

Cystic dystrophy of the duodenal wall is not always associated with chronic pancreatitis

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Abstract

Cystic dystrophy of the duodenal wall is a rare form of the disease which was described in 1970 by French authors who reported the presence of focal pancreatic disease localized in an area comprising the C-loop of the duodenum and the head of the pancreas. German authors have defined this area as a "groove". We report our recent experience on cystic dystrophy of the paraduodenal space and systematically review the data in the literature regarding the alterations of this space. A MEDLINE search of papers published between 1966 and 2010 was carried out and 59 papers

were considered for the present study; there were 19 cohort studies and 40 case reports. The majority of patients having groove pancreatitis were middle aged. Mean age was significantly higher in patients having groove carcinoma. The diagnosis of cystic dystrophy of the duodenal wall can now be assessed by multi-detector computer tomography, magnetic resonance imaging and endoscopic ultrasonography. These latter two techniques may also add more information on the involvement of the remaining pancreatic gland not involved by the duodenal malformation and they may help in differentiating "groove pancreatitis" from "groove adenocarcinoma". In conclusion, chronic pancreatitis involving the entire pancreatic gland was present in half of the patients with cystic dystrophy of the duodenal wall and, in the majority of them, the pancreatitis had calcifications.

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Key words: Pancreatitis; Cystic dystrophy of duodenal wall; Therapy; Outcome

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INTRODUCTION

Cystic dystrophy of the duodenal wall is a rare form of the

disease which was described for the first time in 1970 by Potet and Duclert^[1]. Potet and Duclert and other French authors^[2,3] described the presence of focal pancreatic disease localized in an area comprising the C-loop of the duodenum and the head of the pancreas (Figure 1A). In 1991, Becker and Mischke^[4] defined this area as a “groove” and pointed out that it serves as a bed for the large vessels, lymph nodes, common bile duct (CBD) and main pancreatic duct. These authors also reported that pancreatitis can be found in this area and they suggested the term “groove pancreatitis” which was well received. They also classified groove pancreatitis as “pure groove pancreatitis” (Figure 1B), **segmental pancreatitis of the head** and chronic pancreatitis with groove involvement (Figure 1C). **In addition, in recent years, Adsay and Zamboni^[5] proposed the term “paraduodenal pancreatitis” in patients classified as having “cystic dystrophy of the heterotopic pancreas” or “paraduodenal wall cyst” or “groove pancreatitis”; they also recognized two types of pancreatitis: one characterized by cystic changes and the other characterized by solid lesions. These authors pointed out that the latter type of pancreatitis is difficult to distinguish from an adenocarcinoma originating in this area. Finally, the presence of cystic dystrophy of the duodeno-pancreatic space together with chronic pancreatitis of the remaining pancreas is not always true because there is also the possibility of disease limited to the CBD^[6]. Thus, in this review, we report our recent experience on cystic dystrophy of the space from the C-loop of the duodenum and the pancreas by reporting three cases observed in the last year, and also systematically review and discuss the data in the literature on the alteration of the groove space.**

OUR EXPERIENCE ON THREE RECENT OBSERVED CASES OF CYSTIC DYSTROPHY OF THE DUODENAL WALL

We report our experience on three recently observed cases of cystic dystrophy of duodenal wall. Patients were one female and two males aged 49-65 years having persistent abdominal pain and weight loss. One male patient was a drinker and the diagnosis in all 3 patients was confirmed at laparotomy. The pathological examination in two cases confirmed cystic dystrophy of duodenal wall associated with chronic pancreatitis in one case and autoimmune pancreatitis and pancreatic carcinoma in the remaining one.

Case 1

A 65-year-old female was admitted to our department in April 2009 for persistent abdominal discomfort and progressive weight loss (about 5 kg in two months). Before this admission, she had had a one-year history of recurrent epigastric pain; an ultrasonographic (US) examination showed gallstones and the patient had been cholecystectomized in another hospital. After surgery, she

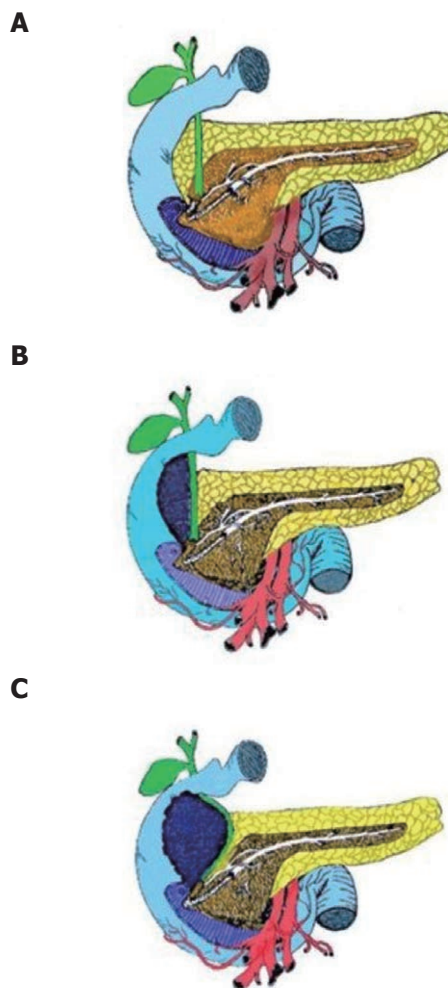


Figure 1 Classification of the various types of groove pancreatitis. A: Typical finding of groove pancreatitis (purple area); B: Segmental head pancreatitis: the scar tissue (dark blue) expands towards the duodenum; C: Pancreatitis of the head: the scar tissue (dark blue) expands to the duodenal area, determining duodenal stenosis and displacement of the common bile duct.

continued to have recurrent and frequent episodes of epigastric pain; US showed a dilation of the CBD and, two months after surgery, she underwent an endoscopic sphincterotomy. One month after this procedure, epigastric pain reappeared and, due to the presence of scleral jaundice (total bilirubin 3.2 mg/dL), the patient underwent another endoscopic retrograde cholangiopancreatography (ERCP). The papilla of Vater was substenotic and another sphincterotomy was carried out without any clinical improvement. On admission to our department, physical examination was unremarkable as was a routine blood examination; her body temperature was 37.2 °C, her arterial pressure was 110/60 mmHg and her cardiac rate was 73 bpm. Contrast-enhanced multidetector computer tomography (MDCT) was carried out. This examination showed the presence of multiple hypodense lesions in the liver (Figure 2); a US fine needle biopsy of one of these lesions was carried out and the pathological specimen was compatible with an abscess; the liver tissue was also cultured and the patient was treated with a spe-



Figure 2 Case No. 1 computer tomography liver evaluation. Liver multiple hypodense lesions compatible with abscesses.



Figure 3 Case No. 1 computer tomography duodenal and pancreatic gland evaluation. A: Presence of duodenal bulging; B: Normal appearance of the pancreatic gland.

cific antibiotic. At computer tomography (CT) examination, there was the presence of biliary sludge and a dilation of the left intrahepatic biliary tree. There was also the presence of duodenal bulging (Figure 3A) while the pancreatic gland was normal (Figure 3B). An endoscopic US (EUS) was finally carried out. It confirmed the presence of duodenal bulging (Figure 4A) and showed CBD sludge; in addition, cysts in the duodenal wall were seen (Figure 4B) and a diagnosis of cystic dystrophy of duodenal wall was made. The patient refused surgery, and conservative treatment with ursodeoxycholic acid was carried out. Twenty months after discharge, the patient

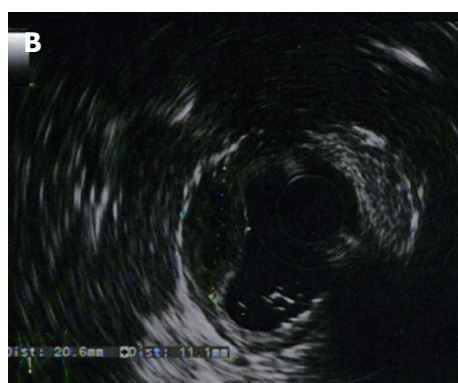
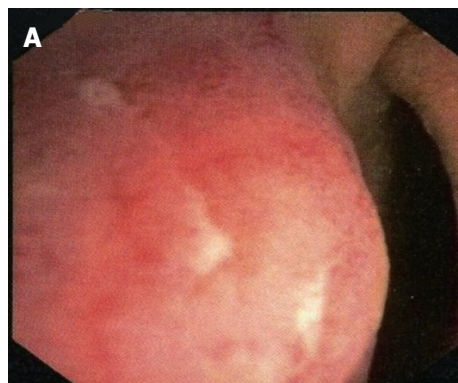


Figure 4 Case No. 1 computer tomography duodenal and pancreatic gland evaluation. A: Presence of duodenal bulging; B: Presence of cysts in the duodenal wall.

was free of abdominal discomfort and regained her lost weight.

