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Paraduodenal pancreatitis as an uncommon cause of gastric outlet obstruction: A case report and review of the literature



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ABSTRACT

INTRODUCTION: Paraduodenal pancreatitis (PP) is an under-recognized form of focal chronic or recurrent pancreatitis. Since PP presents with non-specific symptoms and shares radiological and histopathological features with other entities, it can be challenging to diagnose.

PRESENTATION OF CASE REPORT: Herein, a case of a 64 year-old Caucasian male with PP presenting with recurrent gastric outlet obstruction (GOO) is detailed. Over the course of two years, he underwent multiple balloon dilatations for symptom management. His diagnostic course was complicated by inconclusive and misleading biopsies.

CONCLUSION: PP can rarely present as GOO in otherwise asymptomatic patients. A preoperative pathologic diagnosis can be difficult to obtain, and in this case delayed definitive surgical management. The case is discussed in detail, and a concise review the current literature was undertaken.

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1. Introduction

PP, also commonly known as groove pancreatitis, is a less-recognized form of pancreatitis and often poses a diagnostic and therapeutic challenge [1,2]. We herein report the clinical features and therapeutic management of a complex case of PP presenting with GOO. This work has been reported in line with the SCARE criteria [3]. Informed consent was obtained from the patient for this publication.

2. Presentation of case

A 64 year-old Caucasian male was referred to the Liver and Pancreas Unit at the Ottawa General Hospital for assessment of a cystic duodenal lesion abutting the head of the pancreas consistent. His past medical history was remarkable for multiple episodes of gastric outlet obstruction requiring urgent endoscopic duodenal dilation. Other medical co-morbidities included chronic obstructive pulmonary disease, major depressive disorder, gastro-esophageal reflux disease, hypercholesterolemia, and hypertension. His past surgical history was remarkable for a remote hemorrhoidectomy and right total hip arthroplasty. He quit smok-

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ing 9 years prior to his presentation, and had a 35 pack-year smoking history. There was no significant history of alcohol use.

Two years prior the duodenal lesion was identified when he first presented to his local hospital with symptoms of gastric outlet obstruction (Fig. 1). At the time of presentation to our centre, computed tomography (CT) revealed narrowing at the junction of the 1st and 2nd part of the duodenum in conjunction with a cystic lesion abutting the pancreatic head (Fig. 2). Magnetic resonance imaging (MRI) also obtained at this time revealed a 3.2 by 2.6 cm lesion between the duodenum and pancreatic head. The lesion demonstrated slightly low signal intensity on T2-weighted sequence, and low signal intensity on T1-weighted sequence with delayed enhancement. There was no evidence of common bile duct (CBD) or pancreatic duct dilatation. Based on the radiological findings, the differential diagnosis included ectopic pancreatic tissue, cystic gastrointestinal stromal tumor (GIST), and lymphoma. The possibility of adenocarcinoma was not completely ruled out, though felt less likely given the chronicity of both his symptoms and imaging findings.

Oesophagogastroduodenoscopy (OGD) and biopsy of the lesion were carried out. Duodenoscopy showed a 2 cm stricture at junction of D1-D2 with overlying erythematous mucosa in the absence of any other abnormalities. A CRE balloon dilatation was undertaken to relieve the duodenal stenosis. Echoendoscopic evaluation revealed two hypoechoic, heterogenous, poorly demarcated lesions arising from the duodenal muscularis propria and abutting the pancreatic head; these lesions measured $12\times 4\,\mathrm{mm}$ and $5\times 3\,\mathrm{mm}$

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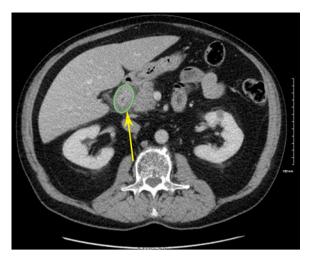


Fig. 1. CT scan from 2 years prior demonstrating duodenitis (green circle) and periduodenal inflammatory change (yellow arrow).

(Fig. 3). The pancreatic parenchyma was noted to be unremarkable. No local lymphadenopathy was present. Biopsies of the stricture showed non-specific benign intestinal cells and lymphocytes admixed with histiocytes. Neither dysplasia or malignancy was present. On EUS evaluation the lesions were felt to be most compatible with PP.

Over the course of the next two years, the patient underwent six endoscopic duodenal dilatations for symptoms that included nausea, vomiting, postprandial fullness, nighttime coughing, and worsening regurgitation. While the endoscopic dilatations provided temporary relief of symptoms, the time between subsequent symptomatic episodes progressively shortened.

Considering the recurrent nature of obstructions, and the necessity for repeated endoscopic management, surgical intervention was recommended. Consideration was given to both resection consisting of pancreaticoduodenectomy, or a gastrojejunal bypass. Since his symptoms predominately related to obstruction without a history of pain, the initial surgical plan was to perform a bypass. Before the scheduled operation he again presented to the emergency department with gastric outlet obstruction. This lead to a repeat endoscopic duodenal dilatation and endoscopic ultrasonography (EUS) guided fine needle aspiration biopsy (FNAB). On this occasion the biopsies showed spindle cells, which raised doubt about the working diagnosis of PP, and the possibility of a mesenchymal tumor such as a GIST was considered. As such, the



Fig. 3. EUS 1 month preoperatively demonstrating cystic lesion in duodenal wall.

operative plan was changed, and he underwent a standard pancreaticoduodenectomy. During the operation, the mass in the head of the pancreas was easily palpated. There was no evidence of metastatic disease. The neck of the pancreas was relatively firm, which was favored to represent chronic inflammatory change as opposed to malignancy. The remaining pancreatic parenchyma was normal and soft. His postoperative course was complicated by paralytic ileus requiring nasogastric tube placement on post-operative day (POD) 3. Furthermore, on POD 7 he developed fever and leukocytosis and was diagnosed with an International Study Group of Pancreatic Surgery (ISGPS) Grade B pancreatic fistula requiring both antibiotics and insertion of a percutaneous peripancreatic drain.

The final pathology demonstrated typical features of PP. There was evidence of pseudocystic changes in the duodenal wall, numerous fibroblasts, and mixed inflammatory infiltrates, including plasma cells, eosinophils, and lymphoid aggregates. Diffuse, marked Brunner gland hyperplasia formed a thick layer with surrounding smooth muscle, adipose tissue and myofibroblastic proliferation. The myofibroblastic proliferation encased areas of cystic change corresponding to the spindle cells identified on the previous FNAB. Extensive duodenal submucosal fibrosis was evident, which replaced most of the muscularis propria of the duodenum, and extended to the adjacent peripancreatic tissues. There was no evidence of dysplasia or malignancy.

He was seen back in the surgical clinic 4 weeks following his hospital discharge. He was clinically well, maintaining weight, and the drain output had ceased. The drain was removed, and antibiotic therapy was discontinued.

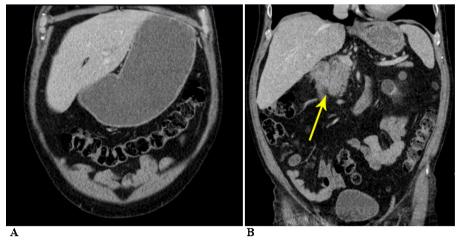


Fig. 2. Preoperative CT scan demonstrating (A) gastric outlet obstruction and (B) paraduodenal inflammatory changes (yellow arrow).

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