The aim of this retrospective study was to highlight the imaging findings of groove pancreatitis (GP) as well as its management.

**Patients and methods:** 16 patients diagnosed to have GP were enrolled in this work. The included patients had complete records of the thorough clinical examination and laboratory workup. All patients had been examined by multi-phase contrast enhanced MDCT tailored for pancreatic imaging. Six of these patients were additionally examined by MRI including MRCP.

**Results:** MDCT Multiple detector computed tomography of the 16 patients revealed the following: (1) a hypodense sheet in the pancreaticoduodenal (PD) groove seen in 12 patients with mild enhancement in the delayed phase seen in 6 of the them; (2) Duodenal wall thickening was seen in 10 patients while (3) associated cysts within the duodenal wall or in PD groove were seen in 6 patients; (4) and pancreatic head enlargement in 8 patients. MRI of Six patients revealed the following: (1) a T1 hypointense and T2 iso to hyperintense sheet at the PD groove in 4 patients with delayed enhancement in 3 of them; (2) Duodenal wall thickening with T2 high signal was seen in 6 patients while associated cysts within the duodenal wall were seen in 4 patients; (3) Pancreatic head enlargement seen in 4 patients; The MRCP of these patients showed dilated CBD with distal tapering and a distance separating its end from the duodenal wall.

**Conclusion:** GP is a disease that should be considered in the list of differential diagnosis of masses implicating the pancreatic head and medial duodenal wall. Imaging findings that are suggestive of GP include chronic inflammatory changes with fibrosis in the PD groove with or without implication of the nearby head of the pancreas, duodenal medial mural thickening with luminal stenosis and cysts at the PD groove or within the duodenal wall. Vascular invasion is a sign against diagnosis of GP.

**Keywords:** Groove pancreatitis, Pancreatic cancer, MRCP
duodenum superiorly. This potential space contains lymph nodes, portion of the common bile duct, distal main pancreatic duct, distal accessory pancreatic duct as well as the major minor papilla. Small vessels are passing within this space, and the most important of these is the superior PD artery [1].

Groove pancreatitis (GP) is a specific type of pancreatitis originating in the PD groove. Sometime it involves the nearby head of the pancreas, second part of the duodenum and the common bile duct [2,3].

Several different terms have been used to describe this inflammatory processes centered in the PD groove, including groove pancreatitis, periampullary duodenal wall cyst, myoadenomatosis, and cystic dystrophy of the duodenal wall. These disorders have clinically been grouped together and are termed “paraduodenal pancreatitis” [4]. Paraduodenal pancreatitis was first described by Becker in 1973 then Stolte et al. [2] introduced the term “groove pancreatitis” for the same condition which is more used in the literature.

The incidence of GP was reported in three different series of pancreaticoduodenectomy in patients with chronic pancreatitis, which was different. The percentage of occurrence was 2.7%, 19.5% and 24.5% in the three different series respectively [2,5].

Patients affected by GP are usually adult males with alcohol abuse while GP has been described in females sporadically [6].

2. Aim of the work

The purpose of this study was to describe the imaging findings of GP as well as its management.

2.1. Patients

16 patients with a diagnosis of GP were enrolled in this study. They included 12 males and 4 females, and their ages ranged between 35 and 73 years with the mean age of 58 years. Inclusion criteria include those patients with the final diagnosis of GP while those with the final diagnosis of pancreatic groove carcinoma, peptic ulcer disease, duodenal cancer, ampullary cancer or pancreatic head cancer were excluded.

2.2. Methods

This study is a retrospective study where patient consent was waived by the Research Ethics Board, assuring respect of the confidentiality of the medical record. We have reviewed our medical records for the diagnosis of GP during the period between January 2011 and October 2015.

The included patients had complete records of the thorough clinical examination, laboratory workup including routine laboratory work as well as lipase, amylase CEA and CA19.9. All patients had been examined by multiphase contrast enhanced MDCT tailored for pancreatic imaging including non-contrast, pancreatic, portal and delayed phases. The machine used was Toshiba Aquilion 128-MDCT unit kV/effective mAs/rotation time (s):120 kV/225 eff. mAs/0.35 s; slice thickness 0.5 mm.

Non-ionic IV contrast was injected with a dose of 1.5 ml/kg (maximum = 150 ml), with average rate of 4 ml/s using automatic pump injector.

