Groove Pancreatitis
A Brief Review of a Diagnostic Challenge

Karyn DeSouza, MD; Laurentia Nodit, MD

Groove pancreatitis is an underrecognized form of recurrent or chronic pancreatitis characterized by scarring in the “sliding plane,” between the duodenum and the head of the pancreas, with compression of blood vessels, tubular stenosis of the common bile duct, duodenal wall rigidity, and luminal narrowing, which can mimic peripancreatic or pancreatic carcinoma. It was first described by Potet and Duclert,1 in 1970, as cystic dystrophy of the periampullary or pancreatic carcinoma. It was first described, type of focal chronic pancreatitis, affecting “the groove”—the area between the head of the pancreas, the duodenum, and the common bile duct. Men aged 40 to 50 years are most commonly affected, with a history of alcohol abuse frequently disclosed. Clinical manifestations are similar to other forms of chronic pancreatitis, and vomiting secondary to duodenal stenosis is the main feature. It is postulated that pancreatitis in the groove area arises from obstruction of pancreatic juices in the ductal system, causing fibrosis and stasis with resultant inflammation of surrounding structures. The minor papilla is frequently the anatomic area of preferential involvement. Groove pancreatitis poses diagnostic challenges, forming a “pseudotumor” that mimics pancreatic carcinoma. The distinction is important, although often impossible to make because of their similar presentation, with groove pancreatitis usually affecting younger patients. Most patients are successfully treated with pancreaticoduodenectomy when definitive pathologic diagnoses can be made.


Groove pancreatitis (GP) is an underrecognized form of recurrent or chronic pancreatitis characterized by scarring in the “sliding plane,” between the duodenum and the head of the pancreas, with compression of blood vessels, tubular stenosis of the common bile duct, duodenal wall rigidity, and luminal narrowing, which can mimic peripancreatic or pancreatic carcinoma. It was first described by Potet and Duclert,1 in 1970, as cystic dystrophy of the duodenal wall developing in the heterotopic pancreas but was discussed almost exclusively in European literature. According to Stolte,2 in 1973, Becker3 had drawn attention to the existence of a localized chronic pancreatitis, using the term Rinnenpankreatitis, in the German literature, but Stolte et al2 brought the term groove pancreatitis to the English literature in 1982 with the addition of other histologic features, such as Brunner gland hyperplasia and common bile duct tubular stenosis. Various names for this entity have been used, including myoadenomatosis, cystic dystrophy of heterotopic pancreas, para-duodenal wall cyst, pancreatic hamartoma of duodenum, and paraduodenal pancreatitis,4 all of them reflecting certain elements that can be more prominent in some cases. Groove pancreatitis arises most commonly in middle-aged men with a known history of alcohol abuse and smoking; women and other age groups are rarely reported,5,6 and no increase in familial frequency has been observed.7 Tobacco and alcohol abuse are thought to cause viscid pancreatic juices, inducing stasis and outflow obstruction. That long-term irritation leads to fibrosis and calcification of the Santorini duct and minor papilla, the surrounding pancreatic head, and the adjacent duodenal wall. The stenotic duodenal wall and exocrine pancreatic dysfunction are the likely causes for the constellation of gastrointestinal symptoms and weight loss. In most cases, the main pancreatic duct system is normal.

The initial report by Becker and Mischke7 recognized 3 forms of the disease: a “pure” groove pancreatitis (2%), a segmental pancreatitis of the head with groove involvement (6.5%), and cases of chronic homogenous pancreatitis with groove involvement (not restricted to segmental spread) (11%)—which may explain the increased percentage of groove pancreatitis cases found in their series (19.5%; 117 of 600 specimens). In another study,2 the incidence of GP reached 24.4% (30 of 123) resections for chronic pancreatitis. That high incidence of the disease in earlier studies may be explained by the increased resection rate for those cases because of the severity of the symptoms and the clinical suspicion for malignancy. Later studies restricted the term groove pancreatitis to only the first 2 forms, which together with inconsistent terminology and a decreased resection rate may explain the rarity of GP, which is represented in recent literature mostly as case reports or studies of small series.5,6–12