Case 2

A 49-year-old male patient with a history of chronic alcoholic consumption (about 40 g of pure alcohol intake per day) was admitted to our Department in May 2010 with persistent epigastric pain of seven months duration associated with nausea and biliary vomiting; there was also weight loss of 13 kg. The following biochemical tests were carried out: Hb 11.9 g/dL, MCV 85.8; amylase 156 U/L (upper reference value 100), CA 19-9: 52 U/mL (upper reference value 37). The patient underwent an upper gastrointestinal endoscopy which was normal. Ultrasonographic examination did not show alterations of the abdominal parenchyma. MDCT showed an enlarged pancreatic head and the presence of multiple cysts between the enlarged pancreatic head and the duodenum (Figure 5A); the remaining pancreas was normal as was demonstrated by magnetic resonance imaging (MRI) (Figure 5B). The patient was operated on and a pancreatic head resection was performed. The pathology of the resected specimen showed cystic dystrophy of the duodenal wall with hypertrophy of the Brunner glands and the presence of an ectopic pancreas (Figure 6A), showing chronic pancreatitis (Figure 6B). Seven months after surgery, the patient was symptom free and in good general health.

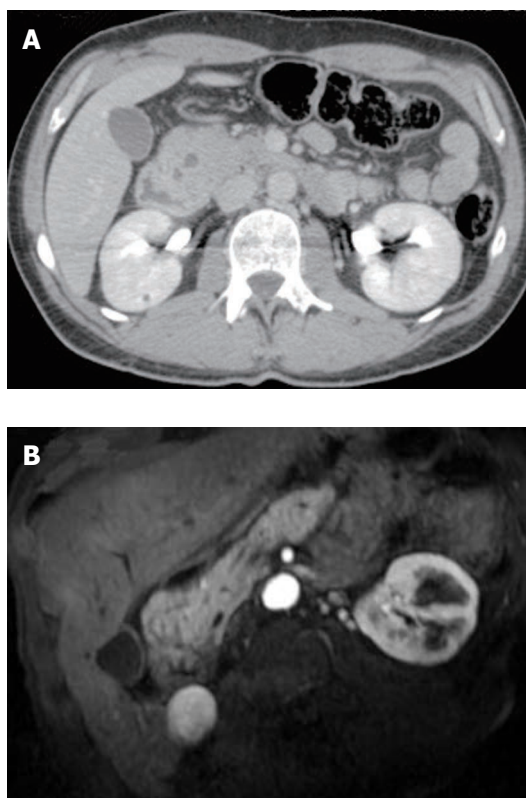


Figure 5 Case No. 2 computer tomography duodenal and pancreatic gland evaluation. A: Enlarged pancreatic head and the presence of multiple cysts between the enlarged pancreatic head and the duodenum (multidetector computer tomography); B: The remaining pancreas was normal as demonstrated by the magnetic resonance imaging.

Case 3

A 56-year-old male affected by Crohn's disease was seen in August 2010 with persistent epigastric pain of one month duration associated with jaundice, weight loss, nausea and intermittent vomiting. The patient was not an alcohol drinker. The following biochemical tests were carried out: total bilirubin, 25.4 mg/dL, AST, 63 U/L (upper normal limit 38), ALT, 66 U/L (upper normal limit 40), alkaline phosphatases, 1105 U/L (normal value 98-280), amylase, 108 U/L (upper normal limit 100), lipase, 293 U/L (upper normal limit 60), CA, 19-9 2345 U/mL (upper reference value 37). The patient underwent US which showed a dilated CBD and a mass of 2.5 cm in the head of the pancreas. The MDCT showed the pancreatic head focally enlarged with a 2.5 cm heterogeneous area extending to and involving the wall of the posterior bulbar duodenum. The main pancreatic duct was uniformly dilated in caliber and appearance with no changes in the pancreatic body or tail. The patient underwent a pancreaticoduodenectomy and surgical pathology showed the presence of cystic dystrophy of the duodenal wall (Figure 7A) with aspects of chronic pancreatitis in the heterotopic pancreas (Figure 7A), aspects of autoimmune pancreatitis (Figure 7B) and, finally, groove adenocarcinoma extending to the pancreatic head (Figure 7C). At present, the patient is still alive and is in adjuvant chemotherapy

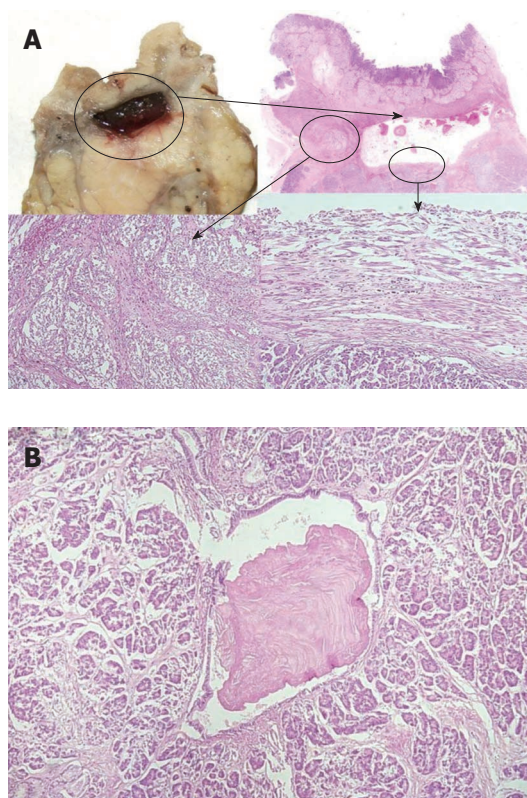


Figure 6 Case No. 2 pancreatic and duodenal surgical specimens. A: Resected specimen showing cystic dystrophy of the duodenal wall with hyperplasia of the Brunner glands and the presence of an ectopic pancreas (arrows); B: Chronic pancreatitis in the remaining pancreas together with cystic dystrophy of the duodenal wall.

with gemcitabine.

CLINICAL CONSIDERATIONS AND AIMS

The present report involving three cases of cystic dystrophy of the duodenal wall represents one of the few case series published concerning this rare entity. All of our patients presented with symptoms consistent with chronic pancreatitis; however, pancreatic diseases were found in two and these two patients improved dramatically after surgical head pancreatic resection while one is symptom free after medical treatment. It is important to diagnose the pathological involvement of the proximal duodenum in order to detect the presence of malignancy and to evaluate the prognosis of these subjects. In order to better establish the features of this rare entity we also undertook a systematic review of the literature.

LITERATURE SEARCH AND DATA EXTRACTION

A search was carried out on December 18, 2010 using the MEDLINE/PubMed database (United States National Library of Medicine National Institutes of Health) in order to select the data existing in the literature under the headings of pancreatitis and groove

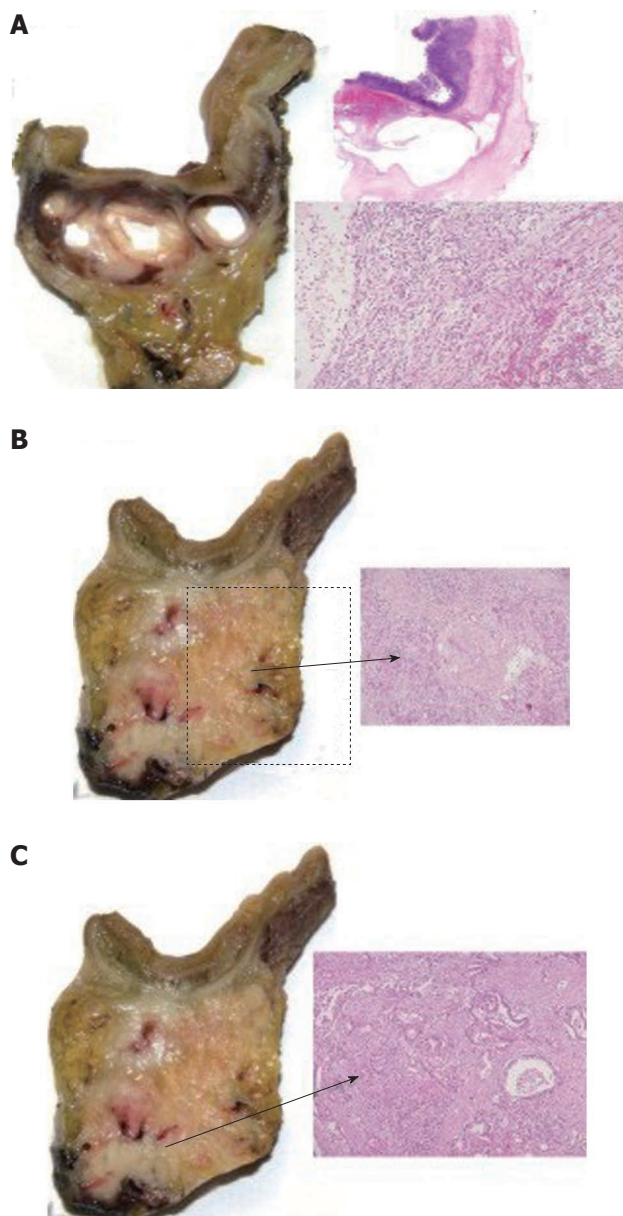


Figure 7 Case No. 3 pancreatic and duodenal pathological specimens. A: Cystic dystrophy of the duodenal wall with aspects of chronic pancreatitis in the heterotopic pancreas; B: Aspects of autoimmune pancreatitis (arrow); C: Groove adenocarcinoma extending to the pancreatic head (arrow).