The pancreatic phase timing was fixed at 45 s, portal phase at 70 s, and delayed phase after 5 min from the start of contrast injection respectively, and examination was done using Siemens Emotion 6 and 64 MSCT. Scanning parameters were as follows: Volumetric High-spatial-frequency kernel algorithm; Slice thickness: 1–1.25 mm; Table speed for volumetric HRCT to enable the least cycles of breath-holds as possible; Tube rotation: 0.6–0.9 s (mean 0.75 s); Detector Collimation 1 mm; Helical mode (volumetric HRCT); and kVp and mA per slice: 120–130 kVp and 200–400 mA, according to weight of the patient and clinical indication.

Six of these patients were additionally examined by MRI including MRCP, using a 1.5 T closed MRI imager (Avanto, Siemens, Erlangen, Germany). The pulse sequences used were transverse T2FSE with and without fat saturation, T1 chemical shift sequences (In/opposed phase), Dynamic pre- and post Gadolinium Volumetric Interpolated Breath-Hold Examination (VIBE) sequences, and MRCP sequences (thin slice 3D, as well as thick slab single shot).

Scanning parameters are as follows:

- Localizing T1-W gradient echo sequences were used.
- Axial 2D T2-W turbo spin echo (HASTE/TSE) fat suppression sequence from the level of lower chest to mid abdomen level as finishing the whole liver span. TR1600, TE 70, flip angle 90, FOV 375, slice thickness 7 mm, NSA 1 total scan time averaging 43 s.
- Axial 2D T2-W turbo spin echo (HASTE/TSE) fat suppression sequence with longer TE = 190. It was important to diagnose the degree of signal intensity of the lesion in long TE.
- In-phase and opposed phase (IP/OP) sequence TR 500, TE in-phase (2.2), TE opposed phase (4.4) FOV 375, flip angle 80, slice thickness 7 mm, NSA 2, average scanning time 48 s. It was important in diagnosis of lesions containing intracellular fat as well as in diagnosis of focal or patchy fatty infiltration or sparing.
- SSFP (BFSE in Philips) TR500, TE60, flip angle 60, FOV255, slice thickness 7 mm, NSA 1 with average scan time 22 s.
- DWI with variable b values 50–1000 s/mm2 with TR 1000, TE 137, flip angle 90, FOV 370, slice thickness 10 mm and average scan time 1.15 min. and automatically computer-generated ADC map.
- 3D T1 spoiled gradient fat-suppressed sequence with TR 50, TE 500, FOV 355, flip angle 10, slice thickness 3 mm with average scan time 19 s and this is repeated in triphasic study as HAP (15 s after the end of contrast injection), PVP (70 s from the end of contrast injection) and delayed phase (about 3 min from the end of contrast injection).

MRCP examinations were obtained with a single-shot, heavy T2W FSE sequence, HASTE (Siemens) by using respi-
ratory gating and fat saturation which allows very rapid image acquisition during brief breath hold and even it is so fast that allows imaging in patients who cannot hold breath efficiently. This sequence includes the following:

- Thin section multisections moderate T2W FSE sequence: A TR of 1600 ms, an echo time (TE) of 290 ms, a slice thickness = 2 mm, FOV = 380, average = 2.0, flip angle, 140°, matrix size = 412 × 576 and turbo factor = 109.
- Thin section multissections heavy T2W FSE sequence: A TR of 2900 ms, an echo time (TE) of 700 ms, a slice thickness = 1 mm, FOV = 380, matrix size = 357 × 384 average = 2.0, flip angle, 140°, and turbo factor = 135.
- A two dimensional thick single slab projectional image: A repetition time (TR) of 4500 ms, an echo time (TE) of 700 ms, a slice thickness = 40 mm, distant factor = 50%, field of view (FOV) = 350 mm, matrix size = 307 × 384, average = 1.0, flip angle, 180° and turbo factor = 256. The MIP images generated from the entire volume of a thin section multissection data set resemble ERCP images or thick slab images.

The management of patients with GP was either conservative in 6 patients or surgical with Whipple procedure in 10 patients. Satisfactory improvement of the symptoms was noted in 4 out of 6 with conservative management and 7 patients with surgical management. The diagnosis was confirmed by histopathology in 12 patients (10 surgical specimens and 2 FNAC), while clinical improvement and follow-up CT confirmed the diagnosis in the other 4 patients.

