Case Report

Groove Pancreatitis: Report of One Case in Thailand

Somchai Meesiri MD*

** Department of Surgery, Srisangwornsukhothai Hospital, Sukhothai, Thailand*

Groove pancreatitis is a rare form of chronic pancreatitis affecting the head of the pancreas localized within the pancreatoduodenal groove. Fibrous scar in this specific topography sometimes makes it hard to differentiate from pancreatic cancer preoperatively. The author reports the case of a 44-year-old man with a long history of alcoholic abuse and experienced intermittent epigastric and nausea vomiting for 2 years. Abdominal ultrasound showed an irregular mixed echogenic mass at the pancreatic head. A computed tomography revealed a poorly enhanced solid mass with small low density cystic areas in the groove, thickening and luminal narrowing of the descending part of the duodenum. Magnetic resonance imaging demonstrated the same mass that was hypointense on T1 weighted images, isointense on T2 weighted images and delayed, progressive inhomogeneous enhancement on dynamic contrast study. MRCP defined a prominent smooth tapering of the common bile duct. Endoscopy disclosed an inflammed sessile mass at the second part of the duodenum. Microscopic examination of the biopsy specimens suggested only chronic inflammation. Then, the patient was treated conservatively and evaluated periodically.

Keywords: *Groove pancreatitis, Brunner's gland, Santorini's duct*

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Groove pancreatitis is an uncommon form of segmental chronic pancreatitis affecting the head of the pancreas which is localized within the groove between the head of the pancreas, the duodenum and the common bile duct (CBD). It was first described by Becker and Bauchspeichel in 1973[1]. It has been reported under numerous names in the literature including cystic dystrophy of heterotopic pancreas, paraduodenal wall cyst, periampullary duodenal wall cyst, pancreatic hamartoma of the duodenal wall, myoadenomatosis, paraduodenal pancreatitis and groove pancreatitis[2,3]. A large series was reported by Stolte et al in 1982[4]. They diagnosed this form of pancreatitis in 30 out of 123 surgical pancreaticoduodenectomy (PD) specimens of chronic pancreatitis. It is still unclear either difficulty in discrimination of this entity from pancreatic head cancer preoperatively and/or high tendency to complicate the natural history of disease from its topography, PD was frequently performed in early reports(19.5-24.4%)[4,5].

Nowadays, imaging and endoscopic technology have markedly improved the accurate diagnosis of pancreatic disease. These armamentaria may solve the problem in the equivocal cases.

Correspondence to: Meesiri S, Department of Surgery, Srisangwornsukhothai Hospital, Sukhothai 64120, Thailand. E-mail: somchaimeesiri@yahoo.com

Case Report

A 44-year-old man with a history of chronic home-made alcoholic consumption for the past 27 years, presented with off and on epigastric pain radiating to the back, severe nausea, vomiting without jaundice, steatorrhoea nor weight loss, lasted for 2 years. Most episodes subsided within a few days after onset. He was admitted for investigation 2 years ago and diagnosed with acute pancreatitis with a pancreatic pseudocyst at the tail of the pancreas. Last admission, the epigastralgia and nausea, vomiting attacked for several hours before coming to the hospital. The physical examination exposed tenderness at the epigastrium, slightly parotid glands enlargement, and spider nevi at the chest wall. The laboratory findings included, serum amylase 292 U/L (normal, 28-100 U/L), urine amylase 2375 U/L (normal, < 460 U/L), liver function test, total protein 7.9 g/dL (normal, 6.6-8.7 g/dL), albumin 3.9 g/dL (normal, 3.4-4.8 g/dL), total bilirubin 1.2 mg/dL (normal, 0-1.1 mg/dL), direct bilirubin 0.2 mg/dL (normal, 0-0.3 mg/dL), alkaline phosphatase 125 U/L (normal, 35-104 U/L), aspartate aminotransferase (AST)

