

# Groove Pancreatitis – A Mimic of Pancreatic and Periapillary Tumors

SIVAKAMI R PRADHEEPKUMAR, D. KARTHIKEYAN

## ABSTRACT

Groove Pancreatitis (GP) is a rare form of focal chronic pancreatitis involving the pancreatico-duodenal groove (PDG). GP was first described by Becker in 1973. Though, GP has been described so many years ago, it is still unfamiliar among most physicians because of lack of sufficient case studies and clinical similarity of GP to conventional pancreatitis. Imaging based differentiation of GP from other lesions, like pancreatic and periampullary adenocarcinoma is also not possible in all the cases, unless there are typical findings favoring GP. Since, the line of treatment and outcome is totally different in these two conditions, appreciation of the fine differences between

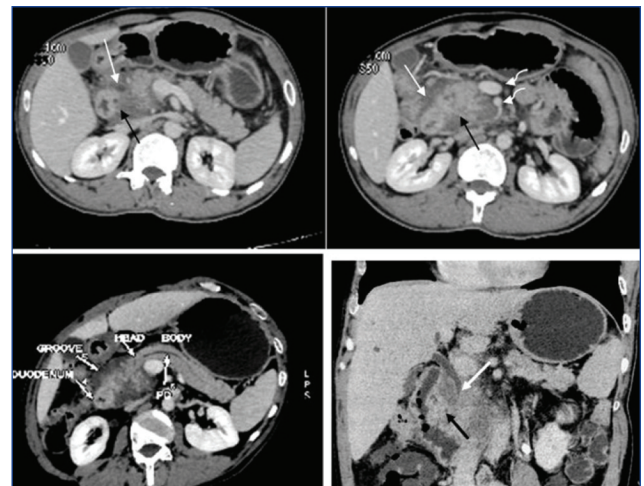
these two entities is very significant. Groove pancreatitis is symptomatically treated with medicines and only for patients with continuous and severe symptoms which are not amenable to medical treatment surgical management is considered. Radiological differentiation of GP from pancreatic and periampullary malignancies will help to avoid unnecessary surgery in the initial stages. We report two cases of GP, one of pure and other of segmental form where we found typical imaging features which pointed to the diagnosis of GP with a small discussion about the Computed tomography (CT) and Magnetic Resonance Imaging (MRI) appearance of this entity as well as its differential diagnosis.

**Keywords:** CT, Focal chronic pancreatitis, Pancreatic duodenal groove, MRI and Pancreatic head adenocarcinoma

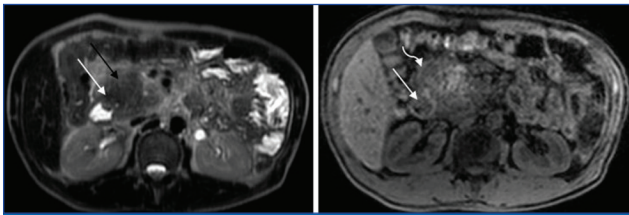
## CASE 1

A 45-year-old chronic alcoholic male presented with recurrent epigastric pain and loss of weight for past 3 months. Pancreatic enzymes, tumor markers (CA 19-9 and CEA) and liver tests were unremarkable. Patient was referred for CT-scan abdomen to rule out gastric and pancreatic pathologies. 128-slice contrast enhanced CT abdomen was done and showed bulky uncinete process & head of pancreas with scattered tiny cystic spaces within. Fibrous sheet like lesion seen in the PDG showing delayed enhancement with contrast (5 & 10 min post contrast). The lesion was seen displacing but not encasing superior mesenteric vessels to left side. Second part of the duodenum showed mildly thickened wall with few tiny submucosal cystic lesions. Proximal segmental prominence of main pancreatic duct (MPD) and smooth long segment distal CBD stricture seen [Table/Fig-1a-d]. An abdominal MRI was performed with T1 TSE, T2 SSH, m-Dixon and 3D MRCP sequences and showed a sheet-like mass in PDG appearing hypo to isointense on T1 and hypointense on T2-WIs suggesting the fibrotic nature of the mass. Duodenal wall thickening was found [Table/Fig-2a,2b].