**CLINICAL SYMPTOMS**

Most patients with GP complain of severe upper abdominal pain, nausea, and recurrent postprandial vomiting, which occurs for weeks to several years, with resulting weight loss.4 One of the characteristic manifestations is duodenal stenosis, which can be severe, and not infrequently, the patient presents with obstructive symptoms. Although tubular stenosis of the common bile duct is frequently described, obstructive jaundice is rare, with
delayed presentation compared with cases of pancreatic adenocarcinoma. Differentiating GP from pancreatic carcinoma in the clinical setting can be difficult because both pathologic processes present with similar clinical findings, radiologic features, and gross pathologic features, including marked scaring and ill-defined borders.13,14

Pathogenesis

The etiology of GP is likely heterogeneous, with the following variety of factors playing a role in its development:

- Anatomic disruption, such as the absence or functional obstruction of the minor papilla, because of thickening or scarring of the duodenal wall. Additionally, neoplasms15 in that area may cause disruption to the flow of pancreatic juice.

- Pancreatic heterotopia in the duodenal wall may be present because of incomplete involution of the dorsal pancreatic head,16 with secondary, localized inflammation, cicatrization, cystic dystrophy, and duct ectasia. Pancreatic divisum (presence of the fetal-type ductal drainage system—dorsal and ventral aspects of the pancreas drained by 2 separate ducts, which is retained in adulthood) is another consideration.4

- Chronic alcohol consumption leading to increased pancreatic fluid viscosity and increased intraductal pressure in the accessory duct with leakage into the groove is a contributing factor. Obstruction of the accessory pancreatic duct by protein deposition,9 calcifications, and neoplasms have been described.

- Obstruction or incompetence of the minor papilla, secondary to variable Brunner gland hyperplasia, may be involved. Elevated pancreatic enzyme levels and increased cholinergic tone leading to stasis of the fluid in the dorsal pancreas2 is a common finding in this entity.

- Secondary modifications of local anatomy because of gastrectomy, gastroduodenal ulcer,17 and biliary disease may also contribute.

DIAGNOSIS

Pancreatic enzymes levels (amylase and lipase) may be slightly or dramatically increased with some patients presenting with leukocytosis. Isolated elevation of either amylase or lipase has been reported, with amylase levels greater than 40 000.18 As with most nonneoplastic inflammatory pancreatic lesions, tumor marker levels (carcinoembryonic antigen and CA 19-9) in GP are usually normal. There are, however, reports of high levels of CA 19-9 and carcinoembryonic antigen18 in the literature.

Endoscopy shows luminal narrowing of the duodenum with edema and inflamed polypoid mucosa, with or without erosions. Duodenal mucosal biopsies are usually inconclusive. Endoscopic retrograde cholangiopancreatography demonstrates a patent Wirsung duct with the absence, irregularity, or stenosis of the Santorini duct. Unsuccessful attempts at endoscopic retrograde cholangiopancreatography are usually attributed to duodenal stenosis.

Computed tomography scans may show mural thickening of the duodenal wall or a hypodense, poorly enhancing, hypovascular mass between the pancreas and duodenum. Other findings include swelling of the pancreatic head and punctuate calcifications.14,19

Magnetic resonance imaging/magnetic resonance cholangiopancreatography correlate well with the computed tomography findings and may provide additional information regarding biliary or pancreatic duct dilation. Such imaging shows a hypointense mass correlating to the pancreatic tissue on T1-weighted images, an isointense or slightly hyperintense mass on T2-weighted images, and hyperintensity on short T1 inversion recovery sequence magnetic resonance imaging.19 In the absence of cystic changes within the lesion or duodenal wall, this radiologic method tends to be less helpful in differentiating GP from pancreatic cancer.14 On magnetic resonance cholangiopancreatography, GP demonstrates either no abnormality or smooth tapering of the common bile duct, rather than the abrupt, complete obstruction of pancreatic carcinomas. The main pancreatic duct is usually spared, and peripancreatic vessels may show displacement without encasement.10

Today, valuable information for diagnosis is collected via ultrasound endoscopy, which allows clearer imaging of the cystic lesions, disease extent, and localization of the inflammatory process. Clearer visualization of the hypoechoic area between the duodenal wall and the pancreatic parenchyma is achieved, but distinction between an inflammatory process and cancerous infiltration can remain a challenge.51,20