The MRI and CT scans were analyzed to highlight imaging features of groove pancreatitis.

3. Results

3.1. Clinical picture (Table 1)

The patients presented with variable clinical pictures including epigastric pain in 10 patients, obstructive jaundice in 8 patients, vomiting in 8 patients, weight loss in 6 patients, and diarrhea in 4 patients. Some of the patients presented with more than one of above. Mild elevation of amylase and lipase was reported in 8 and 6 patients respectively while elevated indirect bilirubin was reported in 8 patients. None of the patients showed significant rise of the CEA, CA19.9 or other tumor markers.

3.2. Multiple findings were seen on the MDCT examinations of the 16 patients (Table 2) including

(1) A hypodense sheet at the PD groove was seen in 12 patients with modest enhancement identified in delayed phase seen in 6 of them. (2) Duodenal wall thickening was seen in 10 patients while associated cysts within the duodenal wall or in PD groove were seen in 6 patients. (3) Pancreatic head enlargement with diffuse enhancement was seen in 8 patients. (4) Mild pancreatic duct dilatation was seen in 8 patients while dilatation of the CBD was seen in 10 patients with distal tapering and intra-hepatic biliary dilatation. (5) None of the patients showed peri-pancreatic fluid collections, vascular invasion or occlusion, ascites, locoregional suspicious nodes or other stigmata of intra abdominal metastatic disease. None of our cases showed distended GB (Figs. 1–3).

3.3. Six patients out of the above described 16 patients had MRI as well (Table 2), showing the following

(1) A T1 low signal and T2 iso to hyperintense signal sheet at the PD groove was seen in 4 patients with delayed enhancement in 3 of them. (2) Duodenal wall thickening with T2 high signal was seen in 6 patients while associated cysts of T2 fluid signal within the duodenal wall were seen in 4 patients. (3) Pancreatic head enlargement with low T1 signal alteration was seen in 4 patients. (4) Mild pancreatic duct dilatation was seen in 4 patients with pancreatic body and tail atrophy in 2 of them while dilatation of the CBD was seen in 4 patients with distal tapering and intra-hepatic biliary dilatation. (5) None of the patients showed peri-pancreatic fluid collections, ascites, loco-regional suspicious nodes or other stigmata of intra abdominal metastatic disease (Figs. 2 and 4).

The MRCP of these patients showed dilated CBD with distal tapering and a distance separating its end from the duodenal wall in addition to fluid filled cysts at the duodenal wall (Figs. 2 and 4) seen in 4 patients while the other 2 patients had almost unremarkable MRCP.

4. Discussion

GP has two types: pure type or form that affects exclusively the PD groove with sparing of the pancreatic head and segmental type which is epicentered in groove with extension medially into the head of the pancreas. The differentiation between these two forms is not usually clear [7,2].

In our study pure form of the disease was seen in CT examinations in 8 cases (50%) presenting with, the classic findings of a hypo-dense sheet like soft tissue density within the PD groove. Four of these showed retained contrast represented by enhancement in the delayed phase representing fibrous tissue. This delayed enhancement is explained by hindered blood flow caused by fibrous tissue growth impeding the arterial flow due to arterial constrictions [8].

The GP segmental type was seen in the other 8 patients (50%) where the sheet like focal hypodense lesions

<table>
<thead>
<tr>
<th>Clinical picture</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epigastric pain referred to the back</td>
<td>10</td>
</tr>
<tr>
<td>Vomiting</td>
<td>8</td>
</tr>
<tr>
<td>Jaundice</td>
<td>8</td>
</tr>
<tr>
<td>Weight loss</td>
<td>6</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>4</td>
</tr>
</tbody>
</table>

NB some patients may have more than one of the above.
extended into the pancreatic head in vicinity of the duodenal wall with pancreatic head enlargement.

Similar findings were reported in previous studies with emphasis on the coronal reconstructions of MDCT data that can allow better identification of ill-defined fat stranding and inflammatory changes in the PD groove accompanied with increasing delayed enhancement as a result of a significant fibrotic component [9].

The pure form is rather easy to identify. On the other hand, the segmental form can be difficult to diagnose, because involvement of the groove is often obscured by mass like involvement of the pancreatic head. The segmental type of GP is confused for a pancreatic head mass, and differentiating the two entities is not easy on the MRI and CT [10,11].