Differential diagnosis considered in this case was conventional



**[Table/Fig-1]:** (a) - Hypodense mass in PD groove (white arrow) and thickened medial wall of second part of duodenum with tiny cyst (black arrow). (b)- Hypodense mass in PD groove (white arrow) and bulky uncinete process with cystic change (black arrow), displaced superior mesenteric artery and vein (cur'ed arrows). (c)- Hypodense mass in PD groove, bulky head of pancreas with cystic changes and proximal segmental prominence of MPD. (d)- Distal CBD smooth stricture (white arrow) and delayed enhancement of groove lesion.



**[Table/Fig-2]:** (a) - T2 WI showing thickened wall of second part of duodenum (white arrow) and hypointense mass in PDG (black arrow). (b)- T1 WI showing second part of duodenum (long arrow) and hypointense mass in PDG (curved arrow).

edematous pancreatitis with PDG involvement, segmental form of groove pancreatitis, pancreatic adenocarcinoma and duodenal and ampullary adenocarcinoma. In case of conventional edematous pancreatitis, pancreatic parenchymal involvement is not limited only to the head in all the cases as seen in our case. Presence of peripancreatic fluid tracking retroperitoneally along pararenal spaces is also common [1]. Serum lipase levels are usually elevated.

Imaging based differentiation of adenocarcinoma arising from pancreatic head just adjacent to the groove from GP is difficult, where the typical pancreatic duct cut-off and distal pancreatic atrophy is not present commonly, which was applicable in our case also. However, few features which helped to exclude possibility of adenocarcinoma was fibrotic nature of the PDG lesion suggested by MRI signal, patchy heterogeneous increasingly dense enhancement of the lesion with time, presence of duodenal wall thickening, cystic changes in the groove & duodenum and absence of retroperitoneal infiltration and vascular encasement. This was in contrast with imaging features of adenocarcinoma namely, homogeneous enhancement pattern, presence of retroperitoneal infiltration and vascular encasement and absence of cystic changes in the groove and duodenum [1-3].

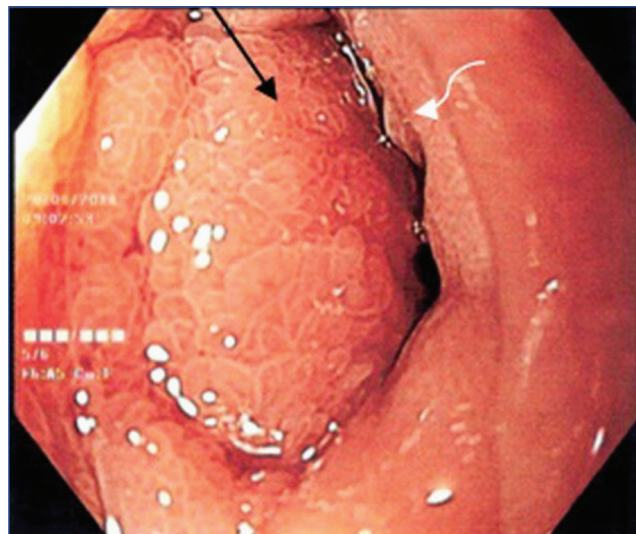
Possibility of duodenal and ampullary adenocarcinoma was excluded by exactly locating the lesion epicenter being the PDG and not the duodenum/ ampulla proper with the use of multiplanar imaging [1-3]. Hence, the final diagnosis made was the segmental form of groove pancreatitis, considering the typical location of the disease in PDG with involvement of the adjacent pancreatic tissue, thickened duodenal wall with cystic changes in duodenum as well as in PDG, sheet like fibrous intensity mass in PDG showing patchy heterogeneous enhancement increasing with time with no evidence of vascular or retroperitoneal enhancement [1-5].

