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Dual-phase CT findings of groove pancreatitis[☆]

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Abstract

Purpose—Groove pancreatitis is a rare focal form of chronic pancreatitis that occurs in the pancreaticoduodenal groove between the major and minor papillae, duodenum and pancreatic head. Radiologic appearance and clinical presentation can result in suspicion of malignancy rendering pancreaticoduodenectomy inevitable. This study reports dual phase CT findings in a series of 12 patients with pathology proven groove pancreatitis.

Materials and methods—Retrospective review of preoperative CT findings in 12 patients with histologically proven groove pancreatitis after pancreaticoduodenectomy. Size, location, attenuation, presence of mass or cystic components in the pancreas, groove and duodenum, calcifications, duodenal stenosis and ductal changes were recorded. Clinical data, laboratory values, endoscopic ultrasonographic and histopathological findings were collected.

Results—Soft tissue thickening in the groove was seen in all patients. Pancreatic head, groove and duodenum were all involved in 75% patients. A discrete lesion in the pancreatic head was seen in half of the patients, most of which appeared hypodense on both arterial and venous phases. Cystic changes in pancreatic head were seen in 75% patients. Duodenal involvement was seen in 92% patients including wall thickening and cyst formation. The main pancreatic duct was dilated in 7 patients, with an abrupt cut off in 3 and a smooth tapering stricture in 4. Five patients had evidence of chronic pancreatitis with parenchymal calcifications.

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Conflict of interest

None.

Conclusion—Presence of mass or soft tissue thickening in the groove with cystic duodenal thickening is highly suggestive of groove pancreatitis. Recognizing common radiological features may help in diagnosis and reduce suspicion of malignancy.

Keywords

Pancreatitis; Chronic pancreatitis; Multidetector computed tomography; Duodenal disease

1. Introduction

Groove pancreatitis (GP, also known as paraampullary duodenal wall cyst) is an uncommon but distinctive form of segmental chronic pancreatitis affecting the pancreaticoduodenal (PD) groove region between the head of the pancreas, the duodenum and the major and minor papillae [1]. This entity remains unknown to most clinicians due to its relative obscurity and uncommon occurrence. Histopathologic features of GP were seen in 30 patients in a series of 123 surgical resections from patients with chronic pancreatitis [1]. Clinical distinction from other forms of chronic pancreatitis and pancreatic adenocarcinoma may be difficult due to the non-specific findings of abdominal pain, nausea and vomiting, jaundice, and weight loss [2,3]. On imaging, the focal nature of this pathology makes the distinction from pancreatic carcinoma difficult [4] and these patients may be subjected to pancreaticoduodenectomy [5]. The histologic findings of GP include a cyst-forming fibroinflammatory mass lesion in PD groove. Brunner gland hyperplasia is often seen in the duodenal wall [6]. The patients often report a history of chronic alcohol abuse.

With improvements in the imaging technique over the past decade, multidetector computed tomography (MDCT) has become the test of choice for the evaluation of abdominal pain including suspected diseases of pancreatic origin. A few studies in the recent literature have evaluated CT findings of GP but have a relatively small number of patients [7–9]. Dual phase MDCT obtained either in the arterial and venous phases or pancreatic and venous phases has become the modality of choice at many institutions for the evaluation of pancreatic pathology especially for the diagnosis, staging and exclusion of pancreatic cancer. Furthermore, pre-operative imaging evaluation of patients requiring pancreaticoduodenectomy as definitive surgery for severe pancreatic insufficiency, weight loss, or intractable abdominal pain may be required.

We sought to evaluate the clinicopathologic and imaging features of pathology proven GP in patients using dual-phase MDCT in an effort to find the frequency of common findings associated with this disease.

2. Materials and methods

2.1. Patient selection

This retrospective study was approved by our Institutional Review Board with waiver of informed consent and Health Insurance Portability and Accountability Act authorization. A retrospective search of the hospital database was conducted for patients undergoing pancreaticoduodenectomy between January 2002 and August 2013, resulting in a total of 12 patients with pathology proven GP. This group of patients formed the study population and

was composed of 10 males and 2 females, with mean age of 51.4 years (median: 47.5 years, range: 41–72 years). The indications for surgery included chronic abdominal pain, suspected carcinoma of the pancreatic head, and duodenal obstruction with suspicion of a mass lesion. All available clinical history was documented, along with laboratory values including amylase, lipase, liver function tests, and when available, tumor markers (CA19-9 and CEA) obtained closest to the day of the CT. Results for endoscopic ultrasound (EUS), if available, were reviewed, as well as for fine needle aspiration (FNA), if performed. Post-surgical findings and details of clinical follow-up were also examined.