The main pancreatic ducts showed mild dilatation in seen at the body and tail of the pancreas in the 8 cases with segmental type, while in the pure form of GP the main pancreatic ducts were not dilated. It is also reported that pancreatic duct can also be narrowed toward the head of the pancreas in a smooth gradual pattern. In more chronic stage, pancreatic parenchymal changes resembling those of ordinary chronic pancreatitis can develop including pancreatic calcifications, ductal dilatation, and ductal beading or irregularity [9,11].

Other important findings were also noted in our series including focal duodenal wall thickening seen in 10 cases (62.5%) and cysts in the duodenal wall itself or in groove between the pancreatic head and the duodenum in 6 cases (37.5%). The cysts were variable in size and number ranging from tiny to large even multi-locular cystic mass like lesion was seen in a single case. Appreciating medial duodenal wall thickening is easier also on the coronal images [8].

Other authors found that these multiple cysts suggest cystic dystrophy in heterotopic pancreatic islands within the duodenal wall. They also claimed that heterotopic pancreatic tissues could not be identified on CT until they got inflamed with cystic changes [12].

The pathology of GP in the literature addressed that the duodenal mucosa between the major and minor papillae is markedly thickened. The involved areas show gelatinous contents, edema and fibrosis with possible cyst formation. The cysts may contain small calculi. Microscopic evaluation reveals duodenal wall thickening, with glandular and muscular inflammation and hyperplasia. Sometimes pancreatic islands heterotopia can be seen. The scarring implicates the lower portion of the common bile duct in the PD groove [13–16].

The origin of the cysts in GP is controversial, and the most popular theory claims that they are cystic dystrophy of the pancreatic heterotopic islands in the duodenal wall [17]. Other authors suggested that these cysts may be dilated Santorini duct branches [18].

The cause of GP is still controversial including coexisting biliary disorder, gastric ulcers, and heterotopic pancreatic tissues or disturbed pancreatic fluid flow through the duct of Santorini [2,5]. The specific location of the lesions around the minor papilla suggests possible anatomical or functional disorder related to this area as GP may occur in cases with pancreas divisum, absent or narrow duct of Santorini [19], or may be due to obstruction of the accessory pancreatic duct [17,19].

An important point noticed in our series and reported previously, is that even in severe GP, the surrounding ves-
sels are spared without thrombosis or infiltration [8,10,20].
Also in our series we have not detected CT signs of acute
pancreatitis or loco regional metastasis or adenopathies.
The previous studies reported rarity to visualize fluid in
the para-renal spaces or surrounding the pancreas [10,11].

In our study CBD dilatation and distal smooth tapering
were seen in 10 patients (62.5%) including all the segmen-
tal types and 2 of the pure type leading to intra- and extra-
hepatic biliary system dilatation.

Previous studies also found that distal common bile
duct can appear attenuated and narrowed in both pure
and segmental types of GP. This was better assessed on
the coronal multiplanar reconstruction (MPR).

In most cases, this narrowing was relatively smooth,
tapered, and regular, without evidence of shouldering or
irregularity [9,11].

As a fact, CT provides superior spatial resolution, but
with less contrast resolution to discriminate pancreatic
cancer from inflammation. The high soft-tissue resolution
of MRI provides more accurate evaluation of the pancreatic
tissues, specifically for tissue characterization in inflamma-
tory and neoplastic processes and analysis of contents of
cysts [21–24].

MRI and MRCP were available in 6 patients in our study.
There was a CT similarity regarding the sheet of tissues
within the pancreaticoduodenal groove. These were seen

Fig. 2. Multiphase MDCT Axial (A and B) in late arterial phase, showing ill defined sheet of hypodensity at the duodeno-pancreatic groove with partial
extension into the pancreatic head. The corresponding Axial T2 WI (C&D) done two weeks later showed similar sheet of tissues expressing mild T2
hyperintense signal as well as mild duodenal wall thickening and enlarged pancreatic head. Coronal single shot MIP (E), showing the intra and extra-hepatic
biliary dilatation with distal tapering of the CBD as well as a relatively gapping distance between its end and the 2nd part of the duodenum. Tiny cystic
changes in the duodenal wall seen (C&E). Segmental form of groove pancreatitis.
expressing T1 hypo-intense and T2 slightly hyperintense signal in 3 patients with depiction of mild enhancement in the delayed phases in three of them (50%). These cases represented the pure type of GP, while in the segmental form there was associated pancreatic head enlargement in the other 3 patients.

