pancreas", "para-duodenal wall cyst", and "groove pancreatitis". *Semin. Diagn. Pathol.* 2004; **21**: 247–54.
5 Değer K, Köker I, Destek S, Toprak H, Yapalak Y, Gönültaş C, Şentürk H. The clinical feature and outcome of groove pancreatitis in a cohort: a single center experience with review of the literature. *Turk. J. Trauma Emerg. Surg.* 2022; **28**: 1186–92.
6 Okasha H, Gouda M, Tag-Adeen M *et al*. Clinical, radiological, and endoscopic ultrasound findings in groove pancreatitis: a multicenter retrospective study. *Turk. J. Gastroenterol.* 2023; **34**: 771–8.
7 Becker V, Mischke U. Groove pancreatitis. *Int. J. Pancreatol. Off. J. Int. Assoc. Pancreatol.* 1991; **10**: 173–82.
8 Ukegjini K, Steffen T, Tarantino I *et al*. Systematic review on groove pancreatitis: management of a rare disease. *BJS Open* 2023; **7**: null.
9 Manzelli A, Petrou A, Lazzaro A, Brennan N, Soonawalla Z, Friend P. Groove pancreatitis. A mini-series report and review of the literature. *J. Pancreas* 2011; **12**: 230–3.
10 Casetti L, Bassi C, Salvia R *et al*. "Paraduodenal" pancreatitis: results of surgery on 58 consecutives patients from a single institution. *World J. Surg.* 2009; **33**: 2664–9.
11 Arora A, Rajesh S, Mukund A, Patidar Y, Thapar S, Arora A, Bhatia V. Clinicoradiological appraisal of 'paraduodenal pancreatitis': pancreatitis outside the pancreas! *Indian J. Radiol. Imaging* 2015; **25**: 303–14.
12 Shin LK, Jeffrey RB, Pai RK, Raman SP, Fishman EK, Olcott EW. Multidetector CT imaging of the pancreatic groove: differentiating carcinomas from paraduodenal pancreatitis. *Clin. Imaging* 2016; **40**: 1246–52.
13 Zamboni G, Capelli P, Scarpa A, Bogina G, Pesci A, Brunello E, Klöppel G. Nonneoplastic mimickers of pancreatic neoplasms. *Arch. Pathol. Lab. Med.* 2009; **133**: 439–53.
14 Vujasinovic M, Mucelli RP, Grigoriadis A *et al*. Paraduodenal pancreatitis—problem in the groove. *Scand. J. Gastroenterol.* 2022; **57**: 726–33.
15 Potet F, Duclert N. Cystic dystrophy on aberrant pancreas of the duodenal wall. *Arch. Fr. Mal. App. Dig.* 1970; **59**: 223–38.

16 Kim JD, Han YS, Choi DL. Characteristic clinical and pathologic features for preoperative diagnosed groove pancreatitis. *J. Korean Surg. Soc.* 2011; **80**: 342–7.
17 Stevens T, Conwell DL, Zuccaro G. Pathogenesis of chronic pancreatitis: an evidence-based review of past theories and recent developments. *Off. J. Am. Coll. Gastroenterol. ACG* 2004; **99**: 2256.
18 Brar HS, Shah NJ, Bukeirat F. Groove pancreatitis. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing, 2023 [cited 2023 Nov 8]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK589647/.
19 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.
20 Bender MT, Sauer B, Shami VM. Groove pancreatitis: management and outcome of patients at a tertiary care center: 22. *Off. J. Am. Coll. Gastroenterol. ACG* 2018; **113**: S13.
21 Tarvainen T, Nykänen T, Parviainen H *et al*. Diagnosis, natural course and treatment outcomes of groove pancreatitis. *HPB* 2021; **23**: 1244–52.
22 de Pretis N, Capuano F, Amodio A *et al*. Clinical and morphological features of paraduodenal pancreatitis: an Italian experience with 120 patients. *Pancreas* 2017; **46**: 489–95.
23 Latham J, Sanjay P, Watt DG, Walsh SV, Tait IS. Groove pancreatitis: a case series and review of the literature. *Scott. Med. J.* 2013; **58**: e28–31.
