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Published In/Presented At

Sullivan, M., Brodsky, J., Shah, S., Shah, H. (2015, October). *In The Groove: A Case Series of Groove Pancreatitis*. Poster presented at: American College of Gastroenterology Annual Scientific Meeting, Honolulu, HI.

Sullivan, M., Brodsky, J., Shah, S., Shah, H. (2016, April 26). *In The Groove: A Case Series of Groove Pancreatitis*. Poster presented at: Poster presented at LVHN Department of Medicine Research Day, Lehigh Valley Health Network, Allentown, PA.

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In The Groove: A Case Series of Groove Pancreatitis

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Background

- Groove pancreatitis is an under recognized form of focal chronic pancreatitis which has also been referred to in the literature as paraduodenal pancreatitis, pancreatic hamartoma of the duodenum, cystic dystrophy of heterotopic pancreas, paraduodenal wall cyst, and myoadenomatosis.¹
- First described in 1970, the pancreatic groove is a theoretical space bounded by the second portion of the duodenum laterally, the pancreatic head medially, the first portion of the duodenum or gastric antrum anteriorly, and the third portion of the duodenum or inferior vena cava posteriorly.¹

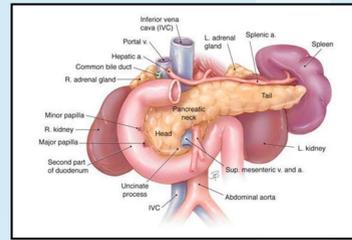


Figure 1. Anatomy of the pancreas and surrounding structures. Image by Jennifer Parsons Brumbaugh used with permission from source website.²

- The proposed pathogenesis involves disruption of flow through the accessory pancreatic duct creating a localized inflammatory response. Chronic alcohol use, anatomic variants, and possibly genetic susceptibility may also contribute.¹ It has also been shown that chronic alcohol use and smoking can increase the viscosity of pancreatic juice which can predispose the patient to stasis and outflow obstruction.³
- Presentation can be similar to classical chronic pancreatitis, but typically with more pronounced post-prandial nausea and vomiting and weight loss.¹
- Esophagogastroduodenoscopy (EGD) and endoscopic retrograde cholangiopancreatography (ERCP) can be helpful to rule out other etiologies. Biopsy is often required and should demonstrate myofibroblast proliferation and Brunner gland hyperplasia. Fine needle aspiration (FNA) will typically reveal benign or atypical inflammatory cells.¹
- Initial treatment consists of pancreatic rest, pain control, and abstinence from tobacco and alcohol. However, surgery is often required due to recurrent, severe symptoms and the need to rule out malignancy.¹
- There are many surgical options for chronic pancreatitis including both drainage and resection.⁴ In groove pancreatitis, Pancreaticoduodenectomy has found a place as the definitive cure³ and has been shown to decrease pain and opioid dependence and result in increased body weight.⁵

Case Presentations

Case 1: A 45 year-old male smoker and chronic alcohol abuser was admitted with his seventh episode of pancreatitis in two years. Prior imaging revealed changes of chronic pancreatitis and enhancement of a multiloculated pseudocyst in the region of the pancreatic head (Image 1), diffuse pancreatic duct (PD) distention (Image 2), dilation of intra- and extrahepatic bile ducts, and duodenal wall thickening. On this admission magnetic resonance cholangiopancreatography (MRCP) demonstrated dilation of both the common bile duct (CBD) and PD. CT scan revealed signs of gastric outlet obstruction (GOO) with duodenal thickening which an endoscope was unable to traverse. EGD and endoscopic ultrasound (EUS) were unable to exclude pancreatic malignancy and he subsequently underwent pancreaticoduodenectomy. Pathology demonstrated chronic pancreatitis, Brunner gland hyperplasia, and chronic duodenitis, but no evidence of malignancy. The pathology report suggested a diagnosis of groove pancreatitis.



Image 4. CT scan of the abdomen demonstrating the abnormal appearing pancreatic head and duodenum leading to obstruction of the CBD and PD.