Cytology

Endoscopic ultrasound-guided fine-needle aspiration is essential in the management of pancreatic lesions, and there is limited information available regarding cytologic features of GP. According to Chute and Stelow,18 the most common findings are spindled stromal cells, foamy cells, and granular debris. Inflammatory cells, duodenal contaminants, and sheets of bland, loosely cohesive epithelial cells, with foamy cytoplasm and small, regular nuclei, consistent with Brunner glands may be seen.18 Although most often interpreted as negative for malignancy, the sampling of an area of spindled cells or Brunner gland hyperplasia can mimic neoplasia. This was the case for one of our patients, a 40-year-old woman, for which the endoscopic ultrasound-guided fine-needle aspiration showed a moderately cellular specimen with tissue fragments containing groups of atypical spindle cells (Figure 1), with plump, epithelioid nuclei; small nucleoli (Figure 2); slight pleomorphism; and nuclear membrane irregularities (Figure 3). Occasional mitotic figures were seen, and background acellular, necrotic debris (Figure 4) was appreciated. The findings were interpreted as suspicious for neoplasm. Other reports also highlight pitfalls in cytologic interpretation of these samples, which can be concerning for malignancy.6,8

GROSS EXAMINATION

Gross inspection is important in diagnosis, with emphasis on documentation of the pathologic changes centered in the duodenal wall near the minor papilla. Usually, there is some degree of thickening and scarring of the duodenal wall, with luminal stenosis, giant duodenal mucosal folds, and cystic changes.4 The cysts embedded in the duodenal wall contain clear fluid, necrotic and granular material, or stones and may achieve large sizes, explaining the designation of parampullary wall cyst still used today.21 The scarring process is focused on the “groove area” and may spill into the adjacent pancreatic tissue, but diffuse pancreatic parenchymal fibrosis is not consistent with GP. Characteristic firmness of the head of the pancreas, stenosis of the bile duct, and thickening of the second part of the duodenal wall are
common; however, patency of the accessory pancreatic duct is not usually demonstrable.

In the late stages of the disease, the pancreatic duct system may be altered by inflammation and fibrotic change, with thickening of the adjacent duodenal wall and subsequent luminal stenosis. Fibrosis may lead to dilation of the ampulla of Vater and the main pancreatic duct, both of which are common gross features.

The minor papilla and the Santorini duct may be absent or partially to completely plugged by calcified, proteinaceous material, leading to narrowing or obliteration of the lumen.9 Most often, that diagnosis cannot be confidently rendered until thorough histologic examination has been performed.

**HISTOLOGIC FEATURES**

The key histologic criteria include (1) dilated ducts and pseudocystic changes in the duodenal wall, (2) duodenal submucosal fibrosis extending to the adjacent soft tissue in the groove area and pancreas, and (3) variable Brunner gland hyperplasia forming a thick layer with surrounding smooth muscle and myofibroblastic proliferation.

The cystic changes are visible in the duodenal wall, traversing the muscularis propria, and superficially reaching

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**Figure 1.** Cytologic features of groove pancreatitis on endoscopic ultrasound fine-needle aspirate. One atypical group of spindle cells is demonstrated (Papanicolaou, original magnification ×20).

**Figure 2.** Cytologic features of groove pancreatitis on endoscopic ultrasound fine-needle aspirate. Higher-power view (than in Figure 1) of spindle cells with plump, epithelioid nuclei, and small nucleoli. One mitotic figure is present in the upper-right corner (Papanicolaou, original magnification ×40).

**Figure 3.** Cytologic features of groove pancreatitis on endoscopic ultrasound fine-needle aspirate. High-power view in cellular groups shows marked nuclear membrane irregularities (Papanicolaou, original magnification ×60).