2.2. Imaging technique and interpretation

Patients were imaged on a range of helical multidetector (4, 16, 64 and 128 slice) on one of the four CT scanners over the course of 11 years. The scanners were manufactured by Siemens Healthcare (Mavern, PA) and included: Somatom Plus 4 Volume Zoom scanner, Somatom Sensation 16, Somatom Sensation 64, and Somatom Definition Flash dual-source. All dual phase (arterial and portal venous) pancreatic imaging protocol (kVp 100–120, mAs 100–500) MDCTs included an intravenous bolus injection of between 100 and 140 mL of iodinated contrast (Visipaque® 320 or Omnipaque® 350, GE Healthcare, Waukesha WI, U.S.A.) by means of an automated power injector and 750–1000 mL water serving as negative oral contrast. The injection rates were routinely 4–5 mL/s with scan delays of 25–30 s (arterial phase) and 60 s (portal venous phase). Most CTs included both thin (0.75–1.5 mm) and thick (3–5 mm) image series, sagittal and coronal reformatted image series, and 3D post processed images.

All patients included had a contrast enhanced dual phase CT performed within three months prior to the pancreaticoduodenectomy. All CT examinations were reviewed on high quality picture archiving and communication systems (PACS) monitors by two board-certified and fellowship-trained radiologists with 12 and 5 years of CT interpretation experience as attending radiologists respectively. A checklist of 9 questions based on findings previously reported in the radiology literature was provided to the radiologists [7,8,10]. Images were reviewed independently and any discrepancies were resolved by consensus. Parameters collected include: size, location of the mass lesion (pancreatic head, PD groove and/or duodenum). The thickness of the pancreatic groove abnormality between the pancreatic head and the medial duodenal wall was measured on the coronal reformatted images. When present, a 1 cm region of interest (ROI) was drawn on the pancreatic lesion and attenuation in arterial and venous phase with respect to the attenuation of the apparently normal pancreatic parenchyma in the tail of the pancreas was recorded on the axial images. Presence of cysts or calcifications in the pancreas, main pancreatic ductal dilatation (defined as present if any portion of the pancreatic duct is ≥ 4 mm), parenchymal atrophy, number of enlarged peripancreatic lymph nodes, and inflammatory changes of the surrounding gland were noted. Presence of biliary dilatation (common bile duct [CBD] measuring >7 mm), and thickening of duodenal walls with luminal narrowing or any visible cystic changes were also recorded.

3. Results

3.1. Clinical findings

The majority of patients presented with abdominal pain ($n = 10$) and weight loss ($n = 8$). Half of the patients had considerable nausea and vomiting and three patients had clinical signs of gastric outlet obstruction. Two patients had suspicion for peptic ulcer disease as part of initial presentation. Two patients also presented with jaundice and dark urine and pruritus. None of the patients had a history of steatorrhea. Five patients had history of type 2 diabetes mellitus including 1 patient with recent onset diabetes. Smoking, alcohol abuse, and history of recurrent pancreatitis were seen in six, five and seven patients respectively (Table 1), and 3 patients had a family history of pancreatic cancer in first degree relatives. The symptom duration ranged between 1 week and 6 months.

Serum amylase, lipase, liver function tests, CEA, CA 19-9 levels within a few days of the CT are listed in Table 2.

3.2. Dual-phase MDCT findings

3.2.1. Pancreas and PD groove findings—Most patients had disease involving the pancreatic head, adjacent duodenal wall, and the PD groove area. A discrete mass lesion with ill-defined borders was seen in half ($n = 6$) of the patients within the pancreatic head mostly demonstrating both solid and cystic components with size ranging from 11 mm to 51 mm. The solid portion of the observed mass lesion was hypodense in both arterial and venous phases (Fig. 1) in all except one patient where it appeared isodense to the adjacent pancreatic parenchyma in both arterial and venous phase (Fig. 3b). Cystic changes in the pancreatic head were seen in 9 (75%) patients and the number of cysts varied between 1 and 7, ranging in size from 6 to 27 mm, without evidence of intramural nodules (Fig. 2). When involved, the pancreatic head appeared heterogeneous compared to the pancreatic parenchyma in the body and tail region. Calcifications were seen in four (33%) patients within the affected region of the pancreatic head, while one patient had diffuse parenchymal calcifications. The presence of calcifications matched the history of alcohol abuse in four of the five patients. Diffuse atrophy of the pancreatic parenchyma was seen in only 3 patients.