pancreatitis. The terms used were “groove pancreatitis” or “duodenal cystic dystrophy” (explanatory variables) and “pancreatic diseases” (outcome variable). The search was limited to human studies written in English. We identified additional studies by means of a hand search of the bibliographies from the primary studies, review articles and key journals. A total of 70 citations were found in MEDLINE/PubMed^[4-73]. Four investigators (Pezzilli R, Morselli-Labate AM, Fabbri D, and Imbrogno A) independently screened all articles for those meeting the broad inclusion criteria. Of the 70 papers, 4 were excluded because they contained data regarding diseases other than those searched for^[8,15,48,71]. Of the remaining 66 papers, 10 were excluded because they

were review articles not containing data useful for the analyses^[4,5,24,41,42,45,56,58,60,68] and one because it was a comment on an article^[19] without new data/cases; therefore, 55 papers with available data remained. Of these 55 papers, 4 were also excluded for the following reasons: 1 because it was a duplicated publication^[55] and 3 because it was not possible to extract useful data^[49,57,73]. Eight papers were added to these 51 papers because they were extracted from the references^[74-81]. Thus, 59 papers were considered for the present study; there were 19 cohort studies^[6,7,10-13,16,18,23,30,36,37,39,40,46,47,52,63,81] and 40 case reports^[9,14,17,20-22,25-29,31-35,38,43,44,50,51,53,54,59,61,62,64-67,69,70,72,74-80].

For each study, the following information was recorded: gender, mean age for the cohort studies or age of the subjects studied in the case reports, interval time from the appearance of the symptoms to diagnosis, alcoholism, the presence of clinical variables (such as abdominal pain, weight loss and jaundice, hyperamylasemia, CBD stenosis, duodenal stenosis), the need for surgery and the type of surgery, the possible presence of chronic pancreatitis in the pancreas together with cystic dystrophy as well as the presence of pseudocysts, the possible presence of pancreatic neoplasms, the time of follow-up and death.

Data are presented as absolute numbers and relative frequencies, mean \pm SD, **medians, ranges, and interquartile ranges (IQR)**; follow-up data are also presented as crude survival.

EVALUATION OF THE SELECTED STUDIES

Due to the low frequency of diseases, such as groove pancreatitis and groove carcinomas, there is a limited number of cohort studies (No. 19) and a large number of case reports (No. 40). All the cohort studies were retrospective and patients were enrolled from 1959^[11] to 2008^[6]. Thus, the changes in diagnostic techniques with the appearance of MRI and EUS in clinical practice render the studies not comparable as to what is the best technique for diagnosing groove diseases. Furthermore, the mean follow-ups vary greatly and the longest follow-up is about 8 years which is that reported by Casetti *et al*^[63]. As shown in Tables 1-8, we found no substantial differences between the data reported in the cohort studies and those we calculated when grouping the series of case reports by gender, age at diagnosis, alcoholism, presence of pain, weight loss, jaundice, hyperamylasemia, CBD stenosis, duodenal stenosis and the need for surgery. The presence of chronic pancreatitis and deaths were more frequently reported in the cohort studies than in the case reports while associated adenocarcinoma and pseudocysts were more frequently reported in the case reports than in the cohort studies.

EPIDEMIOLOGY

We have no epidemiological data regarding the preva-

Table 1 Epidemiological and clinical characteristics of patients in the 18 retrospective studies involving patients with a benign cystic duodenal wall

Author ^[Ref.] yr	Time interval of patient enrollment	n (%)			Age (yr) Mean (range)	Alcohol drinkers n (%)
		Total	Males	Females		
Stolte <i>et al</i> ^[7] 1982	NR	30	30 (100)	-	41.3 (NR)	22 (73.3)
Yamaguchi <i>et al</i> ^[10] 1992	1983-1989	8	8 (100)	-	58.0 (33-70)	4 (50.0)
Fléjou <i>et al</i> ^[11] 1993	1959-1991	10	10 (100)	-	41.0 (31-56)	2 (20.0)
Itoh <i>et al</i> ^[12] 1994	NR	4	3 (75.0)	1 (25.0)	43.0 (37-53)	NR
Fékété <i>et al</i> ^[13] 1996	1989-1993	6	6 (100)	-	40.0 (35-46)	4 (66.7)
Procacci <i>et al</i> ^[16] 1997	1992-1996	10	10 (100)	-	41.0 (32-59)	9 (90.0)
Irie <i>et al</i> ^[18] 1998	1995-1996	5	5 (100)	-	41.0 (33-46)	2 (40.0)
Vullierme <i>et al</i> ^[23] 2000	1988-1998	20	18 (90.0)	2 (10.0)	44.0 (36-56)	NR
Aoun <i>et al</i> ^[81] 2005	NR	4	2 (50.0)	2 (50.0)	69.0 (66-71)	NR
Pessaux <i>et al</i> ^[36] 2006	1990-2004	12	11 (91.7)	1 (8.3)	42.4 (34-54)	9 (75.0)
Jouannaud <i>et al</i> ^[37] 2006	1990-2002	23	20 (87.0)	3 (13.0)	45.0 (30-66)	23 (100)
Tison <i>et al</i> ^[39] 2007	1983-2001	9	8 (88.9)	1 (11.1)	48.0 (37-63)	8 (88.9)
Rebours <i>et al</i> ^[40] 2007	1995-2004	105	96 (91.4)	9 (8.6)	46.0 (24-75)	86 (81.9)
Rahman <i>et al</i> ^[46] 2007	2000-2005	11	10 (90.9)	1 (9.1)	48.0 (35-61)	10 (90.9)
Castell-Monsalve <i>et al</i> ^[47] 2008	NR	5	4 (80.0)	1 (20.0)	47.0 (40-53)	4 (80.0)
Jovanovic <i>et al</i> ^[52] 2008	1996-2006	13	10 (76.9)	3 (23.1)	41.5 (17-60)	6 (6.2)
Casetti <i>et al</i> ^[63] 2009	1990-2006	58	54 (93.1)	4 (6.9)	44.7 (IQR 36.8-51.8)	57 (98.3)
Ishigami <i>et al</i> ^[6] 2010	2001-2008	15	14 (93.3)	1 (6.7)	48.0 (31-64)	NR
Overall	-	348	319 (91.70)	29 (8.30)	-	246/305 (80.70)

IQR: Interquartile range; NR: Not reported.

Table 2 Epidemiological and clinical characteristics of patients in the 18 retrospective studies involving patients with a benign cystic duodenal wall (continues from Table 1) n (%)

Author ^[Ref.] yr	Time interval from the symptoms to the diagnosis	Abdominal pain		Weight loss	Jaundice	Hyperamylasemia
		No. of cases	Type			
Stolte <i>et al</i> ^[7] 1982	NR	NR	NR	30 (100)	NR	NR
Yamaguchi <i>et al</i> ^[10] 1992	NR	3 (37.5)	NR	0	2 (25.0)	NR
Fléjou <i>et al</i> ^[11] 1993	NR	7 (70.0)	Persistent	9 (90.0)	4 (40.0)	NR
Itoh <i>et al</i> ^[12] 1994	NR	3 (75.0)	NR	NR	NR	3 (75.0)
Fékété <i>et al</i> ^[13] 1996	NR	6 (100)	Recurrent	6 (100)	0	6 (100)
Procacci <i>et al</i> ^[16] 1997	4.5 yr (1-9)	10 (100)	Recurrent	4 (40.0)	1 (10.0)	NR
Irie <i>et al</i> ^[18] 1998	NR	4 (80.0)	NR	0	0	NR
Vullierme <i>et al</i> ^[23] 2000	41.5 d (1-140)	NR	NR	NR	NR	NR
Aoun <i>et al</i> ^[81] 2005	NR	3 (75.0)	NR	0	1 (25.0)	NR
Pessaux <i>et al</i> ^[36] 2006	NR	9 (75.0)	Persistent in 4 (44.4)	12 (100)	2 (16.7)	NR
Jouannaud <i>et al</i> ^[37] 2006	NR	22 (95.7)	NR	16 (69.6)	0	NR
Tison <i>et al</i> ^[39] 2007	NR	9 (100)	NR	9 (100)	2 (22.2)	NR
Rebours <i>et al</i> ^[40] 2007	1 yr (0-24)	91 (86.7)	Continuous in 35 (38.4); occasional in 56 (61.5)	73 (69.6)	13 (12.4)	NR
Rahman <i>et al</i> ^[46] 2007	NR	11 (100)	Recurrent in 8 (72.7)	10 (90.9)	0	2 (18.2)
Castell-Monsalve <i>et al</i> ^[47] 2008	NR	5 (100)	Persistent	NR	NR	5 (100)
Jovanovic <i>et al</i> ^[52] 2008	7.5 mo (0.5-36)	12 (92.3)	NR	4 (30.8)	4 (30.8)	NR
Casetti <i>et al</i> ^[63] 2009	NR	46 (79.3)	Persistent	NR	3 (5.2)	NR
Ishigami <i>et al</i> ^[6] 2010	NR	NR	NR	NR	NR	NR
Overall	-	241/283 (85.20)	-	173/246 (70.30)	32/274 (11.70)	16/26 (61.50)