Involvement of the pancreas was reported to be well visualized on MRI compared to CT, with progressive loss of T1 signal intensity in the head of the pancreas as a result of parenchymal atrophy and fibrosis [25,26].

Irie et al. [7] and Blasblag et al. [26] also reported similar MRI findings in GP explaining the T2 iso to hyperintense signal variation of the lesion in the PD groove or in the head of the pancreas head to match with stage of the disease, as the subacute phase disease shows higher T2 signal due to edema, and chronic phase has darker T2 signal due to evolution of fibrosis.

Duodenal wall thickening was seen in the MRI of all the 6 patients with 4 of them showing mural cysts. The medial wall of duodenum is involved in the pure as well as the segmental forms of GP, with multiple T2 hyperintense cysts in both the duodenal wall and PD groove.

Blasblag et al. [26] described similar changes in their study. Also they described peripheral enhancement in the pancreatic and portal phases with progressive fill in the delayed phases. This delayed enhancement was reported also in the related thickened medial wall of the duodenum. This delayed enhancement reflects presence of fibrosis [25].

In our study pancreatic head enlargement with low T1 signal alteration was seen in 4 patients with pancreatic duct dilatation in addition to atrophy of the rest of pancreas. This reflects chronic inflammatory disease with fibrous tissues replacing the glandular tissues of the pancreas. In the pure type of GP, the pancreas appears normal and shows relatively high T1 signal [26].

In our study duodenal wall thickening with T2 high signal was seen in 6 patients while associated small cysts of T2 fluid signal within the duodenal wall were seen in 4 patients. MRCP facilitates determination of relationship between these cysts and CBD and pancreatic ducts [26].

Marked duodenal wall thickening is usually not associated with pancreatic neoplastic processes while it is common in GP [26]. This sign can help in differentiating GP from pancreatic cancers.

In our study duodenal wall thickening with T2 high signal was seen in 6 patients while associated small cysts of T2 fluid signal within the duodenal wall were seen in 4 patients. MRCP facilitates determination of relationship between these cysts and CBD and pancreatic ducts [26].

In our study MRCP was available in 6 patients, showing dilated CBD with distal tapering with a distance separating distal ends from the duodenal walls, fluid filled cysts at the duodenal wall or in the groove with ectatic pancreatic ducts in 4 patients, while the other 2 patients had almost unremarkable MRCP.

Table 2
Summary of the different positive and negative CT and MRI findings among the patients with GP.

<table>
<thead>
<tr>
<th>CT Finding</th>
<th>No. patients</th>
<th>%</th>
<th>MRI Finding</th>
<th>No. patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypodense sheet</td>
<td>12</td>
<td>75</td>
<td>Hypointense sheet</td>
<td>4</td>
<td>66.6</td>
</tr>
<tr>
<td>Duodenal wall thickening</td>
<td>10</td>
<td>62.5</td>
<td>Duodenal wall thickening with high T2 signal</td>
<td>6</td>
<td>100</td>
</tr>
<tr>
<td>CBD dilatation and distal tapering</td>
<td>10</td>
<td>62.5</td>
<td>CBD dilatation and distal tapering</td>
<td>4</td>
<td>66.6</td>
</tr>
<tr>
<td>Pancreatic head enlargement</td>
<td>8</td>
<td>50</td>
<td>Pancreatic head enlargement</td>
<td>3</td>
<td>50</td>
</tr>
<tr>
<td>Pancreatic duct dilatation</td>
<td>8</td>
<td>50</td>
<td>Pancreatic duct dilatation</td>
<td>4</td>
<td>66.6</td>
</tr>
<tr>
<td>Delayed enhancement</td>
<td>6</td>
<td>37.5</td>
<td>Delayed enhancement</td>
<td>3</td>
<td>50</td>
</tr>
<tr>
<td>Duodenal cysts</td>
<td>6</td>
<td>37.5</td>
<td>Duodenal cysts</td>
<td>4</td>
<td>66.6</td>
</tr>
<tr>
<td>Collections and stranded mesentery</td>
<td>0</td>
<td>0</td>
<td>Collections and stranded mesentery</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Locoregional lymph nodes</td>
<td>0</td>
<td>0</td>
<td>Locoregional lymph nodes</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Intra-abdominal metastasis</td>
<td>0</td>
<td>0</td>
<td>Intra-abdominal metastasis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Vascular invasion</td>
<td>0</td>
<td>0</td>
<td>Vascular invasion</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>GB distension</td>
<td>0</td>
<td>0</td>
<td>GB distension</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Blasblag et al. [26] described similar smooth distal tapering which was regular without shoulder sign or abrupt interruption of the ducts in cancers.