Case 3: A 66 year-old male former smoker with a family history of pancreatic cancer in his father presented with one year of intermittent bloating, worsening post-prandial abdominal pain, early satiety, nausea, vomiting, and a 20 pound weight loss over the previous 3 months. Imaging revealed GOO due to an enhancing mass in the second portion of the duodenum, a cystic lesion in the pancreatic head, and several mildly enlarged lymph nodes. EGD revealed a large circumferential polypoid mass in the duodenal bulb and biopsy demonstrated prominent Brunner glands (Image 5). He was scheduled for pancreaticoduodenectomy, but at time of surgery a firm pancreas with inflammatory changes lead to the decision to perform a gastrojejunostomy and leave the duodenal lesion in situ. Pathology was negative for malignancy and again revealed Brunner gland hyperplasia. His condition was felt to be consistent with groove pancreatitis. He has been followed with serial imaging and EUS which has demonstrated resolving inflammation and improved appearance of the duodenum. To date his symptoms have improved and biopsies remain negative for malignancy.



Image 1. EUS demonstrating a 35.4mm x 45.5mm mass in the region of the pancreatic head which had previously been seen on CT scan.



Image 2 and 3. EUS revealing a dilated pancreatic duct (above) and common bile duct (right).



Case 2: A 56 year-old male former smoker with an extensive past medical history including severe vascular disease status post amputations underwent EGD/EUS to evaluate a pancreatic mass. This revealed significant duodenal wall thickening with an edematous villous-like appearance of the second portion of the duodenum and an ampullary mass could not be ruled out. FNA was negative for malignancy and duodenal biopsy demonstrated chronic duodenitis. Two years later he presented with generalized weakness and melena. A CT scan revealed prominence of the pancreatic head and a hypodense mass within a thickened duodenum (Image 4). GOO and concern for malignancy lead to pancreaticoduodenectomy. Pathology revealed extensive scarring of the pancreatic head and duodenal musculature with numerous cysts, some of which were lined with densely inflamed granulation tissue surrounded by scar. These findings were consistent with groove pancreatitis and suggested duct malformation as a possible contributor to the process.

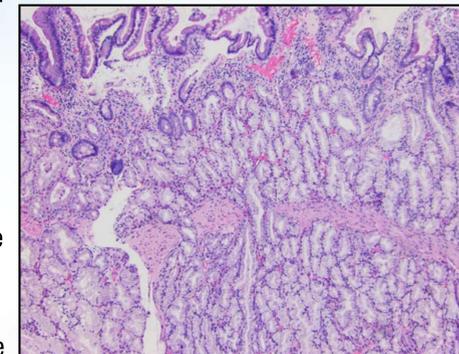


Image 5. Duodenal bulb biopsy revealing prominent submucosal Brunner glands.

Discussion:

- The true incidence of groove pancreatitis remains poorly known.¹ This creates a diagnostic challenge due to the radiographic “pseudotumor” appearance, gross pathologic features, and clinical presentation of groove pancreatitis which can make it “impossible” to differentiate from pancreatic adenocarcinoma.³ Especially in the segmental form which can involve the pancreatic head.⁷ Cases of concomitant groove pancreatitis and pancreatic adenocarcinoma as well as “pancreatic groove carcinoma” have also been described which can further complicate management.^{3,6}
- Our patients demonstrate several classic risk factors and clinical findings of groove pancreatitis including male sex, current or former alcohol and/or tobacco abuse, duodenal stenosis leading to GOO and weight loss, Brunner gland hyperplasia, tubular CBD stenosis, normal CA 19-9 level, and atypical inflammatory cells on FNA. In comparison, pancreatic adenocarcinoma will commonly have findings of an abnormal pancreatic duct, peripancreatic vascular invasion, and obstructive jaundice, all of which are less often seen in groove pancreatitis.^{1,3,7}
- A prolonged course to diagnosis is also quite classic for groove pancreatitis. This is largely due to a lack of pre-operative diagnoses due to the inability to rule out malignancy.^{5,1}
- It has been reported separately in the literature that 50% of patients with chronic pancreatitis will undergo surgical treatment and that 24.5% of patients receiving pancreaticoduodenectomy for chronic pancreatitis have groove pancreatitis. Extrapolating this data would correlate with recent literature which suggests that groove pancreatitis is actually a relatively common form of chronic pancreatitis.^{5,8}
- We also highlight a patient successfully treated without surgical removal of the affected tissue. Instead, a gastrojejunostomy was created to bypass the GOO caused by the inflammatory mass. To date, he remains without abdominal pain. Based on our review of the current literature, management of groove pancreatitis in this manner has not previously been described.

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Acknowledgements:

Victoria Loven, MD for providing biopsy images.

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