24 Jun JH, Lee S, Kim S *et al*. Comparison between groove carcinoma and groove pancreatitis. *Pancreatol. Off. J. Int. Assoc. Pancreatol. IAP Al* 2018; **18**: 805–11.
25 DeSouza K, Nodit L. Groove pancreatitis: a brief review of a diagnostic challenge. *Arch. Pathol. Lab. Med.* 2015; **139**: 417–21.
26 Addeo G, Beccani D, Cozzi D *et al*. Groove pancreatitis: a challenging imaging diagnosis. *Gland Surg.* 2019; **8**: S178–87.
27 Bonatti M, de Pretis N, Zamboni G *et al*. Imaging of paraduodenal pancreatitis: a systematic review. *World J. Radiol.* 2023; **15**: 42–55.
28 Mittal PK, Harri P, Nandwana S *et al*. Paraduodenal pancreatitis: benign and malignant mimics at MRI. *Abdom. Radiol.* 2017; **42**: 2652–74.
29 Wronski M, Karkocha D, Slodkowski M, Cebulski W, Krasnodebski IW. Sonographic findings in groove pancreatitis. *J. Ultrasound Med. Off. J. Am. Inst. Ultrasound Med.* 2011; **30**: 111–5.
30 Hwang JY, Park KS, Cho KB, Hwang JS, Ahn SH, Park SK, Kwon JH. Segmental groove pancreatitis: report of one case. *Korean J. Intern. Med.* 2003; **18**: 234–7.
31 Raman SP, Salaria SN, Hruban RH, Fishman EK. Groove pancreatitis: spectrum of imaging findings and radiology-pathology correlation. *Am. J. Roentgenol.* 2013; **201**: W29–39.
32 Itoh S, Yamakawa K, Shimamoto K, Endo T, Ishigaki T. CT findings in groove pancreatitis: correlation with histopathological findings. *J. Comput. Assist. Tomogr.* 1994; **18**: 911–5.
33 Ray S, Ghatak S, Misra D *et al*. Groove pancreatitis: report of three cases with brief review of literature. *Indian J. Surg.* 2017; **79**: 344–8.
34 Coakley FV, Hanley-Knutson K, Mongan J, Barajas R, Bucknor M, Qayyum A. Pancreatic imaging mimics: part 1, imaging mimics of pancreatic adenocarcinoma. *AJR Am. J. Roentgenol.* 2012; **199**: 301–8.
35 Kalb B, Martin DR, Sarmiento JM *et al*. Paraduodenal pancreatitis: clinical performance of MR imaging in distinguishing from carcinoma. *Radiology* 2013; **269**: 475–81.
36 Blasbalg R, Baroni RH, Costa DN, Machado MCC. MRI features of groove pancreatitis. *AJR Am. J. Roentgenol.* 2007; **189**: 73–80.
37 Castell-Monsalve FJ, Sousa-Martin JM, Carranza-Carranza A. Groove pancreatitis: MRI and pathologic findings. *Abdom. Imaging* 2008; **33**: 342–8.
38 El-Nekidy A, Ibrahim ME, Abdelgawad M, Abouyoussef R, Abdelkader AA, Elfaiomy TA. Groove pancreatitis: imaging features and management. *Egypt. J. Radiol. Nucl. Med.* 2016; **47**: 1175–84.
39 Malde DJ, Oliveira-Cunha M, Smith AM. Pancreatic carcinoma masquerading as groove pancreatitis: case report and review of literature. *JOP J. Pancreas* 2011; **12**: 598–602.
40 Groove pancreatitis in a patient with recurrent abdominal pain and history of alcohol misuse [Internet]. Consultant360 2023 [cited 2024 Jan 18]. Available from: https://www.consultant360.com/article/consultant360/groove-pancreatitis-patient-recurrent-abdominal-pain-and-history-alcohol
41 Sanada Y, Yoshida K, Itoh H, Kunita S, Jinushi K, Matsuura H. Groove pancreatitis associated with true pancreatic cyst. *J. Hepatobiliary Pancreat. Surg.* 2007; **14**: 401–9.