## CASE 2

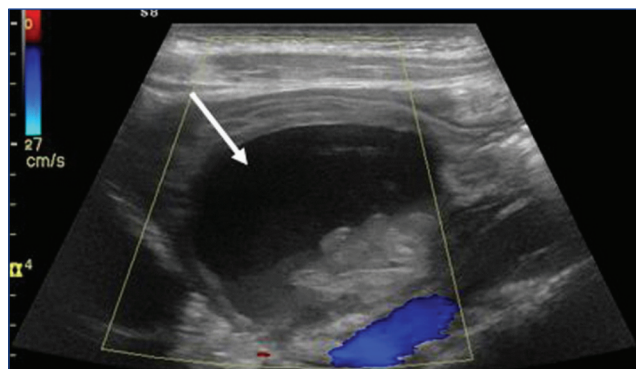
A 39-year-old chronic alcoholic male patient presented with history of recurrent epigastric pain, non bilious vomiting and weight loss for past one year. LFT, pancreatic enzymes and tumor markers were unremarkable. Upper GI endoscopy was

done and showed mass effect in second part of duodenum causing luminal stenosis with smooth overlying mucosa [Table/Fig-3], biopsy taken from the mass lesion which showed chronic nonspecific inflammation with Brunner gland hyperplasia. USG abdomen of the patient showed a thick walled cystic lesion in the PDG [Table/Fig-4]. Patient was further evaluated with 128 slice CECT abdomen showed a fibrous sheet like mass lesion in PDG showing mild contrast enhancement progressing on delayed phase with a large multiloculated thick walled cystic lesion with minimal internal debris. Mild cystic changes seen in second part of duodenum. Smooth stricture of distal CBD and mild dilatation of MPD are seen. Pancreas appeared normal. Right renal subcapsular collection causing mass effect over right kidney was seen probably representing chronic hematoma with secondary page kidney [Table/Fig-5a-5c]. On MRI multiloculated cystic lesion hypo to iso intense on T1 and hyperintense on T2 WIs seen in the PDG [Table/Fig-6a,6b].