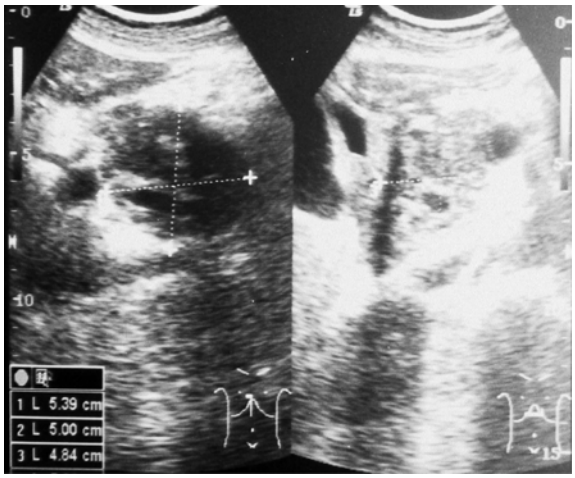

Fig. 1 Ultrasound of the upper abdomen shows a well defined outlined, irregular heterogeneous mass, size 54 x 48 x 50 mm at the pancreatic head

54 U/L (normal, 0-32 U/L), alanine aminotranferase (ALT) 177 U/L (normal, 0-31 U/L), L-lactate dehydrogenase (LDH) 593 U/L (normal, 230-460 U/L), serum calcium 8.7 mg/dL (normal, 8.6-10.2 mg/dL), fasting blood sugar (FBS) 93 mg/dL (normal, 74-106 mg/dL), carcinoembryonic antigen (CEA) 1.05 ng/mL (normal, 0-3.4 ng/mL), carbohydrate antigen 19-9 (CA 19-9) 10.7 U/mL (normal, < 27 U/mL).

Trancutaneous ultrasound of the upper abdomen showed a well defined outlined, irregular mixed echogenic mass, size 54 x 48 x 50 mm at the pancreatic head. Mildly dilated distal CBD about 9.9 mm, tapering into the pancreatic head mass, no evidence of intrahepatic bile duct dilatation, normal body and tail of the pancreas (Fig. 1). A computed tomography (CT) of the abdomen revealed a solid mass with small low density cystic areas in the pancreato-duodenal groove and involving the pancreatic head, approximately 5.7 x 3.7 x 4.9 cm (Fig. 2A). A mass was poorly enhanced and heterogeneous hypodense relative to pancreatic parenchyma on arterial phase (Fig. 2B) and isodense on portovenous phase (Fig. 2C). The descsending part of the duodenum had thickened wall and luminal narrowing (Fig. 2B). A long, smooth tapering, mildly dilated 0.7 cm CBD was observed. Neither peripancreatic vascular encasement nor regional lymphadenopathy was noted. Magnetic resonance imaging (MRI) demonstrated the same mass between the pancreatic head, duodenum and CBD that was hypointense on T1 weighted images (Fig. 3A), isointense on T2 weighted images (Fig. 3B) and