NR: Not reported.

lence and incidence of cystic dystrophy of the duodenal wall in the general population. The data regarding this anomaly mainly describes patients with associated chronic pancreatitis. A recent Italian survey which reviewed the data on chronic pancreatitis in Italy in mixed medical/surgical cases from 2000 to 2005^[57] reported that the frequency of groove pancreatitis was 6.2% (55

out of 893 patients) with a higher frequency in males (7.6%, 50/660) than in females (2.1%, 5/233). In a surgical setting, groove pancreatitis ranges from 2.7% to 24.5%^[4,7,10,63]; in these cases, the frequency in males is also higher than that in females. We have no epidemiological data regarding groove carcinomas or biliary involvement without pancreatitis or pancreatic adenocar-

Table 3 Epidemiological and clinical characteristics of patients in the 18 retrospective studies involving patients with a benign cystic duodenal wall (continues from Table 2) *n* (%)

Author ^[Ref.] yr	Imaging	Duodenal findings	CBD stenosis	Duodenal stenosis
Stolte <i>et al</i> ^[7] 1982	NR	Brunner hyperplasia in 25	15 (50.0)	NR
Yamaguchi <i>et al</i> ^[10] 1992	US, CT, ERCP, PTC	Edema and nodular appearance; Brunner hyperplasia	4 (50.0)	5 (62.5)
Fléjou <i>et al</i> ^[11] 1993	ERCP, EUS	Edema and congestion of the mucosa	0	7 (70.0)
Itoh <i>et al</i> ^[12] 1994	CT	NR	NR	NR
Fékété <i>et al</i> ^[13] 1996	CT, ERCP, EUS	Edema and congestion of the mucosa	0	5 (83.3)
Procacci <i>et al</i> ^[16] 1997	CT, ERCP, EUS	Inflammation in 8	2 (20.0)	2 (20.0)
Irie <i>et al</i> ^[18] 1998	MRI	Brunner hyperplasia in 3	2 (40.0)	3 (60.0)
Vullierme <i>et al</i> ^[23] 2000	CT	NR	3 (15.0)	20 (100)
Aoun <i>et al</i> ^[81] 2005	US, CT, ERCP, EUS	NR	4 (100)	NR
Pessaux <i>et al</i> ^[36] 2006	US, EUS, CT, ERCP, MRI	NR	NR	NR
Jouannaud <i>et al</i> ^[37] 2006	EUS, CT	Inflammation in 3	NR	8 (34.8)
Tison <i>et al</i> ^[39] 2007	US, CT, MRI, angiography	Non specific inflammation in 9	5 (55.6)	9 (100)
Rebours <i>et al</i> ^[40] 2007	CT, EUS	Brunner hyperplasia in 61	26 (24.8)	50 (47.6)
Rahman <i>et al</i> ^[46] 2007	CT, MRI, EUS	Brunner hyperplasia	0	5 (45.5)
Castell-Monsalve <i>et al</i> ^[47] 2008	MRI, EUS	Duodenal stenosis in 3	3 (60.0)	3 (60.0)
Jovanovic <i>et al</i> ^[52] 2008	US, CT, MRI, EUS	NR	6 (46.2)	NR
Casetti <i>et al</i> ^[63] 2009	US, CT, MRI, EUS	NR	3 (5.2)	NR
Ishigami <i>et al</i> ^[61] 2010	CT, MRI	NR	9 (60.0)	NR
Overall	-	-	82/309 (26.50)	117/212 (55.20)

CBD: Common bile duct; NR: Not reported; US: Transabdominal ultrasonography; CT: Computer tomography; MRI: Magnetic resonance imaging; EUS: Endoscopic ultrasonography; ERCP: Endoscopic retrograde cholangiopancreatography; PTC: Percutaneous transhepatic cholangiography.

Table 4 Epidemiological and clinical characteristics of patients in the 18 retrospective studies involving patients with a benign cystic duodenal wall (continues from Table 3) *n* (%)

Author ^[Ref.] yr	Surgery		Associated chronic pancreatitis	Associated neoplasms	Pseudocyst	Follow-up		
	No. of cases	Type				Mean (range)	Death	Lost
Stolte <i>et al</i> ^[7] 1982	30 (100)	PD	4 (13.3) (all with calcification)	No	5 (16.7)	NR	NR	NR
Yamaguchi <i>et al</i> ^[10] 1992	8 (100)	PD	NR	No	No	2 yr	1 (12.5)	NR
Fléjou <i>et al</i> ^[11] 1993	10 (100)	WP in 8; derivative in 2	0	No	No	1-5 yr	1 (10.0)	4 (40.0)
Itoh <i>et al</i> ^[12] 1994	3 (75.0)	PD	NR	No	No	No	NR	NR
Fékété <i>et al</i> ^[13] 1996	6 (100)	PD in 5; antrectomy in 1	NR	No	No	32 mo (18-64)	No	NR
Procacci <i>et al</i> ^[16] 1997	10 (100)	PD	7 (70.0) (calcifications in 5)	No	5 (head) (50.0)	NR	NR	NR
Irie <i>et al</i> ^[18] 1998	3 (60.0)	PD	2 (40.0) (all with calcifications)	No	No	NR	NR	NR
Vullierme <i>et al</i> ^[23] 2000	20 (100)	PD	9 (45.0) (calcifications in 5)	No	No	NR	NR	NR
Aoun <i>et al</i> ^[81] 2005	4 (100)	PD	NR	NR	NR	NR	NR	NR
Pessaux <i>et al</i> ^[36] 2006	12 (100)	PD	8 (66.7) (calcification in 2)	No	No	64 mo (6-158)	1 (8.3)	1 (8.3)
Jouannaud <i>et al</i> ^[37] 2006	14 (60.9)	PD in 11; derivative in 3	17 (73.9) (calcification in 10)	No	No	47 mo	1 (4.3)	NR
Tison <i>et al</i> ^[39] 2007	9 (100)	PD	5 (55.6)	No	No	72 mo	4 (44.4)	NR
Rebours <i>et al</i> ^[40] 2007	29 (27.6)	PD in 17; digestive and biliary by pass in 12	97 (92.4) (calcification in 96)	No	No	15 mo (0-243)	NR	NR
Rahman <i>et al</i> ^[46] 2007	11 (100)	PD	0	No	No	NR	NR	NR
Castell-Monsalve <i>et al</i> ^[47] 2008	4 (80.0)	WP in 3; 1 laparotomy	3 (60.0)	No	No	NR (13-36 mo)	No	NR
Jovanovic <i>et al</i> ^[52] 2008	13 (100)	PD	6 (46.2)	No	No	NR	NR	NR
Casetti <i>et al</i> ^[63] 2009	58 (100)	PD	NR	Neuroendo- crine in 1	No	93.6 mo (IQR 59.7-129.7)	NR	NR
Ishigami <i>et al</i> ^[61] 2010	6 (40.0)	PD in 3, derivative surgery in 3	NR	NR	NR	NR	NR	NR
Overall	250/348 (71.8)	-	158/253 (62.5)	1/329 (0.3%)	10/329 (3.0)	-	8/73 (11.0)	5/22 (22.7)

IQR: Interquartile range; NR: Not reported; PD: Pancreaticoduodenectomy; WP: Whipple procedure.

cinoma. In all these studies, the patients having groove pancreatitis were middle aged (about 45 years of age), having a wide range from 20 mo^[76] to 75 years of age^[40]. Only two of the patients described were children (a

20-mo-old girl and a 15-year-old boy)^[51,76]. Mean age was significantly higher in patients having groove carcinoma than in those having groove pancreatitis, namely 70 years of age (range 57 to 80 years)^[51].