On MRCP, the distance separating distal pancreatic and CBD from duodenal lumen is due to inflammatory lesion in the PD groove and the duodenal wall marked thickening. This is common in GP and not in pancreatic neoplastic lesions [25,26].

The main pancreatic duct is usually not dilated in the pure form of GP, while the segmental form shows stricture within the pancreatic head that is usually longer than those associated with pancreatic neoplasms. Also GP showed milder upstream dilatation of the pancreatic ducts in the rest of the pancreatic body and tail [27].

None of our cases showed abnormal dilatation of the GB. The GB tends to be normally distended in GP. Previous studies described Banana like gallbladder in cases of GP simulating those seen in traditional chronic pancreatitis [26].

MRCP is valuable in diagnosis of GP as it yields diagnostic data more or less similar to ERCP. However, in GP, duodenal stenosis often hinders ERCP [25]. ERCP is limited to visualization of a tapered lower bile duct, which can sometimes be difficult to differentiate GP smooth long stricture from irregular strictures in malignancies [13].

The appearance of GP with both transabdominal and endoscopic ultrasound is not well detailed in the literature. In the early stages with more inflammatory component, U/S may show hypoechoic band like thickening of the PD groove and thickening of the adjacent duodenum with or without hypoechoic heterogeneous pancreatic head. In the chronic stages, the echogenicities of all these lesions become hyper as fibrosis dominates over inflammation [28]. It is common to visualize regular narrowing of the CBD and the Santorini duct on endoscopic ultrasound [29].

Fine needle biopsy even those guided by endoscopic US are challenging to pathologists. The relatively small sample volume may not be adequate to exclude the presence of malignant cells [3,16,30]. Even Fibrosis diagnosed by pathologists does not exclude neoplastic changes, as pancreatic adenocarcinoma may show desmoplastic reaction simulating the fibrotic changes in chronic inflammatory abnormality such as GP putting down the diagnostic merit of the Fine needle biopsy in such cases [31,32].

Similar to our results, previous studies reported limited value of laboratory markers in diagnosis of GP because
bilirubin, alkaline phosphatase, lipase and amylase are often normal or just minimally elevated. Pancreatic tumor markers (CEA and CA-19-9) are usually negative in GP. These negative biomarkers may suggest GP rather than neoplastic process [6,33].

The most challenging differential diagnosis of GP (especially its segmental form) is from pancreatic head adenocarcinoma and malignancies which arise adjacent to the PD groove and do not show the typical pancreatic double duct cutoff and upstream atrophy. The management plans of the GP and pancreatic cancer two are significantly different [6]. This differentiation can be impossible, and many patients may undergo Whipple procedure because of lack of this preoperative discrimination [5]. Even more this differentiation is important in optimizing therapeutic decisions, including the decision of whether or not to use preoperative chemotherapy if GP is excluded [34].

Unlike GP, most pancreatic adenocarcinomas do not show internal cystic change and are much more likely to infiltrate posteriorly into the retroperitoneum and encase the vasculature. Moreover, thickening of the medial duodenal wall, a common finding with GP, is uncommon with pancreatic adenocarcinoma. Enhancement pattern for GP tends to be more patchy and heterogeneous with delayed contrast retained while pancreatic adenocarcinoma usually shows more homogeneous hypodensity [25,26].

Kalb et al. [34] reported that contrast-enhanced MR imaging may help accurately differentiating GP from pancreatic cancer when using suggested 3 diagnostic signs for GP including mural duodenal thickening, delayed enhancement of the second part of the duodenum; and cysts seen within duodenal wall or PD groove. They found correct diagnosis of GP was achieved with accuracy of 87.2% while exclusion of cancer can had a negative predictive value of 92.9%.