The main pancreatic duct was dilated in 8 patients (average diameter 5 mm; range 2–13 mm). The dilatation was diffuse, affecting the head, body and tail of the pancreas in all except one in which there was sparing of the pancreatic tail region. The pancreatic duct in the region of the pancreatic head had an abrupt cut off in three patients (Fig. 3a), while four patients had a smooth tapering stricture. Four (33%) patients had associated dilation of the CBD (maximum 18 mm) with smooth tapering of the distal portion (Fig. 3c), while three had biliary stents in place (Fig. 1a and b).

Minimal peripancreatic inflammatory changes with fat stranding were limited to the area surrounding the pancreatic head in all patients with normal appearing pancreatic tail (Fig. 4). Mildly prominent peripancreatic lymph nodes were seen in 11 (92%) patients with lymph nodes ranging from 2 to 10 in number and size ranging from 3 to 12 mm in short dimension.

Abnormality in the PD groove was seen as either an area of ill-defined soft tissue thickening or a crescent shaped mass like area widening the distance between the pancreatic head and the medial duodenal wall (Figs. 3b and 5). The mean thickening of this area was 9 mm (range 4–20 mm). This area appeared hypodense on both arterial and venous phase on CT.

3.2.2. Duodenal findings—Duodenal involvement was present in 11 (92%) patients characterized by the presence of luminal narrowing due to wall thickening. The medial duodenal wall was involved in 9 patients (75%) (Fig. 6) and circumferential thickening was seen in two (17%) patients (Fig. 7). The mean wall thickening was 13 mm (range 8–22 mm). Nine of the 11 (82%) patients with duodenal narrowing also showed cysts within the duodenal wall, ranging between 1 and 3 in number, the largest reaching a size of 39 mm (Fig. 8). Imaging findings are summarized in Table 3.

3.3. EUS findings

Of the 9 endoscopic ultrasounds performed, two had limited examinations due to duodenal stenosis and distortion, thus inability to insert scope sufficiently. Four EUS reported presence of a hypoechoic mass in the pancreatic head of which only two patients had a corresponding mass seen on dual phase MDCT. Three EUS reported the presence of cysts in the pancreatic head. Duodenal wall thickening was seen only in three (1 of these 3 was the only case that showed no duodenal wall thickening on CT). Five EUS reported the presence of pancreatic duct dilation, maximum of 6.7 mm, of which one was irregular and beaded. Only two EUS reported parenchymal calcifications.

Among the 9 EUS, seven had fine needle aspirations performed. Four patients showed presence of inflammatory changes, fat necrosis and benign ductal epithelium. Gastric mucosa heterotopia and gastric mucin cell metaplasia were seen in one patient without evidence of malignancy. One patient had cytological atypia, raising the possibility of malignancy, and one had a possible mucinous neoplasm.

3.4. Histopathology

All patients underwent surgery for either suspicion of malignant mass, duodenal obstruction, chronic pancreatitis, pain, refractory pseudocyst, or possible annular pancreas (negative upon surgery in a single patient). All patients were diagnosed with GP with concurrent chronic pancreatitis on histopathology. The diagnostic features of GP, including a localized fibroinflammatory mass in the “groove region,” bounded by the pancreas, duodenum and the major and minor papillae, were present in all cases. In almost all cases a single or multiple cysts were present at the center of the lesion. Small concretions were often present in the pancreatic duct system in the region of the GP, and these concretions were often associated with epithelial erosions. The duodenal wall was often extremely edematous. Brunner gland hyperplasia was prominent. The remainder of the pancreatic parenchyma had changes of chronic pancreatitis including acinar dropout, fibrosis, and atrophy with fat necrosis. None of the patients had an adenocarcinoma, however, pancreatic intraepithelial neoplasia (PanIN) was incidentally seen in 5 patients (PanIN-2 in 4 patients, PanIN-1B in 1 patient). In two patients where the abnormality on CT was restricted to the duodenum and the PD

groove, histopathology showed abnormality in the pancreatic head, groove and the duodenum.