**Figure 4.** Cytologic features of groove pancreatitis on endoscopic ultrasound fine-needle aspirate. Background of proteinaceous debris (Papanicolaou, original magnification ×20).
the pancreatic parenchyma (Figure 5). The myofibroblastic proliferation may encase areas of cystic change with inspissated secretions and corresponds to the spindle cells identified on cytology samples (Figure 6). High cellularity, nuclear atypia, and occasional mitotic figures can be present and can be a pitfall in interpretation (Figure 7). The cystic spaces are sometimes lined by pancreatobiliary, columnar epithelium or granulation tissue, and in some cases, the lining is completely denuded. These cysts are filled with inspissated mucoprotein substance or acellular debris, suggesting pseudocysts, and can rupture to induce a foreign-body giant cell reaction. The remaining pancreatic parenchyma, distal bile duct, and distal gastric tissue can be entirely unremarkable or show secondary changes because of alcohol consumption. Chronic inflammation may be present but is usually not a prominent feature. Entrapped, ectopic pancreatic elements can be seen.

IMMUNOHISTOCHEMISTRY

The florid spindle cell proliferation can present diagnostic challenges given the anatomic location of this lesion. Ruling out a gastrointestinal stromal tumor or a vascular neoplasm can be facilitated by immunohistochemistry. The myofibroblastic cells in GP are immunoreactive to smooth muscle markers and nonreactive to CD117 and to vascular markers like CD34. Rare cases of pancreatic schwannoma have been reported in this region and can be ruled out with antibodies to S100.

DIFFERENTIAL DIAGNOSIS

Despite modern imaging techniques, the diagnosis of GP remains difficult, especially when inflammatory changes suggest a tumor/mass. The main differential diagnosis includes pancreatic adenocarcinoma and ampullary/duodenal neoplasms occurring in the same anatomic area. Diagnostic challenges often arise because of the striking similarity in their clinical presentation and overlapping radiologic and endoscopic features to include fibrosis, with or without enlargement of the pancreatic head, and a thickened adjacent duodenal wall. Distinction between GP and pancreatic adenocarcinoma has important clinical implications and can only be accurately made after surgical resection in most cases. To further complicate the matter, patients with concomitant GP and pancreatic adenocarcinoma have been described. When careful consideration of clinical history, imaging studies, serology, and cytologic features leads to a high suspicion for GP, conservative treatment is attempted. Autoimmune pancreatitis also enters in the differential diagnosis. The dense, plasma cell–rich inflammation centered on the pancreatic ducts, along with venulitis, can extend into the peripancreatic adipose tissue and create a clinical picture of a pseudotumor. Cystic tumors of the pancreatic head and acute relapsing pancreatitis are also included in the differential diagnosis.

TREATMENT

Abstinence from alcohol and tobacco, rest, and opioid analgesics are the most frequently used conservative

Figure 5. Histologic features of groove pancreatitis. Irregular, cysts in the groove area are filled with proteinaceous material in relation to the duodenum (right side) and the pancreas (left side) (hematoxylin-eosin, original magnification ×2).

Figure 6. Histologic features of groove pancreatitis. Myofibroblastic proliferation surrounding cystic areas corresponds to spindle cells identified on cytology aspirates (hematoxylin-eosin, original magnification ×20).

Figure 7. Histologic features of groove pancreatitis. Occasional atypical mitotic figures are seen in the myofibroblastic proliferation (hematoxylin-eosin, original magnification ×40).
measures. Notwithstanding, surgery is commonly the treatment of choice for symptomatic patients, with resultant decreased pain, decreased opioid dependence, increased body weight, and alleviation of abdominal pain. The Whipple procedure is the definitive cure for disease limited to the groove area. One report described curing GP by endoscopic drainage of minor papilla, which may be an effective treatment in a subset of patients; however, in another center’s experience, the relief was temporary because new cystic areas developed, and the patients eventually needed surgical therapy with pancreaticoduodenectomy. These medical and endoscopic therapies can be considered as “bridge” treatments before definitive surgery could be performed.

CONCLUSIONS

Regardless of the terminology employed, GP is a distinct form of chronic pancreatitis associated with minor pancreatic duct dysfunction and exposure to alcohol. Its accent is on the duodenal wall and can masquerade clinically as periampullary or pancreatic head carcinoma. Correct preoperative identification depends on knowledge of clinical, radiologic, and cytologic features. Traditional or pylorus-preserving pancreaticoduodenectomy usually leads to resolution of clinical symptoms and is the treatment of choice.

Clinical and pathologic features of GP are summarized in the Table.