The differentiation between GP and scirrhou adenocarcinoma invading the groove is difficult on CT and MR imaging. Both produce similar T1 low signal with or without delayed enhancement [35–37]. However, carcinomas often have more discrete and round configuration while fibrosis is more diffuse and ill defined [26]. The presence of vascular invasion is highly suggestive of pancreatic carcinoma and not reported in GP [38,35].

Furthermore, it is more difficult to differentiate between GP and carcinomas arising in the PD groove, because they may be associated with duodenal mural thickening and stenosis. Duodenal biopsies may correctly reach a diagnosis [35].

Differentiating GP from acute pancreatitis is rather easy due to fluid collections and inflammation in the PD groove that evolves rapidly on serial follow-up imaging and should usually resolve later on, whereas the imaging findings associated with GP often persist. Acute edematous pancreatitis, involves a large portion of the pancreatic parenchyma, and not only epicentered in the groove with peripancreatic fluid and inflammation tracking into the pararenal spaces, while GP typically shows little retroperitoneal inflammation or fluid, and even in the segmental form, involvement of the pancreas is usually limited to the pancreatic head. Elevated lipase level is also an important differentiating marker [39].

Differential diagnosis of GP from chronic pancreatitis with acute with pseudocysts within the duodenal wall is rather easy because the later has no mural duodenal thickening or luminal stenosis [40].

Pure form of GP should be differentiated from other conditions including duodenal cancer as well as distal CBD and ampullary carcinomas. The later produces focal malignant lesions at the ampulla, while GP is more ill-defined crescentic soft tissue, still larger ampullary carcinomas may not be easily differentiated from GP on imaging basis [40].

MRCP can help in differentiating GP from cholangiocarcinomas involving the CBD, since GP shows a longer smooth CBD stenosis or tapering compared to the irregular narrowing, shouldering and abrupt termination of the CBD in cholangiocarcinomas [40].

Carcinoids and gastrinomas may rarely originate within the PD groove. These tumors show early hyper-enhancement due to high vascularity and hyperintense T2 signal compared to the delayed centripetal enhancement of GP. Duodenal gastrointestinal stromal tumor (GIST) is more hypodense lesion and still more hypervascular and should not be easily confused with GP [11].

Acute phase of GP is conservatively treated using bed rest, analgesics and intra-venous nutrition. Most patients improve with this conservative treatment, but some patients suffer from relapses of acute pancreatitis especially those with an anatomic or functional disturbance of pancreatic duct system [41].

Sometimes GP resists medical treatment and surgical treatment may be inevitable at late stages of the disease as the patient may develop marked duodenal stenosis endocrine and exocrine pancreatic failure, or extensive fibrotic changes causing severe pain. Those patients usually benefit from surgery with symptomatic relief [42,43]. A pancreaticoduodenectomy using the Whipple procedure or less frequently a pylorus-preserving pancreaticoduodenectomy are the surgical procedures of choice in groove pancreatitis [6]. Both treatment plans were present in our cases with satisfactory improvement of the symptoms that were noted in 4 out of 6 with conservative management and 7 out of 10 patients with surgical management.

Another option was reported as a promising treatment for GP including ERCP drainage of the duct of Santorini [44].

Radiological suggestion of the suspicion or suggestion that a pancreatic head lesion may represent GP should direct the surgeons for further workup before the decision of radical surgery. On the other hand, the diagnosis of groove pancreatitis should not be confirmed until the other possibility of adenocarcinoma is carefully excluded.

Limitations of our study include retrospective design, and we limited the scope of our study to describe the imaging features of the documented cases of GP with lack of comparison with cases of pancreatic head cancer that may simulate GP. Another limitation is that not all cases have MRI and MRCP examinations and we did not provide comparison between CT and MRI.
5. Conclusion

GP is a disease that should be considered in the list of differential diagnosis of masses implicating the pancreatic head and medial duodenal wall. GP has two types: pure type that affects exclusively the PD groove and segmental type which is epicentered in groove with extension medi- ally into the head of the pancreas. Imaging findings that are suggestive of GP include chronic inflammatory changes with fibrosis in the PD groove with or without implication of the nearby head of the pancreas, duodenal medial mural thickening with luminal stenosis and cysts at the PD groove or within the duodenal wall. Vascular invasion is a sign against diagnosis of GP. Sometime the differentiation between the two forms of GP is not usually clear.

Conflict of interest

The authors declared that there are no conflict of interests.

References