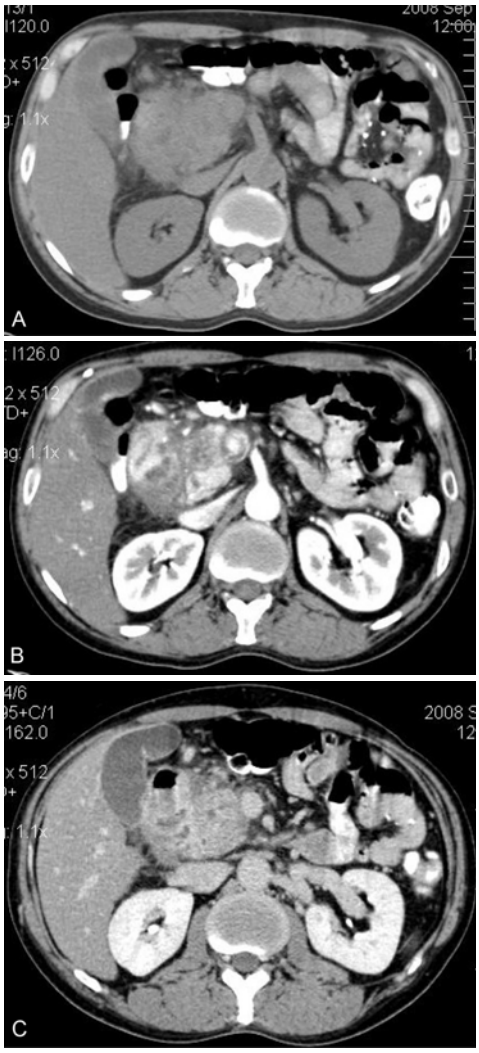

Fig. 2 A CT of the abdomen reveals a solid mass with small low density cystic areas in the pancreatoduodenal groove in pre-contrast phase (A). A mass is heterogeneous hypodense on arterial phase (B) and isodense on portovenous phase (C)

delayed, progressive inhomogeneous enhancement on contrast-enhanced dynamic images (Fig. 4A-E). Magnetic resonance cholangiopancreatography (MRCP) defined a prominent long, regular, smooth tapering of the CBD without intraluminal filling defect. Pancreatic duct was normal. Santorini's duct was not demonstrable (Fig. 5). Upper gastrointestinal endoscopy disclosed an edematous, reddish and hemorrhagic raised mucosa having a polypoid appearance which slightly compromised the second part of the duodenal lumen (Fig. 6A-B). Several

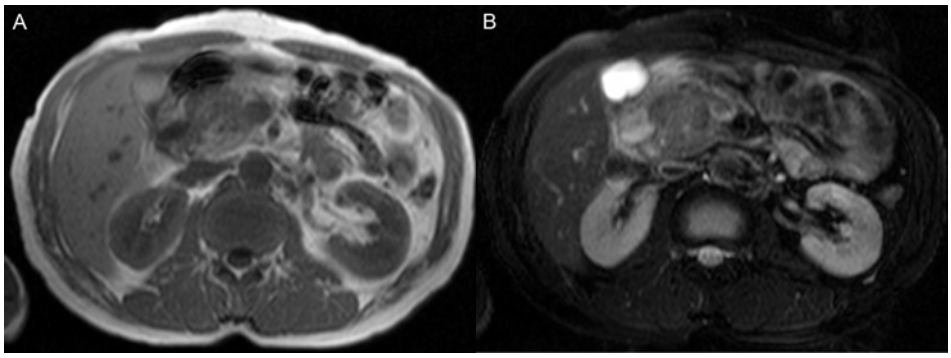

Fig. 3 MRI images demonstrate a corresponding CT mass which is hypointense on T1 weighted images (A), isointense on T2 weighted images (B)