3.5. Clinical follow-up

Follow-ups for the patients ranged between 1 and 30 months (mean: 10 months). Regarding acute complications, two patients experienced post-operative chyle leak. One of these two patients also suffered from recurrent hyperglycemia, delirium, fever, pleural effusion, and wound infection, during the same hospital stay. The other patient developed a *Clostridium difficile* infection one month later. The right hepatic artery was injured and repaired intra-operatively in one patient; this patient developed severe nausea and acute kidney injury while admitted. Only two patients among the 12 reported no relief of pain post-surgery followed up at 1 and 3 months respectively. One patient had a clinical history indicative of narcotic analgesics dependence, but stated the pain to have significantly decreased at a follow-up visit 3 months later. This patient also developed candidal esophagitis 3 weeks after surgery. One patient developed mild acute pancreatitis 3 months post surgery along with hepatic steatosis. One patient was admitted with diabetic ketoacidosis and acute pancreatitis 4 months later. Two patients experienced mild steatorrhea 2–4 months after surgery. Only one other patient later reported 4–5 bouts of abdominal pain that lasted a few seconds each, one month after their pancreatoduodenectomy. One patient died during the same hospitalization 10 days post-op due to cardiac arrest and hemorrhage.

4. Discussion

GP is a form of segmental chronic pancreatitis involving either the PD groove, the so called “pure” form, or with the involvement of the pancreatic head, known as the “segmental” form [11]. The term “paraampullary duodenal wall cyst” has also been used as a unifying terminology for GP. Other terms such as cystic dystrophy of the pancreas, paraduodenal pancreatitis, paraduodenal wall cyst, pancreatic hamartoma of duodenum and myoadenomatosis are used due to their common clinicopathologic features [6]. The proposed mechanisms for development of GP is disruption of the pancreatic secretion through the minor papilla due to a disturbed Santorini duct which may be due to chronic alcohol consumption or pancreatic heterotopia within the duodenum or the minor papilla with leakage of digestive enzymes into the PD groove and subsequent inflammation [12,13]. Intraductal concretions and calcifications, as can be seen in patients with chronic alcohol abuse, may contribute to the obstructions of the small ducts around the minor ampulla and thereby promote the disease process.

Most of our patients were men who presented in the fourth and fifth decade with symptoms of long standing abdominal pain, associated weight loss and occasional jaundice. Half of them had a known history of prior episodes of acute pancreatitis possibly associated with alcohol abuse [14]. Blood tests showed abnormal pancreatic enzymes and occasionally deranged liver function tests. The falsely elevated Carbohydrate Antigen 19-9 (CA 19-9) seen in a few patients may be attributed to obstructive jaundice [15]. EUS guided fine needle aspirations were mostly non-diagnostic, showing inflammatory changes or raising the possibility of a neoplasm.

The most characteristic finding on CT was the presence of a hypodensity located in the PD groove resulting in the widening of the distance between the pancreas and the duodenum. This area appeared hypodense on both phases and the appearance ranged from a mass like area to peripancreatic stranding around the head. This abnormality, previously been described as an area of “sheet-like” curvilinear and crescentic morphology [16] representing chronic inflammatory and fibrotic changes, was seen in all patients. The hypoenhancement on the arterial and venous phases on dual phase MDCT is similar to pancreatic cancer [9,17] when it arises in the PD groove, or similar to the focal pancreatitis that is sometimes seen in association with a mass obstructing the pancreatic duct. Pancreatic ductal dilatation, a common feature of pancreatic adenocarcinoma is usually not a feature of GP. Additionally, peripancreatic vessel encasement, a feature of locally advanced pancreatic adenocarcinoma, is not seen in GP.

In our study, the PD groove findings were associated with abnormalities of either the pancreatic head or the duodenum, making it the so-called “segmental” form of GP [11]. The “pure” form is less frequent and was seen in only 2% cases in a series of 600 surgically resected cases of CP [11]. Most of the cases in our series had involvement of the pancreas, duodenum and the PD groove with sparing of the pancreatic head seen in only 2 cases on CT but with changes of CP seen on histopathology. Abnormalities restricted to the PD groove and duodenum have also been described and in one series were seen in more than 50% of patients [17]. Most of our patients demonstrated heterogeneity of the pancreatic head with 50% patients showing a discrete hypoenhancing mass lesion in the pancreatic head raising suspicion for an underlying adenocarcinoma. This also included a single patient with abnormality restricted to the pancreas and the PD groove with a normal appearing duodenum on CT. In general, pancreatic adenocarcinoma is usually more homogeneously hypodense compared to the patchy heterogeneity seen in GP [7]. However, this distinction may not always be possible as was the case in our patients.