Table 5 Epidemiological and clinical characteristics of patients in the 38 case report papers involving 46 subjects with a benign cystic duodenal wall (a paper may report more than one patient), the three cases reported in the present paper are also shown

Author ^[Ref.] yr	Gender	Age (yr)	Alcohol drinker
Bill <i>et al</i> ^[74] 1982	Male	64	Yes
Holstege <i>et al</i> ^[75] 1985	Male	44	Yes
Tio <i>et al</i> ^[9] 1991	Male	48	NR
Tio <i>et al</i> ^[9] 1991	Male	53	NR
Flaherty <i>et al</i> ^[75] 1992	Female	20 mo	No
Izbicki <i>et al</i> ^[77] 1994	Male	25	NR
Fujita <i>et al</i> ^[14] 1997	Male	42	Yes
Shudo <i>et al</i> ^[17] 1998	Male	66	Yes
Wu <i>et al</i> ^[78] 1998	Male	39	NR
Babál <i>et al</i> ^[79] 1998	Female	70	NR
Rubay <i>et al</i> ^[21] 1999	Male	46	Yes
Balachandar <i>et al</i> ^[22] 1999	Male	18	NR
Mohl <i>et al</i> ^[25] 2001	Male	44	Yes
Mohl <i>et al</i> ^[25] 2001	Male	42	Yes
Munthali Lovemore <i>et al</i> ^[26] 2001	Male	24	No
Indinnimeo <i>et al</i> ^[27] 2001	Male	46	Yes
Shudo <i>et al</i> ^[28] 2002	Male	53	Yes
Glaser <i>et al</i> ^[29] 2002	Male	51	Yes
Hwang <i>et al</i> ^[31] 2003	Male	46	Yes
Jovanovic <i>et al</i> ^[32] 2004	Male	38	No
McFaul <i>et al</i> ^[80] 2004	Male	29	Yes
McFaul <i>et al</i> ^[80] 2004	Male	62	Yes
Isayama <i>et al</i> ^[33] 2005	Male	56	Yes
Chatelain <i>et al</i> ^[34] 2005	Male	47	Yes
Chatelain <i>et al</i> ^[34] 2005	Female	44	Yes
Balzan <i>et al</i> ^[35] 2005	Male	47	NR
Sanada <i>et al</i> ^[43] 2007	Male	81	No
Balakrishnan <i>et al</i> ^[44] 2007	Male	40	Yes
de Tejada <i>et al</i> ^[50] 2008	Male	47	Yes
Stefanescu <i>et al</i> ^[51] 2008	Male	15	No
Varma <i>et al</i> ^[53] 2008	Female	23	NR
Galloro <i>et al</i> ^[54] 2008	Male	44	Yes
Thomas <i>et al</i> ^[59] 2009	Male	43	NR
Levenick <i>et al</i> ^[61] 2009	Female	35	Yes
Levenick <i>et al</i> ^[61] 2009	Male	47	Yes
Levenick <i>et al</i> ^[61] 2009	Female	36	Yes
Levenick <i>et al</i> ^[61] 2009	Female	54	NR
Yoshida <i>et al</i> ^[62] 2009	Male	63	Yes
Meesiri ^[64] 2009	Male	44	Yes
Funamizu <i>et al</i> ^[65] 2009	Female	54	NR
Viñolo Ubiña <i>et al</i> ^[66] 2010	Male	40	Yes
Tezuka <i>et al</i> ^[67] 2010	Male	55	Yes
Lee <i>et al</i> ^[69] 2010	Male	75	NR
Egorov <i>et al</i> ^[70] 2010	Male	32	Yes
Egorov <i>et al</i> ^[70] 2010	Male	43	NR
German <i>et al</i> ^[72] 2010	Male	34	Yes
Pezzilli 2011 Present paper	Female	65	No
Pezzilli 2011 Present paper	Male	49	Yes
Pezzilli 2011 Present paper	Male	56	No
Overall	Males: 40 (81.6%) Females: 9 (18.4%)	45.3 ± 15.2	29/36 (80.50%)

NR: Not reported.

CLINICAL AND BIOCHEMICAL FEATURES

As shown in Tables 1-8, the main symptoms of cystic dystrophy of the duodenal wall were epigastric pain, weight loss and jaundice. These symptoms were similar in those patients having associated chronic groove pancreatitis and in those patients having groove carcinoma. All these symptoms can be present, further complicating

the differential diagnosis with ampullary and periampullary cancers. Pain may be persistent or recurrent, and nausea and vomiting are usually present as accompanying symptoms. The majority of these patients are heavy alcohol drinkers (275/341, 80.6%), and this may explain the fact that most of the patients with groove pancreatitis are males. In addition, in the 18 patients with groove adenocarcinoma, the majority of cases were males (11/18, 61.1%) (Tables 9-12).

Regarding the laboratory examinations, serum amylase activity was usually abnormally high in these patients (38/59, 64.4%) (Tables 1-8), but the magnitude of this elevation varied greatly. An increase in bilirubin may have also been present, along with an increase in alkaline phosphatases in patients with jaundice. Finally, it has also been reported in the literature that tumor markers, such as serum CA 19-9, are usually within the normal limits^[10,58].

ASSOCIATED DISEASES

The majority of patients with cystic dystrophy of the duodenal wall have been reported to have chronic groove pancreatitis or groove carcinoma. However, the lesions in the remaining pancreatic gland not affected by groove pancreatitis have not been fully evaluated. As shown in Tables 1-12, in patients with groove pancreatitis as well as in those with groove carcinoma, the pancreatic gland above the groove lesion is generally not affected by chronic pancreatitis. Chronic pancreatitis of the entire pancreas was reported in 166 of the 302 (55.1%) patients and there were pancreatic calcifications in 125 of these 166 patients (75.3%) (Tables 1-8). The presence of pancreatic pseudocysts was usually rare (13 out of 378, 3.4%) (Tables 1-8), and, in most cases, they were localized in the head of the pancreas (7/13, 53.8%). In addition, some authors have reported that groove pancreatitis is associated with the occasional findings of neuroendocrine tumors^[63] or pancreatic cystadenoma^[54].

IMAGING ASSESSMENT

As shown in Tables 3, 7, 8 and 11, the imaging diagnosis of dystrophy of the duodenal wall is rarely assessed using a single radiological modality. Even if US is the first line imaging modality in these patients, it is rarely diagnostic. ERCP, which was frequently used in the past, is feasible and in typical cases it demonstrates smooth tubular stenosis at the distal part of the CBD without abnormality of the main pancreatic duct or, occasionally, with only slight irregularities^[44,65]. ERCP may also demonstrate irregularity, tapering obstruction or dilatation of the Santorini duct and its branches, sometimes with intraductal stones or protein plugs^[44]. At present, ERCP is used mainly for endoscopic therapy^[33]; in fact, successful treatment for groove pancreatitis by endoscopic drainage *via* the minor papilla was carried out in only one patient^[33].

Table 6 Epidemiological and clinical characteristics of patients in the 38 case report papers involving 46 subjects with a benign cystic duodenal wall (a paper may report more than one patient), the three cases reported in the present paper are also shown (continues from Table 5)