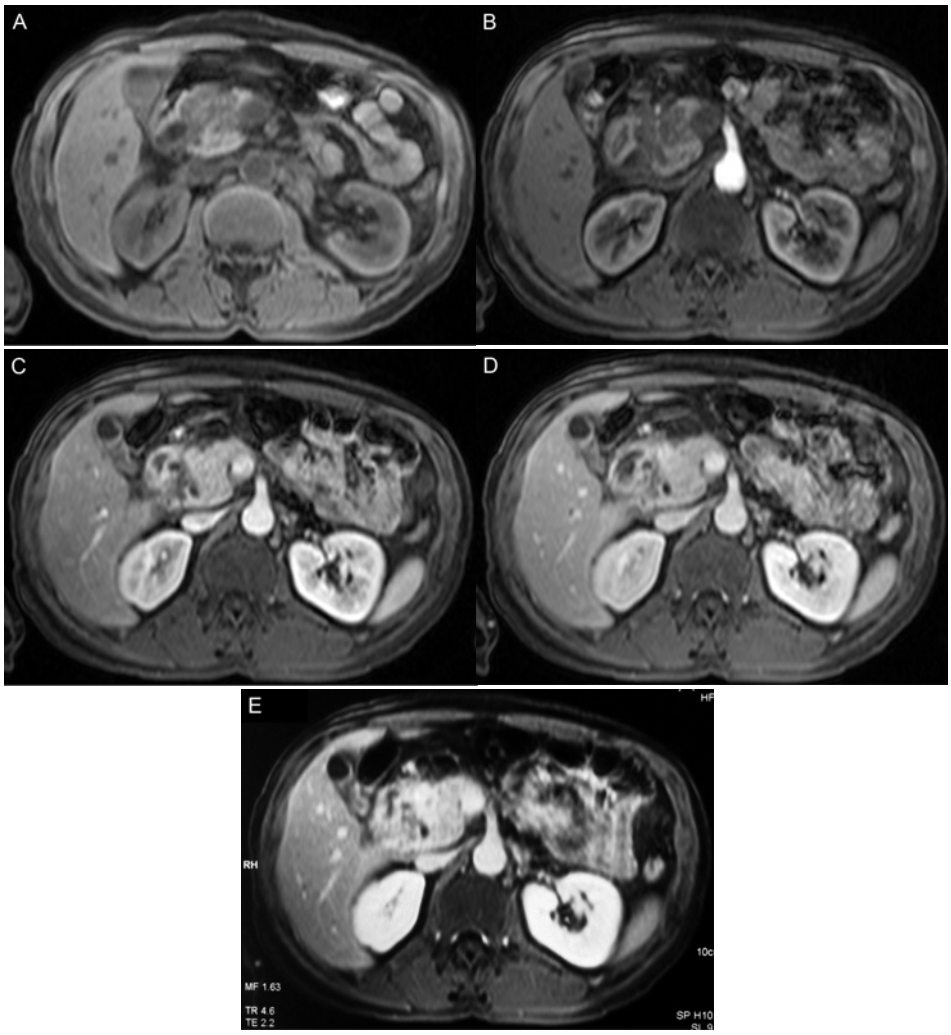

Fig. 4 Delayed, progressive inhomogeneous enhancement on contrast-enhanced dynamic images (A, pre-contrast; B, 10 seconds; C, 30 seconds; D, 60 seconds; E, 5 minutes after gadobutrol injection)

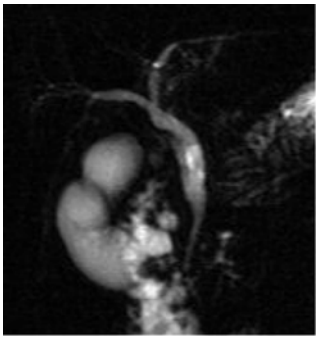

Fig. 5 MRCP image defines a CBD tapering which is classical of chronic pancreatitis

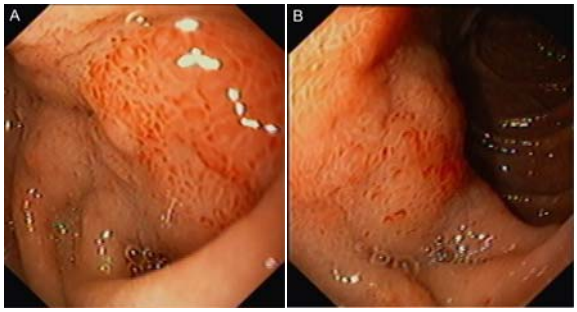

Fig. 6 Endoscopic study discloses a sessile mass with edematous, reddish and hemorrhagic mucosa (A) and slightly compromised the second part of duodenal lumen (B)

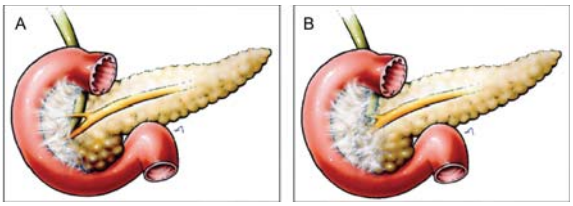

Fig. 7 Groove pancreatitis in 2 forms: a pure form (A), and a segmental form (B) (from reference number 6)

duodenal mucosal biopsies were taken from the mass, and periphery including ampulla. Histopathological examination seen infiltration of mononuclear inflammatory cells with eosinophils in lamina propria that suggested chronic inflammation. There was no evidence of malignancy. These clinical investigation findings appeared to be compatible with the diagnosis of groove pancreatitis. Then, the patient was treated conservatively and evaluated periodically.