The other common finding on CT was the presence of duodenal involvement characterized by eccentric wall thickening involving the medial duodenal wall leading to luminal narrowing, and the presence of cysts in the duodenal wall. A high incidence of these findings have been previously described in patients with GP [8,18]. These changes in the duodenum seen on CT correspond to Brunner gland hyperplasia, edema, spindle cell proliferation and duodenal wall cysts on histological examination of the duodenum. Duodenal wall thickening has been attributed to Brunner gland hyperplasia linked to chronic alcohol consumption but may also be attributed to biliary and peptic ulcer disease [1]. Self-reported history of alcohol abuse was seen in half of our study population, and this may represent an under estimation of the true prevalence of alcohol abuse in this population. On imaging a key differentiating feature between GP and cancer is the duodenal wall thickening and cystic changes, a finding extremely uncommon in pancreatic adenocarcinoma. These features have been used to accurately differentiate GP from pancreatic cancer and can be easily identified even by inexperienced radiologists [19]. Other differential considerations such as duodenal adenocarcinoma and ampullary cancer may also be sometimes difficult to differentiate from GP. However, pancreatic changes usually do not accompany the primary lesions in these entities.

The heterogeneous appearance of the pancreatic head seen on both the arterial and venous phase is likely secondary to poor enhancement caused by delayed circulation caused by fibrosis and edema related to chronic inflammation [9]. Furthermore, presence of calcifications, a specific sign of chronic pancreatitis was observed in 5 patients, 4 of which had focal calcifications in the pancreatic head. Chronic pancreatitis with inspissated secretions is often seen histologically in the pancreas [6,20]. Dilatation of the main pancreatic duct seen in 8 patients was likely secondary to the presence of a stricture from chronic inflammation. Abrupt cut-off of the pancreatic duct in the region of the pancreatic head, seen in three patients, is typically seen in the presence of pancreatic adenocarcinoma making the differentiation of the two entities difficult. The CBD strictures seen in the patients were smooth and tapering indicative of an inflammatory etiology. Furthermore, cystic changes seen in the pancreatic head may be mistaken for intrapancreatic mucinous neoplasm (IPMN) which are pre-cancerous lesions leading to surgery in a significant number of patients because of an inability to reliably make the differentiation on imaging [1,18,21]. Although the cystic lesions seen in the pancreatic head in our patient group represented chronic pseudocysts associated with chronic pancreatitis, chronic pancreatitis itself may be a risk factor for IPMN and subsequent development of pancreatic cancer [22].

Due to improvement in imaging techniques such as MDCT and MRI/MRCP, the number of pancreaticoduodenectomies performed on benign lesions for the suspicion of malignancy has decreased [5]. Although the prospective diagnosis of this entity may be difficult especially when a neoplasm cannot be effectively excluded, the characteristic findings, when observed, may strongly suggest the diagnosis of GP leading to conservative therapy in poor surgical candidates. Avoiding surgery, when clinically appropriate, may be particularly important in this patient population considering the numerous complications they can develop after surgery and these patients may be closely monitored clinically and by imaging. Analgesics with smoking cessation and alcohol abstinence may help in the management of patients suffering an acute attack [5]. Medical therapy including the use of somatostatin and minimally invasive treatments such as endoscopic drainage of the minor papilla have shown good short term results [23–25]. Furthermore, these therapies may provide a bridge treatment before definitive surgery with either a classic or pylorus-sparing pancreaticoduodenectomy performed for chronic abdominal pain, weight loss and severe pancreatic insufficiency. Improvement in abdominal pain was seen in most patients after surgical intervention in our study. However, the mean follow up in our data set is only 10 months ranging between 1 and 30 months. Although most post-operative complications were non-fatal, mortality in a single patient due to cardiovascular risk factors was observed, highlighting the need for reliable imaging and laboratory diagnostic tools for an accurate prospective diagnosis.

In conclusion, the diagnosis of GP may be difficult on MDCT and exclusion of malignancy such as pancreatic adenocarcinoma, ampullary and duodenal carcinomas is important. However, certain features seen on MDCT such as sheet like hypodensity in the PD groove, medial duodenal wall thickening and cystic changes in the duodenal wall along with evidence of chronic pancreatitis in the pancreatic head are highly suggestive of GP. Although, a significant number of these patients may require definitive surgery for symptom

relief, recognition of these findings may be helpful in reducing the number of pancreaticoduodenectomies performed for mere suspicion of cancer.