Author ^[Ref.] yr	Time interval from the onset of symptoms to diagnosis	Abdominal pain	Weight loss	Jaundice	Hyperamylasemia
Bill <i>et al</i> ^[74] 1982	NR	Yes (Persistent)	Yes	No	No
Holstege <i>et al</i> ^[75] 1985	6 mo	Yes (Persistent)	Yes	No	Yes
Tio <i>et al</i> ^[9] 1991	NR	Yes (NR)	No	No	NR
Tio <i>et al</i> ^[9] 1991	NR	Yes (NR)	No	Yes	NR
Flaherty <i>et al</i> ^[76] 1992	NR	Yes (NR)	No	No	NR
Izbicki <i>et al</i> ^[77] 1994	NR	Yes (Recurrent)	No	No	No
Fujita <i>et al</i> ^[14] 1997	NR	Yes (Recurrent)	Yes	No	No
Shudo <i>et al</i> ^[17] 1998	NR	Yes (Persistent)	No	No	Yes
Wu <i>et al</i> ^[78] 1998	10 yr	Yes (Recurrent)	Yes	No	NR
Babál <i>et al</i> ^[79] 1998	NR	No	No	No	NR
Rubay <i>et al</i> ^[21] 1999	7 yr	Yes (Recurrent)	Yes	No	Yes
Balachandar <i>et al</i> ^[22] 1999	NR	No	No	Yes	No
Mohl <i>et al</i> ^[25] 2001	1 yr	Yes (Recurrent)	Yes	No	NR
Mohl <i>et al</i> ^[25] 2001	1 yr	Yes (Persistent)	Yes	No	NR
Munthali Lovemore <i>et al</i> ^[26] 2001	NR	Yes (Persistent)	NR	Yes	Yes
Indinnimeo <i>et al</i> ^[27] 2001	10 yr	Yes (Recurrent)	No	No	Yes
Shudo <i>et al</i> ^[28] 2002	NR	Yes (Persistent)	No	No	Yes
Glaser <i>et al</i> ^[29] 2002	NR	Yes (Persistent)	Yes	No	No
Hwang <i>et al</i> ^[31] 2003	NR	Yes (Persistent)	Yes	No	Yes
Jovanovic <i>et al</i> ^[32] 2004	NR	Yes (Persistent)	Yes	No	Yes
McFaul <i>et al</i> ^[80] 2004	13 mo	Yes (Recurrent)	Yes	No	NR
McFaul <i>et al</i> ^[80] 2004	2 yr	Yes (Recurrent)	Yes	Yes	NR
Isayama <i>et al</i> ^[33] 2005	2 yr	Yes (Persistent)	Yes	No	No
Chatelain <i>et al</i> ^[34] 2005	1 yr	Yes (Recurrent)	Yes	No	No
Chatelain <i>et al</i> ^[34] 2005	NR	Yes (Persistent)	Yes	No	No
Balzan <i>et al</i> ^[35] 2005	2 yr	Yes (Persistent)	No	No	Yes
Sanada <i>et al</i> ^[43] 2007	NR	Yes (Persistent)	No	No	Yes
Balakrishnan <i>et al</i> ^[44] 2007	NR	Yes (Persistent)	Yes	No	Yes
de Tejada <i>et al</i> ^[50] 2008	2 mo	Yes (Persistent)	Yes	No	NR
Stefanescu <i>et al</i> ^[51] 2008	5 mo	Yes (Persistent)	Yes	No	NR
Varma <i>et al</i> ^[53] 2008	3 mo	Yes (Persistent)	Yes	No	NR
Galloro <i>et al</i> ^[54] 2008	NR	Yes (Recurrent)	Yes	No	Yes
Thomas <i>et al</i> ^[59] 2009	NR	Yes (NR)	Yes	No	NR
Levenick <i>et al</i> ^[61] 2009	NR	Yes (Recurrent)	NR	No	NR
Levenick <i>et al</i> ^[61] 2009	NR	Yes (Recurrent)	Yes	No	NR
Levenick <i>et al</i> ^[61] 2009	NR	Yes (Recurrent)	Yes	No	NR
Levenick <i>et al</i> ^[61] 2009	NR	No	Yes	No	NR
Yoshida <i>et al</i> ^[62] 2009	NR	Yes (Persistent)	No	No	Yes
Meesiri ^[64] 2009	NR	Yes (Recurrent)	No	No	Yes
Funamizu <i>et al</i> ^[65] 2009	NR	Yes (Persistent)	No	Yes	Yes
Viñolo Ubiña <i>et al</i> ^[66] 2010	3 mo	Yes (Persistent)	No	No	Yes
Tezuka <i>et al</i> ^[67] 2010	NR	Yes (NR)	No	No	Yes
Lee <i>et al</i> ^[69] 2010	NR	Yes (Recurrent)	No	No	Yes
Egorov <i>et al</i> ^[70] 2010	2 mo	Yes (Persistent)	Yes	No	Yes
Egorov <i>et al</i> ^[70] 2010	1 yr	Yes (Persistent)	Yes	Yes	No
German <i>et al</i> ^[72] 2010	NR	Yes (Recurrent)	Yes	No	Yes
Pezzilli 2011 Present paper	1 yr	Yes (Recurrent)	Yes	Yes	No
Pezzilli 2011 Present paper	7 mo	Yes (Persistent)	Yes	No	Yes
Pezzilli 2011 Present paper	1 mo	Yes (Persistent)	Yes	Yes	Yes
Overall	2.1 ± 3.1 yr	46/49 (93.90%)	30/47 (63.80%)	8/49 (16.30%)	22/33 (66.70%)

CBD: Common bile duct; NR: not reported.

For many years, CT has been an excellent imaging modality for diagnosing chronic pancreatitis or adenocarcinoma associated with cystic dystrophy of the duodenal wall^[6,16]. In the pure form of groove pancreatitis, it may be visualized as a poorly enhancing hypodense lesion between the pancreatic head and the duodenum, near the minor papilla, reflecting the pathological characteristics of the mass. The delayed enhancement is mainly

due to delayed blood circulation caused by fibrous tissue proliferation and artery constriction^[12]. In addition, CT may reveal the presence of duodenal stenosis with wall thickening and cystic lesions in the duodenal wall or in the groove area. The cysts may be tiny even if multilocular cystic lesions may be observed. The main pancreatic duct may be mildly dilated above the lesion while, in the pure form, paraduodenal pancreatitis can be expected.

Table 7 Epidemiological and clinical characteristics of patients in the 38 case report papers involving 46 subjects with a benign cystic duodenal wall (a paper may report more than one patient), the three cases reported in the present paper are also shown (continues from Table 6)

Author ^[Ref.] yr	Imaging	Duodenal findings	CBD stenosis	Duodenal stenosis
Bill <i>et al</i> ^[74] 1982	US, ERCP, angiography	NR	Yes	No
Holstege <i>et al</i> ^[75] 1985	US, CT, ERCP	Severe erosive gastritis + bulging of the duodenum	No	Yes
Tio <i>et al</i> ^[9] 1991	ERCP, EUS, US	Polypoid lesion	Yes	Yes
Tio <i>et al</i> ^[9] 1991	ERCP, EUS, US	NR	No	Yes
Flaherty <i>et al</i> ^[73] 1992	US	No	No	No
Izbicki <i>et al</i> ^[77] 1994	US, angiography, ERCP	NR	Yes	Yes
Fujita <i>et al</i> ^[14] 1997	US, CT, ERCP	Inflammation	No	Yes
Shudo <i>et al</i> ^[17] 1998	CT, US, ERCP, EUS, celiac angiography	Edema duodenal wall. Brunner hyperplasia	No	Yes
Wu <i>et al</i> ^[78] 1998	CT	NR	No	No
Babál <i>et al</i> ^[79] 1998	NR	NR	No	No
Rubay <i>et al</i> ^[21] 1999	CT, ERCP, MRI, EUS	No alterations	NR	Yes
Balachandar <i>et al</i> ^[22] 1999	CT, ERCP	No duodenal alteration	Yes	No
Mohl <i>et al</i> ^[25] 2001	CT	Stenosis	No	Yes
Mohl <i>et al</i> ^[25] 2001	US, CT, ERCP	Normal duodenal mucosa	No	No
Munthali Lovemore <i>et al</i> ^[26] 2001	US, CT, ERCP	NR	Yes	No
Indinnimeo <i>et al</i> ^[27] 2001	CT, MRI, EUS	No alterations	No	No
Shudo <i>et al</i> ^[28] 2002	CT, US, ERCP, EUS, celiac angiography	Irregular polypoid bulging; inflammation of the mucosa	NR	Yes
Glaser <i>et al</i> ^[29] 2002	US	Severe deformation + inflammatory changes	No	Yes
Hwang <i>et al</i> ^[31] 2003	US, CT, MRI	Duodenal inflammation, duodenal stenosis	NR	Yes
Jovanovic <i>et al</i> ^[32] 2004	US, CT, EUS, MRI	Stenosis	No	Yes
McFaul <i>et al</i> ^[80] 2004	US, CT, MRI	Brunner hyperplasia	Yes	Yes
McFaul <i>et al</i> ^[80] 2004	US, PET-CT	Brunner hyperplasia	No	No
Isayama <i>et al</i> ^[33] 2005	CT, EUS, MRCP, ERCP	NR	No	Yes
Chatelain <i>et al</i> ^[34] 2005	EUS, CT	Duodenal stenosis, inflammation	No	Yes
Chatelain <i>et al</i> ^[34] 2005	EUS, CT	Duodenal stenosis	No	Yes
Balzan <i>et al</i> ^[35] 2005	US, MRI, CT	NR	NR	NR
Sanada <i>et al</i> ^[43] 2007	CT, ERCP	Edema duodenal wall. Brunner hyperplasia	Yes	No
Balakrishnan <i>et al</i> ^[44] 2007	CT, ERCP, EUS	Edematous, shiny, reddish raise mucosa with polypoid appearance; Brunner hyperplasia	No	No
de Tejada <i>et al</i> ^[50] 2008	MRI, EUS	Bulging, Brunner hyperplasia	No	No
Stefanescu <i>et al</i> ^[51] 2008	CT, EUS	NR	No	Yes
Varma <i>et al</i> ^[53] 2008	US, CT	Brunner hyperplasia	No	No
Galloro <i>et al</i> ^[54] 2008	US, CT, EUS	Duodenal stenosis	No	Yes
Thomas <i>et al</i> ^[59] 2009	US, CT, EUS, octreotide scan	Brunner hyperplasia	No	Yes
Levenick <i>et al</i> ^[61] 2009	EUS, MRCP	Duodenal stenosis	No	Yes
Levenick <i>et al</i> ^[61] 2009	CT, EUS	Duodenal inflammation, duodenal stenosis	NR	Yes
Levenick <i>et al</i> ^[61] 2009	CT, EUS	Edema with acute and chronic inflammation	No	Yes
Levenick <i>et al</i> ^[61] 2009	CT, EUS, ERCP	NR	Yes	No
Yoshida <i>et al</i> ^[62] 2009	CT, MRCP	Normal mucosa	No	Yes
Meesiri ^[64] 2009	US, CT, MRI	Edema and hemorrhagic mucosa with inflammation	NR	No
Funamizu <i>et al</i> ^[65] 2009	ERCP, CT, angiography	NR	Yes	No
Viñolo Ubiñuet <i>et al</i> ^[66] 2010	CT	Stenosis	NR	Yes
Tezuka <i>et al</i> ^[67] 2010	CT, ERCP	Edema duodenal wall	No	Yes
Lee <i>et al</i> ^[69] 2010	CT, MRCP	Active ulcer	Yes	No
Egorov <i>et al</i> ^[70] 2010	US, CT, EUS	Deformation, infiltration and ulcer; Inflammation	No	Yes
Egorov <i>et al</i> ^[70] 2010	US, CT, MRI, EUS	NR	Yes	Yes
German <i>et al</i> ^[72] 2010	US, CT, MRI	Edema duodenal wall; Brunner hyperplasia	Yes	Yes
Pezzilli 2011 Present paper	US, CT, EUS, ERCP	No	Yes	No
Pezzilli 2011 Present paper	US, CT, MRI	Hypertrophy of the Brunner glands	No	No
Pezzilli 2011 Present paper	US, CT	No	Yes	No
Overall	-	-	14/42 (33.30%)	28/48 (58.30%)

CBD: Common bile duct; NR: Not reported; US: Transabdominal ultrasonography; CT: Computer tomography; MRI: Magnetic resonance imaging; EUS: Endoscopic ultrasonography; ERCP: Endoscopic retrograde cholangiopancreatography; MRCP: Magnetic resonance cholangiopancreatography; PET-CT: Positron emission tomography with associated computer tomography.