Discussion

Groove pancratitis is classified as a rare disease but this might be partly due to lack of awareness of this disease. Becker and Mischke classified this condition into a pure form and a segmental form[5]. The pure form affects exclusively the groove while the segmental form extends to the pancreatic head (Fig. 7)[6].

Patients are predominantly male, 40-50 years old with a long history of alcoholic intake[1,2,4,7] present with clinical setting similar to common chronic pancreatitis but more pronounced recurrent vomiting from duodenal stenosis and impaired motility, often leading to significant weight loss[4,7]. Jaundice is uncommon (only one from 38 reported cases of groove pancreatitis in Japan[7]), and if present, often fluctuates, in contrast to the continuously progressive jaundice found in patients with pancreatic carcinoma[8]. Serum pancreatic enzymes often elevate, occasionally of serum hepatic enzymes[7] and rarely of tumour markers, CEA and CA 19-9[8].

Abdominal ultrasound usually unveils a heterogeneous hypoechoic mass. A CT scan often uncovers a non-homogeneous poorly enhanced mass in the groove, representing scar component, as well as duodenal wall cyst, duodenal wall thickening and duodenal narrowing[9]. MRI usually demonstrates a hypointense relative to pancreatic parenchyma on T1-weighted images, hypo-, iso-, or slightly hyperintense on T2 weighted images. Contrast-enhanced dynamic images display a delayed and progressive inhomogeneous enhancement reflecting its fibrous nature[6]. ERCP/MRCP unmasks a long, smooth concentric tubular stonosis of the CBD (differs from the circumscribed, irregular ductal stenosis of carcinoma of the head of the pancreas or complete ductal obstruction) with normal main pancreatic duct or rarely, with only slight irregularities[6]. The Santorini's duct is sometimes dilated, irregular can contain stone, protein plug, abscess or even intraductal carcinoma[1,4,7-8,10]. Widening of the distance between the distal CBD and duodenal lumen because of the presence of a space-occupying lesion in the groove and marked duodenal wall thickening can be identified[6]. Endoscopy detects an inflamed, raised duodenal mucosa with stenosis of duodenal lumen[7,11-13]. Grossly there is either thickening and scarring of the duodenal wall, extending to the adjacent pancreatic head tissue and/or sieve-like cystic changes in the duodenal wall. Stenosing distal CBD may be noticed. The pancreatic ductal system is grossly normal[1,3-4,8]. Microscopically the chronic inflammatory process resides in the duodenal submucosa, duodenal

wall and adjacent pancreatic tissue. Brunner's gland hyperplasia is typically present. Several small foci of necrosis environed by a dense myoid proliferation and intervening cystic ductal elements, acinar lobules and some islets create a histologic picture reminiscent of myoadenomatosis, pancreatic hamartoma or even leiomyoma in some cases. The duodenal wall contains dilated ducts, pseudocystic lesions as well as adjacent stromal reactions including hypercellular granulation tissue and foreign-body type giant cell reaction. Occasionally, there are also clusters of eosinophils[2,3]. These lesions often mimic periampullary tumours due to marked scarring and poorly defined borders of the process. Therefore the discrimination of groove pancreatitis from pancreatic cancer is often difficult or impossible in some patients[7,8].