42 Laugier R, Grandval P. Does paraduodenal pancreatitis systematically need surgery? *Endoscopy* 2014; **46**: 588–90.
43 Brosens L, Leguit RJ, Vleggaar FP, Veldhuis WB, van Leeuwen MS, Offerhaus GJA. EUS-guided FNA cytology diagnosis of paraduodenal pancreatitis (groove pancreatitis) with numerous giant cells: conservative management allowed by cytological and radiological correlation. *Cytopathol. Off. J. Br. Soc. Clin. Cytol.* 2015; **26**: 122–5.
44 Chute DJ, Stelow EB. Fine-needle aspiration features of paraduodenal pancreatitis (groove pancreatitis): a report of three cases. *Diagn. Cytopathol.* 2012; **40**: 1116–21.
45 Iglesias García J, Lariño Noia J, Domínguez Muñoz JE. Endoscopic ultrasound in the diagnosis and staging of pancreatic cancer. *Rev. Esp. Enferm. Dig.* 2009; **101**: 631–8.
46 Nordaas IK, Engjom T, Gilja OH, Havre RF, Sangnes DA, Haldorsen IS, Dimcevski G. Diagnostic accuracy of transabdominal ultrasound and computed tomography in chronic pancreatitis: a head-to-head comparison. *Ultrasound Int. Open* 2021; **7**: E35.
47 Rehan A, Shabbir Z, Shaukat A, Riaz O. Diagnostic accuracy of modified CT severity index in assessing severity of acute pancreatitis. *J. Coll. Physicians Surg--Pak. JCPSP* 2016; **26**: 967–70.
48 Kotwal V, Talukdar R, Levy M, Vege SS. Role of endoscopic ultrasound during hospitalization for acute pancreatitis. *World J Gastroenterol: WJG* 2010; **16**: 4888–91.
49 Siegel RL, Miller KD, Jemal A. Cancer statistics, 2016. *CA Cancer J. Clin.* 2016; **66**: 7–30.
50 Ishigami K, Tajima T, Nishie A *et al*. Differential diagnosis of groove pancreatic carcinomas vs. groove pancreatitis: usefulness of the portal venous phase. *Eur. J. Radiol.* 2010; **74**: e95–100.
51 Bello HR, Sekhar A, Filice RW, Radmard AR, Davarpanah AH. Pancreaticoduodenal groove: spectrum of disease and imaging features. *Radiographics* 2022; **42**: 1062–80.
52 Yadav AK, Sharma R, Kandasamy D *et al*. Perfusion CT—can it resolve the pancreatic carcinoma versus mass forming chronic pancreatitis conundrum? *Pancreatology* 2016; **16**: 979–87.
53 Zhang TT, Wang L, Liu HH, Zhang CY, Li XM, Lu JP, Wang DB. Differentiation of pancreatic carcinoma and mass-forming focal pancreatitis: qualitative and quantitative assessment by dynamic contrast-enhanced MRI combined with diffusion-weighted imaging. *Oncotarget* 2016; **8**: 1744–59.
54 Marano L, Boccardi V, Marrelli D, Roviello F. Duodenal gastrointestinal stromal tumor: From clinicopathological features to surgical outcomes. *Eur. J. Surg. Oncol. J. Eur. Soc. Surg. Oncol. Br. Assoc. Surg. Oncol.* 2015; **41**: 814–22.
55 Yang F, Jin C, Du Z, Subedi S, Jiang Y, Li J *et al*. Duodenal gastrointestinal stromal tumor: clinicopathological characteristics, surgical outcomes, long term survival and predictors for adverse outcomes. *Am. J. Surg.* 2013; **206**: 360–7.
56 Fong ZV, Fernández-Del CC. Intraductal papillary mucinous neoplasm of the pancreas. *Surg. Clin. North Am.* 2016; **96**: 1431–45.

57 Sakorafas GH, Smyrniotis V, Reid-Lombardo KM, Sarr MG. Primary pancreatic cystic neoplasms revisited. Part III. Intraductal papillary mucinous neoplasms. *Surg. Oncol.* 2011; **20**: e109–18.