Table 8 Epidemiological and clinical characteristics of patients in the 38 case report papers involving 46 subjects with a benign cystic duodenal wall (a paper may report more than one patient), The three cases reported in the present paper are also shown (continues from Table 7)

Author ^[Ref.] yr	Surgery	Type of surgery	Endoscopic treatment	Associated chronic pancreatitis	Associated neoplasms	Pseudocyst	Follow-up	Death
Bill <i>et al</i> ^[74] 1982	Yes	PD	No	NR	No	No	No	NR
Holstege <i>et al</i> ^[75] 1985	Yes	WP	No	No	No	No	No	NR
Tio <i>et al</i> ^[9] 1991	No	Derivative surgery	No	No	No	No	7 yr	No
Tio <i>et al</i> ^[9] 1991	Yes		No	No	No	No	6 mo	NR
Flaherty <i>et al</i> ^[75] 1992	Yes		No	No	No	No	9 mo	No
Izbicki <i>et al</i> ^[77] 1994	Yes		No	No	No	No	6 yr	No
Fujita <i>et al</i> ^[14] 1997	Yes	PD	No	No	No	No	3 yr	No
Shudo <i>et al</i> ^[17] 1998	Yes	PD	No	No	No	No	NR	NR
Wu <i>et al</i> ^[78] 1998	Yes	WP	No	No	No	No	9 mo	No
Babál <i>et al</i> ^[29] 1998	No		No	No	No	No	NR	During hospitalization
Rubay <i>et al</i> ^[21] 1999	Yes	PD	No	No	No	No	2 mo	No
Balachandar <i>et al</i> ^[22] 1999	Yes	Derivative	No	Yes	No	No	NR	NR
Mohl <i>et al</i> ^[25] 2001	Yes	PD	No	No	No	No	No	NR
Mohl <i>et al</i> ^[25] 2001	Yes	PD	No	No	No	No	4 wk after surgery	No
Munthali Lovemore <i>et al</i> ^[26] 2001	Yes	Derivative CBD	No	No	No	No	No	NR
Indinnimeo <i>et al</i> ^[27] 2001	Yes	PD	No	No	No	No	2 yr	No
Shudo <i>et al</i> ^[28] 2002	Yes	PD	No	No	No	No	NR	NR
Glaser <i>et al</i> ^[29] 2002	No		No	No	No	No	No	NR
Hwang <i>et al</i> ^[31] 2003	No		No	No	No	No	NR	NR
Jovanovic <i>et al</i> ^[32] 2004	Yes	PD	No	No	No	No	No	NR
McFaul <i>et al</i> ^[80] 2004	Yes	PD	No	Yes	No	No	2 yr	No
McFaul <i>et al</i> ^[80] 2004	Yes	WP	No	Yes	No	No	NR	No
Isayama <i>et al</i> ^[33] 2005	No		Yes	No	No	No	12 mo	No
Chatelain <i>et al</i> ^[34] 2005	Yes	PD	No	No	No	No	6 mo	No
Chatelain <i>et al</i> ^[34] 2005	Yes	PD	No	No	No	No	12 mo	No
Balzan <i>et al</i> ^[35] 2005	Yes	PD	No	Yes	No	Yes (head)	No	NR
Sanada <i>et al</i> ^[43] 2007	Yes	PD	No	No	No	Yes (head)	No	NR
Balakrishnan <i>et al</i> ^[44] 2007	Yes	Laparotomy	No	Yes	No	No	NR	NR
de Tejada <i>et al</i> ^[50] 2008	Yes	WP	No	No	No	No	3 mo	No
Stefanescu <i>et al</i> ^[51] 2008	Yes	Derivative	No	No	No	No	8 mo	No
Varma <i>et al</i> ^[53] 2008	Yes	WP	No	No	No	No	9 mo	No
Galloro <i>et al</i> ^[54] 2008	Yes	WP	No	Yes (with calcifications)	Cystadenoma	Yes	14 mo	No
Thomas <i>et al</i> ^[59] 2009	Yes	PD	No	No	No	No	NR	NR
Levenick <i>et al</i> ^[61] 2009	Yes	PD	No	No	No	No	3 yr	No
Levenick <i>et al</i> ^[61] 2009	Yes	PD	No	Yes	No	No	NR	NR
Levenick <i>et al</i> ^[61] 2009	Yes	PD	No	No	No	No	NR	NR
Levenick <i>et al</i> ^[61] 2009	Yes	PD	No	No	No	No	NR	NR
Yoshida <i>et al</i> ^[62] 2009	Yes	PD	No	No	No	No	Yes (time NR)	No
Meesiri ^[64] 2009	No		No	No	No	No	Yes (time NR)	No
Funamizu <i>et al</i> ^[65] 2009	Yes	PD	No	No	Yes	No	15 mo	No
Viñolo Ubiña <i>et al</i> ^[66] 2010	Yes	PD	No	No	No	No	NR	No
Tezuka <i>et al</i> ^[67] 2010	Yes	PD	No	No	No	No	NR	No
Lee <i>et al</i> ^[69] 2010	No		No	No	No	No	NR	NR
Egorov <i>et al</i> ^[70] 2010	Yes	Pancreas-preserving duodenal resection	No	No	No	No	6 mo	No
Egorov <i>et al</i> ^[70] 2010	Yes	Pancreas-preserving duodenal resection	No	No	No	No	5 mo	No
German <i>et al</i> ^[72] 2010	Yes	PD	No	No	No	No	2 mo	NR
Pezzilli 2011 Present paper	No		Yes	No	No	No	20 mo	No
Pezzilli 2011 Present paper	Yes	PD	No	No	No	No	7 mo	No
Pezzilli 2011 Present paper	Yes	PD	No	Autoimmune pancreatitis	Yes	No	4 mo	No
Overall	41/49 (83.70%)	-	2/49 (4.10%)	8/48 (16.70%)	3/49 (6.10%)	3/49 (6.10%)	17.9 ± 20.6 mo	1/28 (3.60%)

CBD: Common bile duct; NR: Not reported; PD: Pancreaticoduodenectomy; WP: Whipple procedure.

In groove pancreatitis and in groove carcinoma, the CBD may be stenosed in its distal part and a dilation of the extra- and intra-hepatic biliary system can be observed^[6,16].

The same CT findings can also be observed when utilizing MRI which may reveal a mass between the head of the pancreas and the duodenum associated with duodenal wall thickening. The mass visualized in the groove

Table 9 Epidemiological and clinical characteristics of patients in the two retrospective studies and two case report papers involving two subjects with groove adenocarcinoma

Author ^[Ref.] yr	Type of study	Time interval of patient enrollment	No. of patients			Age (yr) Mean (range)	Alcohol drinkers
			Total	Males	Females		
Suehara <i>et al</i> ^[20] 1998	Case report	1995	1	1	-	61	Yes
Gabata <i>et al</i> ^[30] 2003	Retrospective	1998-2001	9	4 (44.4%)	5 (55.6%)	72 (56-87)	NR
Tan <i>et al</i> ^[38] 2006	Case report	NR	1	-	1	69	NR
Ishigami <i>et al</i> ^[6] 2010	Retrospective	2001-2008	7	6 (85.7%)	1 (14.3%)	70 (57-80)	NR

NR: Not reported.

Table 10 Epidemiological and clinical characteristics of patients in the two retrospective studies and two case report papers involving two subjects with groove adenocarcinoma (continues from Table 9)

Author ^[Ref.] yr	Abdominal pain	Weight loss	Jaundice	Hyperamylasemia
Suehara <i>et al</i> ^[20] 1998	Yes (Persistent)	No	Yes	Yes
Gabata <i>et al</i> ^[30] 2003	NR	NR	NR	NR
Tan <i>et al</i> ^[38] 2006	Yes (Persistent)	Yes	Yes	Yes
Ishigami <i>et al</i> ^[6] 2010	NR	NR	NR	NR

NR: Not reported.