References

1. Stolte M, Weiss W, Volkholz H, Rosch W. A special form of segmental pancreatitis: "groove pancreatitis". *Hepatogastroenterology*. 1982; 29(5):198–208. [PubMed: 7173808]
2. Shudo R, Yazaki Y, Sakurai S, et al. Groove pancreatitis: report of a case and review of the clinical and radiologic features of groove pancreatitis reported in Japan. *Intern Med*. 2002; 41(7):537–42. [PubMed: 12132521]
3. Levenick JM, Gordon SR, Sutton JE, Suriawinata A, Gardner TB. A comprehensive, case-based review of groove pancreatitis. *Pancreas*. 2009; 38(6):e169–75. [PubMed: 19629001]
4. Irie H, Honda H, Kuroiwa T, et al. MRI of groove pancreatitis. *J Comput Assist Tomogr*. 1998; 22(4):651–5. [PubMed: 9676462]
5. Casetti L, Bassi C, Salvia R, et al. Paraduodenal pancreatitis: results of surgery on 58 consecutive patients from a single institution. *World J Surg*. 2009; 33(12):2664–9. [PubMed: 19809849]
6. Adsay NV, Zamboni G. Paraduodenal pancreatitis: a clinicopathologically distinct entity unifying "cystic dystrophy of heterotopic pancreas", "paraduodenal wall cyst", and "groove pancreatitis". *Semin Diagn Pathol*. 2004; 21(4):247–54. [PubMed: 16273943]
7. 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(3):e95–100. [PubMed: 19450943]
8. Rahman SH, Verbeke CS, Gomez D, McMahon MJ, Menon KV. Pancreaticoduodenectomy for complicated groove pancreatitis. *HPB*. 2007; 9(3):229–34. [PubMed: 18333228]
9. 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(6):911–5. [PubMed: 7962798]
10. Manzelli A, Petrou A, Lazzaro A, Brennan N, Soonawalla Z, Friend P. Groove pancreatitis. A mini-series report and review of the literature. *JOP*. 2011; 12(3):230–3. [PubMed: 21546697]
11. Becker V, Mischke U. Groove pancreatitis. *Int J Pancreatol*. 1991; 10(3–4):173–82. [PubMed: 1787332]
12. Chatelain D, Vibert E, Yzet T, et al. Groove pancreatitis and pancreatic heterotopia in the minor duodenal papilla. *Pancreas*. 2005; 30(4):e92–5. [PubMed: 15841034]
13. Barsotti P, Schloegel M, Ollier JC, Ghnassia JP, Potet F, Adloff M. Cystic dystrophy on an aberrant duodenal pancreas. Apropos of a case. *J Chir*. 1995; 132(12):503–5.
14. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013; 144(6):1252–61. [PubMed: 23622135]
15. Ballehaninna UK, Chamberlain RS. The clinical utility of serum CA 19-9 in the diagnosis, prognosis and management of pancreatic adenocarcinoma: an evidence based appraisal. *J Gastrointest Oncol*. 2012; 3(2):105–19. [PubMed: 22811878]
16. Kwak SW, Kim S, Lee JW, et al. Evaluation of unusual causes of pancreatitis: role of cross-sectional imaging. *Eur J Radiol*. 2009; 71(2):296–312. [PubMed: 18538971]
17. Gabata T, Kadoya M, Terayama N, Sanada J, Kobayashi S, Matsui O. Groove pancreatic carcinomas: radiological and pathological findings. *Eur Radiol*. 2003; 13(7):1679–84. [PubMed: 12835985]
18. Vullierme MP, Vilgrain V, Flejou JF, et al. Cystic dystrophy of the duodenal wall in the heterotopic pancreas: radiopathological correlations. *J Comput Assist Tomogr*. 2000; 24(4):635–43. [PubMed: 10966201]
19. Kalb B, Martin DR, Sarmiento JM, et al. Paraduodenal pancreatitis: clinical performance of MR imaging in distinguishing from carcinoma. *Radiology*. 2013; 269(2):475–81. [PubMed: 23847255]
20. Kloppel G. Chronic pancreatitis, pseudotumors and other tumor-like lesions. *Mod Pathol*. 2007; 20(Suppl 1):S113–31. [PubMed: 17486047]