58 Kim AL, Choi YI. Safety of duodenal ampullectomy for benign periampullary tumors. *Ann. Hepato-Biliary-Pancreat Surg.* 2017; **21**: 146–50.
59 Chini P, Draganov PV. Diagnosis and management of ampullary adenoma: the expanding role of endoscopy. *World J. Gastrointest. Endosc.* 2011; **3**: 241–7.
60 Alessandrino F, Ivanovic AM, Yee EU, Radulovic D, Souza D, Mortele KJ. MDCT and MRI of the ampulla of Vater. Part I: technique optimization, normal anatomy, and epithelial neoplasms. *Abdom. Imaging* 2015; **40**: 3274–91.
61 Dimastromatteo J, Brentnall T, Kelly KA. Imaging in pancreatic disease. *Nat. Rev. Gastroenterol. Hepatol.* 2017; **14**: 97–109.
62 Aguilera F, Tsamalaidze L, Raimondo M, Puri R, Asbun HJ, Stauffer JA. Pancreaticoduodenectomy and outcomes for groove pancreatitis. *Dig. Surg.* 2018; **35**: 475–81.
63 Vareedayah AA, Alkaade S, Taylor JR. Pancreatic adenocarcinoma. *Mo. Med.* 2018; **115**: 230–5.
64 Schawkat K, Manning MA, Glickman JN, Mortele KJ. Pancreatic ductal adenocarcinoma and its variants: pearls and perils. *Radiographics* 2020; **40**: 1219–39.
65 Cloyd JM, George E, Visser BC. Duodenal adenocarcinoma: advances in diagnosis and surgical management. *World J. Gastrointest. Surg.* 2016; **8**: 212–21.
66 Ushiku T, Arnason T, Fukayama M, Lauwers GY. Extra-ampullary duodenal adenocarcinoma. *Am. J. Surg. Pathol.* 2014; **38**: 1484–93.
67 Nishida T, Blay JY, Hirota S, Kitagawa Y, Kang YK. The standard diagnosis, treatment, and follow-up of gastrointestinal stromal tumors based on guidelines. *Gastric Cancer* 2016; **19**: 3–14.
68 Burch J, Ahmad I. Gastrointestinal stromal cancer. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing, 2023 [cited 2024 Jan 18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK554541/.
69 Rivera JA, Fernández-del Castillo C, Pins M, Compton CC, Lewandrowski KB, Rattner DW *et al*. Pancreatic mucinous ductal ectasia and intraductal papillary neoplasms. A single malignant clinicopathologic entity. *Ann. Surg.* 1997; **225**: 637–46.
70 Agostini-Vulaj D. Intraductal papillary mucinous neoplasm [Internet]. *Pathology Outlines* 2023 [cited 2024 Jan 18]. Available from: https://www.pathologyoutlines.com/topic/pancreasipmn.html.
71 Gapp J, Tariq A, Chandra S. Acute pancreatitis. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing, 2023 [cited 2024 Jan 18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK482468/.
72 Singh VK, Bollen TL, Wu BU *et al*. An assessment of the severity of interstitial pancreatitis. *Clin. Gastroenterol. Hepatol. Off. Clin. Pract. J. Am. Gastroenterol. Assoc.* 2011; **9**: 1098–103.
73 Puckett Y, Sharma B, Kasi A. Intraductal papillary mucinous cancer of the pancreas. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing, 2023 [cited 2024 Jan 18]. Available from:.
74 Shah I, Sheth SG, Kothari DJ. Pain management in chronic pancreatitis incorporating safe opioid practices: challenge accepted. *World J. Gastroenterol.* 2021; **27**: 3142–7.
75 de Parades V, Roulot D, Palazzo L, Chaussade S, Mingaud P, Rautureau J, Coste T. Treatment with octreotide of stenosing cystic dystrophy on heterotopic pancreas of the duodenal wall. *Gastroenterol. Clin. Biol.* 1996; **20**: 601–4.
76 Kapural L, Cywinski JB, Sparks DA. Spinal cord stimulation for visceral pain from chronic pancreatitis. *Neuromodulation J. Int. Neuromodulation Soc.* 2011; **14**: 423–6 discussion 426–427.