Table 11 Epidemiological and clinical characteristics of patients in the two retrospective studies and two case report papers involving two subjects with groove adenocarcinoma (continues from Table 10)

Author ^[Ref.] yr	Imaging	Duodenal findings	CBD stenosis	Duodenal stenosis
Suehara <i>et al</i> ^[20] 1998	US, EUS, CT, MRI, angiography	NR	Yes	No
Gabata <i>et al</i> ^[30] 2003	CT, RMI, ERCP, angiography	Edema with erosions	9 (100%)	9 (100%)
Tan <i>et al</i> ^[38] 2006	US, MRI, ERCP	NR	Yes	No
Ishigami <i>et al</i> ^[6] 2010	CT, MRI	NR	7 (100%)	NR

CBD: Common bile duct; NR: Not reported; US: Transabdominal ultrasonography; CT: Computer tomography; MRI: Magnetic resonance imaging; EUS: Endoscopic ultrasonography; ERCP: Endoscopic retrograde cholangiopancreatography.

Table 12 Epidemiological and clinical characteristics of patients in the two retrospective studies and two case report papers involving two subjects with groove adenocarcinoma (continues from Table 11)

Author ^[Ref.] yr	Surgery		Associated chronic pancreatitis	Pseudocyst	Follow-up
	No. of cases	Type			
Suehara <i>et al</i> ^[20] 1998	Yes	PD	No	No	NR
Gabata <i>et al</i> ^[30] 2003	9 (100%)	PD in 7; derivative in 2	No	No	NR
Tan <i>et al</i> ^[38] 2006	Yes	By-pass surgery	No	No	NR
Ishigami <i>et al</i> ^[6] 2010	6 (85.7%)	PD in 5; derivative in 1	No	NR	NR

NR: Not reported; PD: Pancreaticoduodenectomy.

and/or in the adjacent head of the gland is hypointense

to the pancreatic parenchyma. Delayed enhancement may also be seen in the thickened duodenal wall. These imaging features reflect the fibrous involvement of the lesions of groove pancreatitis. Cysts, which may be present in the groove area and the duodenal wall, have high signal intensity. An important diagnostic aspect of MRI, which cannot be evaluated by CT, is the fact that MRI can be followed by magnetic resonance cholangiopancreatography (MRCP); this additional evaluation provides images similar to those of ERCP without the morbidity of this latter technique. In addition, MRCP may visualize those lesions which are not seen in ERCP in the case of serrated duodenal stenosis^[18]. The diagnostic value of MRI is superior to CT in evaluating biliary ducts in para-duodenal pancreatitis as well as in groove carcinomas. The stricture, or narrowing of the CBD, may be better approached by using MRCP rather than CT and/or ERCP. The dilation of the space comprising the main pancreatic duct, the CBD and the duodenum is another sign which can be observed in patients with groove pancreatitis or groove carcinoma when using MRCP^[47].

In the last few years, EUS has emerged as a useful technique for diagnosing pancreatic diseases because of the accurate evaluation of the biliopancreatic structures through the gastro-duodenal lumen without interference of the abdominal wall or other organs^[82]. EUS can easily demonstrate the hypoechoic area between the duodenal wall and the pancreatic parenchyma, narrowing of the duodenal lumen and stenosis of the CBD and/or pancreatic duct in both groove pancreatitis and groove carcinomas^[50]. Furthermore, the diagnosis can be confirmed by EUS-guided fine-needle aspiration of the mass visualized.

PATHOLOGY

Macroscopically, groove pancreatitis is associated with an absent or narrow Santorini duct or the presence of pancreas divisum^[17], and the difficult outflow of pancreatic fluid may be hypothesized for lesions of the groove similar to those of chronic pancreatitis^[17]. The duodenal wall contains dilated ducts, in the majority of cases with thickened secretions, pseudocystic changes as well as adjacent stromal reactions, foreign-body type giant cell reaction engulfing mucoprotein material and myofibroblastic proliferation. Brunner gland hyperplasia is usually present as is dense myoid stromal proliferation, with intervening rounded lobules of pancreatic acinar

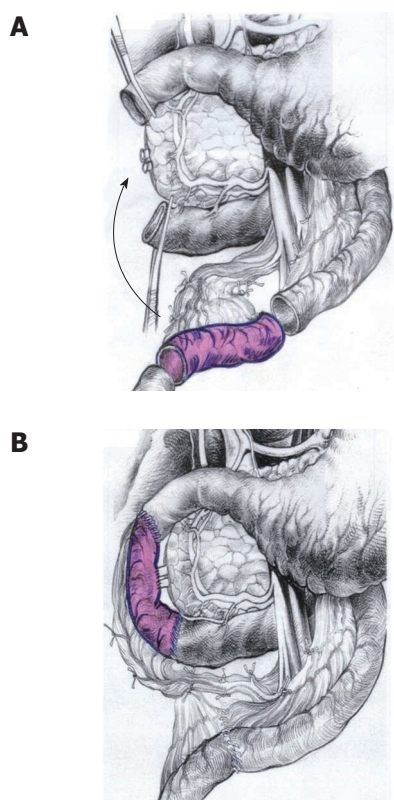


Figure 8 New surgical option for patients having cystic dystrophy of duodenal wall. A: Scheme of the pancreas-preserving resection of the second portion of the duodenum. The second part of the duodenum, including the main papilla, is removed and the segment of the proximal jejunum supplied by the artery and vein is cut out and prepared for transposition between the 1st and 3rd portions of the duodenum; B: The shifted segment is interposed between the 1st and the 3rd parts of the duodenum. Jejunum-jejunum- and duodenum-jejunum-anastomoses are performed. The bile and the pancreatic ducts were implanted in the neoduodenum 4 cm below the proximal duodenum-jejunum-anastomosis (from Egorov *et al.*^[70] with the kind permission of the authors).

tissue. Fibrosis into the adjacent pancreas and soft tissue occurs, especially in the groove area which involves the CBD^[5].

In groove carcinoma, the macroscopic pathology is similar to that of groove pancreatitis while the pancreatic tissue has the same histology as that of pancreatic adenocarcinoma^[6,20,30,38].

TREATMENT

Conservative treatment is the main option in the acute phase of the disease, including analgesia and parenteral nutrition. In some patients, enteral nutrition is not always possible due to the presence of duodenal stenosis^[17]. The main therapeutic option for these patients is a surgical approach in benign as well as in malignant diseases of the groove, as shown in Tables 1-12. **The most frequent surgical approach is a pylorus-preserving pancreaticoduodenectomy or a Whipple procedure;** in a limited number of patients, a gastrointestinal by-pass, with or without biliary by-pass, has been carried out. More recently, a new approach has been reported by Egorov *et al.*^[70];

these authors have described a new surgical approach carried out on two patients who were successfully treated by two modifications of a pancreas-preserving duodenal resection with reimplantation of the bile and pancreatic ducts into the neoduodenum (Figure 8). The authors have claimed that these two cases are a good example of a pancreas-preserving approach to duodenal dystrophy treatment and that the technique may be an alternative to the Whipple procedure in cases of mild changes of the orthotopic gland.

Only in a few cases was a medical approach carried out (see Case 1 of our three patients), mainly because the patients refused surgery, and also in one patient in whom successful treatment for groove pancreatitis was carried out by endoscopic drainage *via* the minor papilla^[33].

THE FATE OF PATIENTS

The first important question arising from the studies analyzed is the extreme length of time necessary from the onset of the symptoms to reach a diagnosis in patients with groove pancreatitis: it varies from a few days to ten years (Tables 1-8). **In one of the larger studies in this field**, such as that of Rebours *et al.*^[40], the mean time from the appearance of the symptoms and the diagnosis is 1 year with a range of 0 to 24 mo. This long time period is similar to that previously reported in chronic pancreatitis^[83]. In patients with groove adenocarcinoma, we have no information on time to diagnosis. The perioperative mortality rate seems to be negligible, the only death being reported by Babál *et al.*^[79]. In the only study reporting this information (Tables 1-8), **the mortality rate was 8.9% (9/101)** in the follow-up period in patients with benign disease. However, this information should be taken with caution because, as previously stated, the follow-up period is not quite as long in the majority of studies.

CONCLUSION

The diagnosis of cystic dystrophy of the duodenal wall can be easily assessed by MDCT, MRI and EUS. These latter two techniques may also add more information on the involvement of the part of the pancreatic gland not involved in the duodenal malformation.

Chronic pancreatitis involving the entire pancreatic gland is present in half the patients with cystic dystrophy of the duodenal wall, and the pancreatitis has calcifications in the majority of them. We have no information about exocrine function in these patients and this topic requires additional study. In subjects without pancreatitis, the patients with cystic dystrophy of the duodenal wall are usually in satisfactory general condition after surgical treatment and they regain weight after surgery.

The fact that only two children have been reported to have cystic dystrophy of the duodenal wall confirms the hypothesis that pancreatic and biliary diseases develop over a long period of time.

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