21. Kim JD, Han YS, Choi DL. Characteristic clinical and pathologic features for preoperative diagnosed groove pancreatitis. *J Korean Surg Soc.* 2011; 80(5):342–7. [PubMed: 22066058]
22. Capurso G, Boccia S, Salvia R, et al. Risk factors for intraductal papillary mucinous neoplasm (IPMN) of the pancreas: a multicentre case-control study. *Am J Gastroenterol.* 2013; 108(6):1003–9. [PubMed: 23458848]
23. Arisaka Y, Tada H, Hongoh Y, et al. A case of groove pancreatitis successfully treated with conservative therapy. *Nihon Shokakibyo Gakkai Zasshi.* 1998; 95(10):1157–61. [PubMed: 9805936]
24. de Parades V, Roulot D, Palazzo L, et al. Treatment with octreotide of stenosing cystic dystrophy on heterotopic pancreas of the duodenal wall. *Gastroenterol Clin Biol.* 1996; 20(6–7):601–4. [PubMed: 8881576]
25. Triantopoulou C, Dervenis C, Giannakou N, Papailiou J, Prassopoulos P. Groove pancreatitis: a diagnostic challenge. *Eur Radiol.* 2009; 19(7):1736–43. [PubMed: 19238393]

Fig. 1. 62-year-old man with painless jaundice and dark urine. Dual-phase CECT demonstrates a 3.4 cm hypoenhancing mass seen on both arterial (a) and venous phase (b) imaging (thick arrow). A CBD stent is also in place (thin arrow in a). The imaging findings were highly suggestive of cancer and the patient underwent pancreaticoduodenectomy. Pathology revealed findings of groove pancreatitis including fat necrosis and fibrous proliferation in the pancreatic head.

Fig. 2. 72-year-old man with abdominal pain and weight loss. Venous phase coronal reformat CT demonstrates cystic changes in the pancreatic head (thick arrows). Chronic pancreatitis is evident from multiple foci of calcifications (thin arrow). Pancreaticoduodenectomy demonstrated changes of groove pancreatitis including chronic pancreatitis in the head with cystic dilation of the pancreatic ducts.

Fig. 3.

41 year-old man with jaundice. Axial venous phase CT (a) demonstrates dilated pancreatic duct measuring 13 mm (thick white arrow) with abrupt cut-off along with intra- and extra hepatic biliary duct dilatation (thin arrows). Axial venous phase CT (b) demonstrates a mass like area which is isodense to the pancreatic parenchyma (thin arrows) in the pancreatic head. In addition, ill-defined hypodense soft tissue thickening in the PD groove widening the distance between the pancreatic head and the medial duodenal wall is also seen (thick arrow). Venous phase coronal reformat CT (c) shows smooth tapering stricture of the CBD (thin arrow) and intrahepatic biliary ductal dilatation (thick arrows).

Pancreaticoduodenectomy demonstrated changes of groove pancreatitis including chronic pancreatitis in the head region with extensive fibrosis.

Fig. 4.

50-year-old man with abdominal pain and weight loss. Arterial phase coronal curved reformat CT demonstrates a heterogeneous appearance of the pancreatic head with cystic changes and peripancreatic stranding (thick arrow) and normal appearing pancreatic body (thin arrow). Pancreaticoduodenectomy demonstrated histopathological changes of groove pancreatitis including acute on chronic pancreatitis with acinar drop out and fibrosis.

Fig. 5. 45-year-old woman with abdominal pain, weight loss and gastric outlet obstruction. Coronal venous phase CT demonstrates hypodense mass like area measuring 20 mm in the PD groove. Histology after pancreaticoduodenectomy showed changes of groove pancreatitis including inflammatory changes in the PD groove.

Fig. 6. 50-year-old man with abdominal pain and weight loss. Arterial phase coronal reformat CT image after the administration of positive oral contrast demonstrates eccentric duodenal wall thickening (arrows) giving the appearance of a mass lesion. Pancreaticoduodenectomy demonstrated changes of groove pancreatitis including chronic inflammation and Brunner gland hyperplasia of the duodenal wall.

Fig. 7. 45-year-old man with recurrent pancreatitis. Venous phase coronal reformat CT demonstrates circumferential duodenal wall thickening (arrows) giving the appearance of an annular duodenal carcinoma. Histopathology post pancreaticoduodenectomy revealed groove pancreatitis with inflammatory changes in the duodenal wall including submucosal abscess formation and reactive spindle cell proliferation.

Fig. 8.