77 Sasahira N, Tada M, Isayama H *et al*. Outcomes after clearance of pancreatic stones with or without pancreatic stenting. *J. Gastroenterol.* 2007; **42**: 63–9.
78 Fujimori N, Igarashi H, Asou A *et al*. Endoscopic approach through the minor papilla for the management of pancreatic diseases. *World J. Gastrointest. Endosc.* 2013; **5**: 81–8.
79 Yamamoto N, Isayama H, Sasahira N *et al*. Endoscopic minor papilla balloon dilation for the treatment of symptomatic pancreas divisum. *Pancreas* 2014; **43**: 927–30.
80 Chantarojanasiri T, Isayama H, Nakai Y *et al*. Groove pancreatitis: endoscopic treatment via the minor papilla and duct of Santorini morphology. *Gut Liver* 2017; **12**: 208–13.
81 Patriti A, Castellani D, Partenzi A, Carlani M, Casciola L. Pancreatic adenocarcinoma in paraduodenal pancreatitis: a note of caution for conservative treatments. *Updates Surg.* 2012; **64**: 307–9.
82 Arvanitakis M, Rigaux J, Toussaint E *et al*. Endotherapy for paraduodenal pancreatitis: a large retrospective case series. *Endoscopy* 2014; **46**: 580–7.
83 Sachdev AH, Gress FG. Celiac plexus block and neurolysis: a review. *Gastrointest. Endosc. Clin. N. Am.* 2018; **28**: 579–86.
84 Rahman SH, Verbeke CS, Gomez D, McMahon MJ, Menon KV. Pancreatico-duodenectomy for complicated groove pancreatitis. *HPB* 2007; **9**: 229–34.
85 Tezuka K, Makino T, Hirai I, Kimura W. Groove pancreatitis. *Dig. Surg.* 2010; **27**: 149–52.
86 Egorov V, Petrov R, Schegolev A *et al*. Pancreas-preserving duodenal resections vs pancreatoduodenectomy for groove pancreatitis. Should we revisit treatment algorithm for groove pancreatitis? *World J. Gastrointest. Surg.* 2021; **13**: 30–49.
87 Rebours V, Lévy P, Vullierme MP *et al*. Clinical and morphological features of duodenal cystic dystrophy in heterotopic pancreas. *Am. J. Gastroenterol.* 2007; **102**: 871–9.
88 Zhu C, Huang Q, Zhu J, Zhang X, Qin X. Groove resection of pancreatic head in groove pancreatitis: a case report. *Exp. Ther. Med.* 2017; **14**: 1983–8.
89 Issa Y, Kempeneers MA, Bruno MJ *et al*. Effect of early surgery vs endoscopy-first approach on pain in patients with chronic pancreatitis: the ESCAPE Randomized Clinical Trial. *JAMA* 2020; **323**: 237–47.
90 Cahen DL, Gouma DJ, Laramée P *et al*. Long-term outcomes of endoscopic vs surgical drainage of the pancreatic duct in patients with chronic pancreatitis. *Gastroenterology* 2011; **141**: 1690–5.
91 Cahen DL, Gouma DJ, Nio Y *et al*. Endoscopic versus surgical drainage of the pancreatic duct in chronic pancreatitis. *N. Engl. J. Med.* 2007; **356**: 676–84.
92 Gorris M, Hoogenboom SA, Wallace MB, van Hooft JE. Artificial intelligence for the management of pancreatic diseases. *Dig. Endosc.* 2021; **33**: 231–41.
93 Ahmed TM, Kawamoto S, Hruban RH, Fishman EK, Soyer P, Chu LC. A primer on artificial intelligence in pancreatic imaging. *Diagn. Interv. Imaging* 2023; **104**: 435–47.
94 Gillies RJ, Kinahan PE, Hricak H. Radiomics: images are more than pictures, they are data radiology. *Radiology* 2016; **278**: 563–77.
95 Low DJ, Hong Z, Lee JH. Artificial intelligence implementation in pancreaticobiliary endoscopy. *Expert Rev. Gastroenterol. Hepatol.* 2022; **16**: 493–8.