With these findings the major differentials diagnosis considered

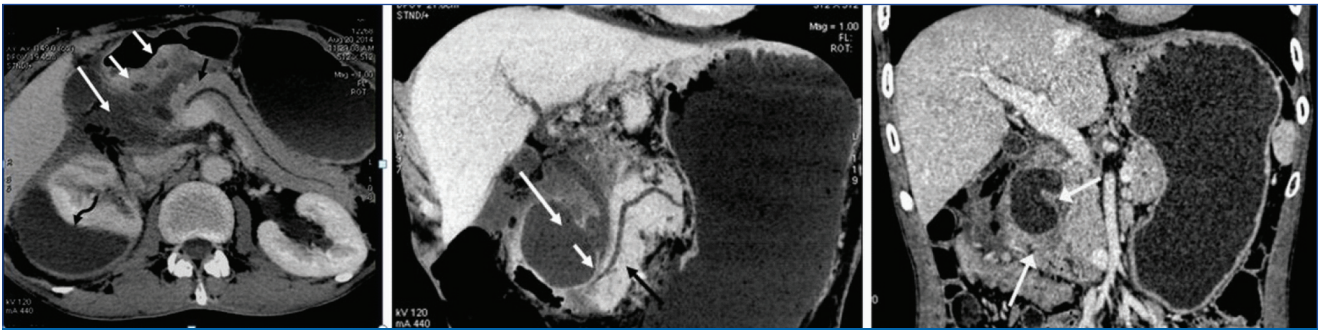


**[Table/Fig-3]:** Upper GI endoscopic image showing proximal duodenal luminal narrowing with smooth mucosal surface.

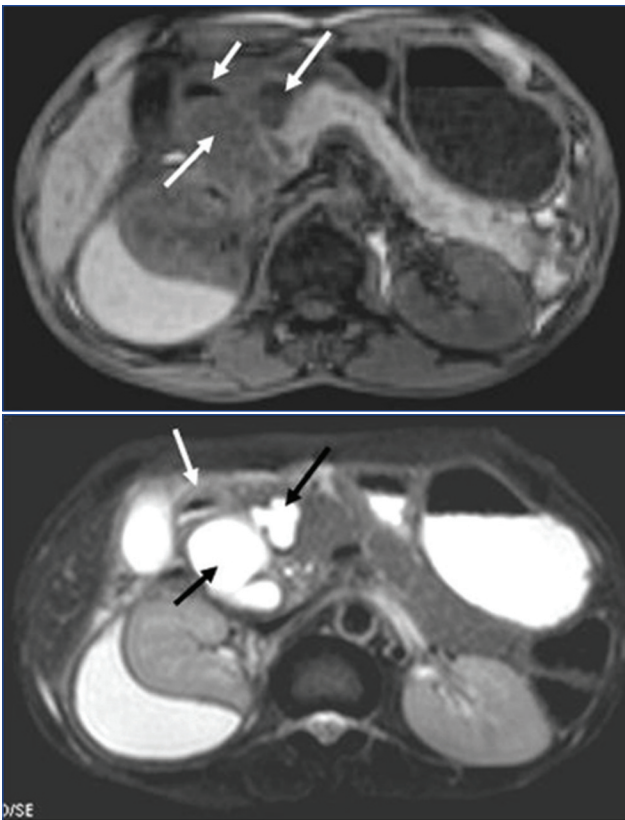


**[Table/Fig-4]:** High resolution USG abdomen showing thick-walled cystic lesion in PDG with settled debris (arrow).





**[Table/Fig-5a,b&c]:** (a) - Post contrast processed axial image showing hypodense lesion with cystic change in PDG (long white arrow), thickened duodenal wall with tiny cysts (short white arrows), mildly dilated MPD (black arrow) and right renal subcapsular collection (curved arrow). (b) - Post contrast processed coronal image showing hypodense lesion with cystic change in PDG (long white arrow), smooth stricture of distal CBD (short white arrow) and mildly dilated MPD (black arrow). (c) - Delayed phase coronal image showing increasing enhancement in the groove lesion.



**[Table/Fig-6a&b]:** (a) - T1W image showing multiloculated hypo to iso intense cystic lesion (long arrows) in the groove between duodenum second part (short arrow) and pancreas with. Pancreas appears normal. Right renal subcapsular hyperintense collection seen. (b) - T2W image showing multiloculated hyperintense cystic lesion (black arrows) in the groove between duodenum second part (white arrow) and pancreas. Pancreas appears normal. Right renal subcapsular hyperintense collection seen.

was pure form of groove pancreatitis, and neoplasms arising from second part of the duodenum or ampulla.

Possibility of duodenal and ampullary neoplasms like

adenocarcinoma, Gastrointestinal stromal tumors or neuro endocrine tumors arising from medial wall of the duodenum was excluded by exactly locating the lesion epicenter being the PDG and not the duodenum/ ampulla proper and by the absence of typical intense post contrast enhancement, seen in most of the GIST and neuro endocrine tumors of the duodenum [1-3]. It was supported by the upper GI endoscopy reports showing normal duodenal mucosa and histopathology being negative for duodenal /ampullary neoplastic pathologies. Hence, the final diagnosis made was the pure form of groove pancreatitis.

Both of the patients were managed with medical treatment including analgesics, abstinence from alcohol and somatostatins. Both patients showed gradual improvement of clinical symptoms in three weeks time. Later patients were advised to take analgesics on as on when required basis, to continue abstinence from alcohol and somatostatins and were called for follow-up after a month. Orally informed consent taken from the patients for publications of the case reports in future.

## DISCUSSION

GP is a distinct form of focal chronic pancreatitis involving the PDG. PDG is an anatomic space bounded medially by the pancreatic head, laterally by second part of the duodenum, posteriorly by third part of the duodenum and inferior vena cava, and superiorly by duodenal bulb. The space is occupied by distal end of common bile duct, main pancreatic duct, major & minor papilla, multiple small arteries and veins and multiple lymph nodes [6].

GP is of two types, the "segmental" and the "pure". Segmental type involves both the groove and the head of pancreas. Pure type involves only the groove [7]. The incidence of this disease is not well known due to lack of enough case studies and clinical similarity of GP to conventional pancreatitis [4]. The pathogenesis is not yet clearly defined. Acid peptic disease

[2], obstruction of Santorini duct, heterotopic pancreatic tissue and abnormal minor papilla [4] has been suggested as possible etiologies. Alcohol abuse is considered to be a precipitating factor. Majority of the GP patients are in 30-50 years of age with history of alcoholism [4], usually presenting with upper abdominal pain and recurrent vomiting which is secondary to the duodenal stenosis. Lab test show variable pancreatic enzymes levels and normal tumor markers [5]. Most of the patients are managed conservatively with analgesics and abstinence from alcohol consumption and is effective in initial stage [3]. Surgery is reserved only for patients with severe clinical symptoms not amenable to medical treatment or to rule out malignancy when imaging and or histopathology cannot confirm the diagnosis.