59-year-old man with chronic pancreatitis with multiple risk factors including PRSS1 gene mutation, smoking and a strong family history of chronic pancreatitis. Venous phase coronal reformat CT demonstrates a 30 mm cyst in the duodenal wall (thick arrow). Additionally a cyst is also seen in the pancreatic head (thin arrow). Pancreaticoduodenectomy demonstrated histopathological changes of groove pancreatitis including chronic pancreatitis in the pancreatic head with extensive fibrosis as well as inflammatory changes in the duodenal wall with Brunner gland hyperplasia.

Table 1

Clinical data of the 12 patients including demographics and clinical history.

Patient number	Sex	Age	Abdominal pain	Weight loss	GOO	N/V	PUD	Diabetes	Jaundice	History of pancreatitis	Alcohol	Smoking
1	M	72	Yes	Yes	No	Yes	No	Yes	No	No	Yes	No
2	M	45	Yes	Yes	Yes	Yes	Yes	No	No	Yes	Yes	No
3	F	62	Yes	Yes	No	Yes	No	Yes	No	No	No	Yes
4	M	46	No	Yes	No	No	No	Yes	No	No	Yes	Yes
5	F	45	Yes	Yes	Yes	No	Yes	No	No	Yes	Yes	Yes
6	M	45	Yes	No	No	No	No	No	No	Yes	No	No
7	M	62	No	No	No	No	No	No	Yes	No	No	No
8	M	41	Yes	No	No	No	No	Yes (new onset)	No	No	No	No
9	M	49	Yes	Yes	No	Yes	No	No	No	Yes	No	Yes
10	M	41	Yes	Yes	No	Yes	No	Yes	Yes	Yes	Yes	Yes
11	M	50	Yes	Yes	No	No	No	No	No	Yes	No	Yes
12	M	59	Yes	No	No	Yes	No	No	No	Yes	No	No

GOO – gastric outlet obstruction; N/V – nausea/vomiting; PUD – peptic ulcer disease.

Table 2
Laboratory values corresponding closest with time of CT for each of the 12 patients.

Patient no.	Amylase 28–100 (U/L)	Lipase 16–63 (U/L)	CEA 0–3.0 (ng/mL)	CA19-9 0–36 (U/mL)	ALT 0–40 (U/L)	AST 0–37 (U/L)	Bilirubin 0.1–1.2 (mg/dL)	AP 30–120 (U/L)
1	144		9.4	60.6				
2	382	105			37	61	1.4	162
3								
4				100.1	15	14	0.3	180
5				16.6	18	17	0.5	82
6								
7	193	509	2.6	34.8	29	25	0.8	110
8					33	16	1	58
9	103	60		<1.0	14	29	0.5	99
10	111	227		334	52	33	1.7	942
11	65	81			72	11		
12	173	130	72.7	11.3	13	6	0.4	75

CEA – carcinoembryonic antigen; CA19-9 – carbohydrate antigen 19-9; ALT – Alanine aminotransferase; AST – aspartate aminotransferase; AP: alkaline phosphatase.

Table 3

Imaging characteristics of the 12 patients on MDCCT.

Patient no.	Involvement	Discrete lesion in pancreatic head	Enhancement in lesion		Pancreatic head cysts	PD groove soft tissue thickness (mm)	Duodenal wall thickening	Duodenal cyst (s)
			Arterial phase	Venous phase				
1	P + G + D	No	-	-	Yes	7	Eccentric	Yes
2	P + G + D	No	-	-	Yes	11	Eccentric	Yes
3	P + G + D	No	-	-	Yes	6	Eccentric	Yes
4	P + G + D	No	-	-	Yes	4	Eccentric	No
5	G + D	Yes	Hypodense	Hypodense	Yes	20	Eccentric	Yes
6	G + D	No	-	-	No	6	Circumferential	No
7	P + G	Yes	Hypodense	Hypodense	No	10	None	No
8	P + G + D	Yes	Hypodense	Hypodense	Yes	16	Eccentric	Yes
9	P + G + D	Yes	Hypodense	Hypodense	Yes	10	Circumferential	Yes
10	P + G + D	Yes	Isodense	Isodense	No	8	Eccentric	Yes
11	P + G + D	Yes	Hypodense	Hypodense	Yes	7	Eccentric	Yes
12	P + G + D	No	-	-	Yes	5	Eccentric	Yes

Involvement: P – pancreatic head; G – groove area; D – duodenal wall.