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Matthew Sullivan DO
Lehigh Valley Health Network, Matthew.Sullivan@lvhn.org

Lisa Yoo DO
Penn State-Milton S. Hershey Medical Center

Nancy Khov MD
Penn State-Milton S. Hershey Medical Center

Andrew Tinsley MD
Penn State-Milton S. Hershey Medical Center

John Levenick MD
Penn State-Milton S. Hershey Medical Center

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A Not So Groovy Masquerader: A Case Report of Groove Pancreatitis

Matthew J. Sullivan, DO¹, Lisa Yoo, DO², Nancy Khov, MD², Andrew Tinsley, MD², and John M. Levenick, MD²

¹Department of Internal Medicine, Lehigh Valley Health Network, Allentown, Pennsylvania
²Department of Gastroenterology and Hepatology, Penn State-Milton S. Hershey Medical Center, Hershey, Pennsylvania

Background

• Groove pancreatitis is an under recognized form of focal chronic pancreatitis which has also been referred to in the literature as paraduodenal pancreatitis, paraduodenal hamartoma of the duodenum, cystic duplication of heterotopic pancreas, or paraduodenal wall cyst, and myoadenomatosis.¹

• First described in 1970, the paraduodenal 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

• 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 consumption and smoking can increase the viscosity of pancreatic juice which can predispose the patient to stasis and outflow obstruction.²

• Presentation is similar to classical chronic pancreatitis, but with more pronounced prandial nausea and vomiting and weight loss.²

• Esophagogastrroduodenoscopy (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 of the groove can typically reveal benign or atypical inflammatory cells.³

• 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.³ Pancreatoduodenectomy is the definitive cure⁴ and has been shown to decrease pain and opioid dependence and result in increased body weight.⁴

Case Presentation

A 54-year-old male former-smoker with a history of chronic alcohol consumption presented to an outside hospital with complaints of refractory pyrosis and nausea three years prior to his admission to our facility. EGD demonstrated duodenal luminal thickening (Image 1) and a CT scan of his abdomen revealed duodenal inflammation of the pancreatic head with fat stranding. Biopsies demonstrated borderline high-grade dysplasia, but were not diagnostic for malignancy. He was followed clinically with progressively worsening epigastric pain and weight loss. Repeat CT scan revealed an infiltrative and heterogeneously enhancing process between the descending duodenum and pancreatic head as well as atrophy of the pancreatic body and tail without ductal dilation (Image 2). Magnetic resonance cholangiopancreatography (MRCP) revealed a 2.7 x 2.3 x 2.3 cm mass lesion in the same area with associated soft tissue infiltration and narrowing of the distal common bile duct (CBD) with an overall appearance concerning for malignancy. Persistent symptoms and a 60 pound weight loss over 6 months lead to endoscopic ultrasound (EUS) which demonstrated diffuse pancreatic parenchymal abnormalities (Image 3), 10mm CBD dilation, and enlarged peripancreatic lymph nodes. i1ga and CA-19-9 levels were within normal limits. A pancreatic duct was placed with transient relief.

Symptoms again progressed and repeat MRCP revealed changes consistent with acute and chronic pancreatitis with duodenal wall thickening. Duodenal biopsies demonstrated focal active duodenitis with prominent Brunneran glands and FNA of the pancreatic head mass was consistent with chronic pancreatitis. Transaminases, bilirubin, and lipase were all within normal limits. At this point a working diagnosis of groove pancreatitis was established.

He remained unable to tolerate oral intake and a percutaneous gastrostomy tube with jejunostomy extension was placed. Despite enteral feeds he required total parenteral nutrition prior to definitive therapy with pancreatectoduodenectomy that confirmed the procedure well and now lives symptom free without chronic pain issues and continues to regain weight.

Discussion:

• The true incidence of groove pancreatitis remains poorly known; and due to its under recognized nature can easily be misdiagnosed as pancreatic malignancy.⁶ Cases of concomitant groove pancreatitis and pancreatic adenocarcinoma as well as “pancreatic groove carcinoma” have also been described which can further complicate the differential diagnosis.⁵

• This disease entity 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.⁶ Especially in the segmental form which can involve the pancreatic head.⁴

• Important distinctions seen in groove pancreatitis but less often in pancreatic adenocarcinoma include Brunner gland hyperplasia and tubular CBD dilation, both of which were seen in our patient. An abnormal pancreatic duct, peripancreatic vascular invasion, and obstructive jaundice are less often seen in groove pancreatitis.⁵,⁶

• Our patient also demonstrates other classic risk factors and clinical findings of groove pancreatitis including alcohol and tobacco abuse, severe weight loss secondary to duodenal strictures/i1ga, a normal CA 19-9 level and atypical inflammatory cells on FNA.⁶

• His prolonged course to diagnosis is also quite classic for groove pancreatitis. Recent literature suggests that groove pancreatitis is actually a relatively common form of chronic pancreatitis, but diagnosis is often not made pre-operatively.⁷ It has been reported that 24.5% of patients receiving pancreatectoduodenectomy for chronic pancreatitis have groove pancreatitis.⁶

• Our patient ultimately underwent pancreatectoduodenectomy with resolution of symptoms. This procedure has become the definitive treatment of groove pancreatitis, ¹,² and both Whipple and pylorus-preserving (Longmire-Travers) procedures have been shown to be effective.³

References:


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