The largest series was published by Shudo et al in 2002[7]. They reviewed 38 cases of groove panreatitis reported in Japan. The 36 men and 2 women with a median age of 51 years (range, 37-69 years). Only 2 out of them were asymptomatic. Almost all patients were alcoholic. Duodenal stenosis was found in 32 out of 35 patients and abnormal CBD, characterized by stenosis, compression, or wall rigidity were found in 24 out of 31 patients. In all 27 successful ERCP cases, Wirsung's duct was normal mostly, whereas Santorini's duct was intact in only 1 (Santorini's duct was not demonstrated in 10 patients). Therefore, they suggested that Santorini's duct abnormality was crucial to the diagnosis. Furthermore, excluded preoperative diagnosis ruled out pancreatic cancer, at least 30% of all groove pancreatitis cases need surgical treatment. Among 28 surgical-treated patients, 16 cases of preoperative diagnosis ruled out pancreatic cancer were included. These findings highlight the truth that groove pancreatitis is still a challenging and threatening disease.

The pathogenesis of this condition is still unknown but the reasonable explanation seems to be caused by a disturbance of pancreatic juice outflow in Santorini's duct from the obstacle such as a stone, impacted protein plug, tumour, absence of the minor papilla or hyperviscosity/hyperstimulation in the consequence of chronic alcoholism[1,4,7,8,14].

Initial management of choice is conservative treatment resembling usual chronic pancreatitis. Because the risk of coexistent carcinoma in Santorini's duct has been discovered[10] and natural history of disease is still unclear, careful follow-up is required. A PD preserved in complicated or equivocal case gives an excellent result in a selected one[12,15].

Conclusion

Attributed to the varying clinical presentation and often masquerades as pancreatic cancer and/or duodenal stenosis, a knowledge of presence of groove pancreatitis suggests a differential diagnosis is the best way to avoid unnecessary radical surgery.

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รายงานผู้ป่วยโรค groove pancreatitis 1 รายจากประเทศไทย

สมชาย มีศิริ

Groove pancreatitis เป็นรูปแบบหนึ่งของตับอ่อนอักเสบเรื้อรังที่พบได้น้อยมาก เกิดขึ้นที่ตำแหน่ง pancreatoduodenal groove การมีเนื้อเยื่อพังผืดในตำแหน่งดังกล่าว บางครั้งทำให้วินิจฉัยแยกโรคก่อนผ่าตัดจากมะเร็งตับอ่อนได้ยาก ผู้นิพนธ์รายงานกรณีศึกษาผู้ป่วยโรค groove pancreatitis เป็นชายไทยอายุ 44 ปี มาด้วยอาการปวดท้องบริเวณลิ้นปี่ คลื่นไส้อาเจียน เป็น ๆ หาย ๆ นาน 2 ปี และดื่มสุราเป็นนิตย์เป็นระยะเวลานาน ผลการตรวจอัลตราซาวด์ของช่องท้องพบ an irregular mixed echogenic mass ที่ตำแหน่งหัวของตับอ่อน ผลการตรวจซีทีสแกนพบ a poorly enhanced solid mass with small low density cystic areas ที่ตำแหน่ง groove, ส่วนที่ 2 ของลำไส้เล็กส่วนดูโอดีนั่มมีผนังหนาขึ้น และรูกลวงในลำไส้ตีบแคบลง ผลการตรวจเอ็มอาร์ไอพบก้อนในตำแหน่งตรงกันที่มีลักษณะ hypointense on T1 weighted images, isointense on T2 weighted images และเมื่อศึกษาด้วยวิธี dynamic contrast study พบว่ามี delayed, progressive inhomogeneous enhancement ผลการตรวจ เอ็มอาร์ซีพี พบท่อน้ำดีโต ผิวเรียบ เรียวเล็กลง การศึกษาด้วยวิธีส่องกล้องทางเดินอาหารพบ sessile mass ที่ตำแหน่งส่วนที่ 2 ของลำไส้เล็กส่วนดูโอดีนัมมีลักษณะอักเสบ ผลการตรวจในระดับจุลทรรศน์จากชิ้นเนื้อที่ถูกตัดพบว่า เป็นเพียงการอักเสบเรื้อรังแพทย์จึงให้การรักษาแนวอนุรักษ์นิยม และนัดผู้ป่วย มาประเมินเป็นระยะ ๆ