### Imaging Features

**On USG:** Hypoechoic mass lesion in PDG, thickened duodenal wall with cystic changes with or without luminal stenosis.

**On CT-scan and MRI:** Sheet-like, hypo attenuating (CT), T1 hypointense and T2 variable intense sheet like mass lesion in the groove, showing poor patchy progressive enhancement on venous and delayed phases. Thickened duodenal wall with cystic change and smooth stricture of distal CBD [1-5].

On histopathology, common feature of GP is scarring and fibrosis in the PDG in pure form and both the groove and pancreatic head in segmental form [2,4] Chronic inflammatory changes with fibrosis, Brunner's glandular hyperplasia in the duodenum with cystic changes in the wall are seen which are believed by many authors to be representing heterotopic pancreas in the duodenal wall with cystic dystrophic changes [4].

## CONCLUSION

On imaging groove pancreatitis is a close mimic of pancreatic and periampullary adenocarcinoma and the line of management significantly vary in these two entities. Hence, familiarity regarding the typical imaging features of groove pancreatitis and fine differentiating features of this condition from its mimics is essential for each radiologist to avoid unnecessary surgery in the initial stages. Here we report two cases of GP, one of pure and other of segmental form where we found typical imaging features which pointed to the diagnosis of GP.

## REFERENCES

- [1] Raman S, Salaria S, Hruban R, Fishman E. Groove pancreatitis: spectrum of imaging findings and radiology-pathology correlation. *AJR Am J Roentgenol.* 2013;201(1):W29-39.
- [2] Triantopoulou C, Dervenis C, Giannakou N, Papailiou J, Prassopoulos P. Groove pancreatitis: a diagnostic challenge. *Eur Radiol.* 2009;19(7):1736-43.
- [3] Ishigami K, Tajima T, Nishie A, Kakihara D, Fujita N, Asayama Y et al. Differential diagnosis of groove pancreatic carcinomas vs. groove pancreatitis: Usefulness of the portal venous phase. *European Journal of Radiology.* 2010;74(3):e95-e100.
- [4] Chatelain D, Vibert E, Yzet T, Geslin G, Bartoli E, Manaouil D, et al. Groove pancreatitis and pancreatic heterotopia in the minor duodenal papilla. *Pancreas.* 2005;30(4):e92-e95.
- [5] Shanbhogue A, Fasih N, Surabhi V, Doherty G, Shanbhogue D, Sethi S. A clinical and radiologic review of uncommon types and causes of pancreatitis. *Radio Graphics.* 2009;29(4):1003-26.
- [6] Hernandez-Jover D, Pernas J, Gonzalez-Ceballos S, Lupu I, Monill J, Pérez C. pancreatoduodenal junction: review of anatomy and pathologic conditions. *J Gastrointest Surg.* 2011;15(7):1269-81.
- [7] Levenick J, Gordon S, Sutton J, Suriawinata A, Gardner T. A comprehensive, case-based review of groove pancreatitis. *Pancreas.* 2009;38(6):e169-75.

### AUTHOR(S):

1. Dr. Sivakami R PradheepKumar
2. Dr. D. Karthikeyan

### PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Radio Diagnosis, Sri Manakulavinayagar Medical College and Hospital, Kalitheerthal Kuppam, Madagadipet, Puducherry, India.
2. Senior Consultant and HOD, Department of Radio Diagnosis, SRM Institute for Medical Science and Imaging, Jawaharlal Nehru Salai, Vadapalani, Chennai, Tamil Nadu, India.

### NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sivakami R PradheepKumar,  
Sri Manakulavinayagar Medical College and Hospital,  
Kalitheerthal Kuppam, Madagadipet,  
Puducherry- 605107, India.  
E-mail: sivakamij@yahoo.co.in

### FINANCIAL OR OTHER COMPETING INTERESTS:

None.

Date of Online Ahead of Print: **Oct 28, 2016**

Date of Publishing: **Oct 01, 2017**