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Pancreatic Head Adenocarcinoma Masquerading as Groove Pancreatitis: Diagnostic Pitfalls and the Importance of Re-Evaluation in Progressive Failure to Thrive

Authors' Contribution:

Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

ABCDEFG Samer Shaja

Department of Family Medicine, Dedicated Senior Medical Center, Warren, MI, USA

Corresponding Author: Samer Shaja, Department of Family Medicine, Dedicated Senior Medical Center, 1921 E. 8 Mile Rd. Warren, Michigan, 48091, USA
Phone: 586-840-1333, e-mail: Samer.Shaja@gmail.com

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Patient: Male, 65-year-old

Final Diagnosis: Pancreatic adenocarcinoma

Symptoms: Abdominal pain • loss of appetite • nausea

Clinical Procedure: Biopsy • CT abdomen and pelvis • ERCP • gastrojejunostomy

Specialty: Gastroenterology and Hepatology

Objective: Unusual clinical course

Background: Groove pancreatitis (GP) is a rare and poorly studied form of chronic pancreatitis involving inflammation of the area bounded by the head of the pancreas, the common bile duct, and the adjacent loop of the duodenum. There is significant symptomatic and diagnostic overlap between GP and pancreatic adenocarcinoma, which can complicate the diagnosis of both conditions.

Case Report: A 65-year-old man with a history of alcohol dependence presented with 4 weeks of post-prandial nausea, bloating, and abdominal discomfort that was initially believed to be due to GP based on symptoms and imaging findings. The initial computed tomography (CT) scan showed loss of the fat plane between the pancreatic head and the duodenum, consistent with GP. The patient's clinical course continued to deteriorate despite conservative management. A subsequent CT scan showed a 1.9 × 1.5 × 1.6 cm pancreatic head mass, which was confirmed as adenocarcinoma on endoscopic ultrasound guided biopsy with IMP3 positivity and retained SMAD-4 expression on immunohistochemical staining, with T1NOM1 tumor staging (with metastatic disease due to peritoneal carcinomatosis) and markedly elevated tumor marker CA-19-9. His clinical course was complicated by malignant pyloric stenosis contributing to anorexia and malnutrition. He died 3 months after diagnosis.

Conclusions: This case illustrates how patients presenting with signs of chronic pancreatitis may instead have pancreatic adenocarcinoma. This case further illustrates how diagnostic re-evaluation is needed in patients with chronic pancreatitis who have progression of disease despite conservative management, with failure to thrive being a significant motivation for diagnostic re-evaluation in this example.

Keywords: Abdominal Neoplasms • Abdominal Pain • Cancer Pain • Failure to Thrive • Pancreatic Diseases • Pancreatic Neoplasms • Pancreatitis, Chronic

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Introduction

GP is a rare form of chronic pancreatitis defined as inflammation of the area bounded by the pancreatic head, the c-loop of the duodenum as it courses around the head of the pancreas, and the common bile duct. GP usually presents with abdominal pain, anorexia, weight loss, and post-prandial vomiting, symptoms that are similar to those of pancreatic adenocarcinoma [1-3]. The cause of GP is unknown but alcohol use is believed to be a strong risk factor and moderate to heavy alcohol use is seen in most patients with GP in population-based studies [1,3]. Other contributing factors include tobacco smoking, peptic ulcer disease, or functional obstruction of the minor papilla or the duct of Santorini of the pancreas [1,4]. It is commonly seen in the fourth or fifth decade of life and is more common in men [1,2,5]. The incidence of GP is unknown but it is believed to be extremely rare. A population-based study in southern Finland from 2005 to 2015 yielded only 33 cases [1]. The management of GP has not been well studied. Treatment usually involves supportive care, including treatment of pain, alcohol and smoking cessation, and dietary changes (eg, avoidance of fatty foods) [1,3]. Differentiating GP from pancreatic adenocarcinoma is challenging [2]. This case report discusses the patient's initial presentation and the treatment plan for a preliminary diagnosis of chronic pancreatitis (groove pancreatitis). We then discuss the importance of re-evaluating the diagnosis in light of new clinical information and worsening clinical course (specifically failure to thrive with unintentional weight loss and gastric outlet obstruction). This case report describes a case of pancreatic head adenocarcinoma initially managed as groove pancreatitis and discusses the clinical and diagnostic features that may facilitate earlier differentiation between the 2 entities.

Written informed consent for participation in this case report was obtained from the patient.

IRB approval was not required for a case report.

Case Report

The patient was a 65-year-old man with a medical history including alcohol dependence with daily use (he reported drinking between 470 to 750 mL of liquor per day), tobacco use (he reported smoking 0.5 to 1 pack of cigarettes per day, and he had been smoking since his teenaged years) with a 25 to 50 pack-year history, and hyperlipidemia. He did not take any medications. He presented to the primary care clinic on March 10, 2025 with a 4-week history of nausea and bloating worsening after meals, epigastric abdominal discomfort, and diarrhea. He did have chills for the first few days of the illness, but these had since resolved. *H. pylori* breath testing and stool studies,

including a stool culture, ova and parasites exam, stool white blood cells, and a urinalysis were all normal 1 day after the initial visit. No blood tests were performed. The patient was counseled on supportive care including fluid hydration and relative bowel rest. He was then lost to follow up.

He presented to the clinic again on June 26, 2025 with the same symptoms, which had not improved in the interim. He reported stopping all alcohol use. In June, a complete blood count, glucose level, kidney function, electrolytes, liver enzymes, and lipase level were all normal (Table 1). A CT scan of the abdomen and pelvis with intravenous contrast (non-pancreatic protocol) in July 2025 (Figure 1) showed loss of the fat plane in the area between the pancreatic head and the duodenal sweep, consistent with groove pancreatitis. No pancreatic masses were seen on imaging and there was no dilation of the pancreatic or bile ducts. The patient continued to abstain from all alcohol use and reported greatly reducing tobacco smoking. He was started on oral naproxen as needed for pain due to suspected chronic pancreatitis. During the visit of July 7, 2025, he reported greatly reduced abdominal pain, increased appetite, and increased oral intake. He also reported continued constipation. He was started on oral lactulose as a stool softener and oral pancreatic enzymes. From March 10 to July 22, 2025 he had lost 13 kg with his weight decreasing from 77 to 64 kg. During the visit on August 22, 2025, he reported worsening epigastric abdominal discomfort, bloating, and new-onset esophageal dysphagia with all solid foods, with vomiting undigested food shortly after ingestion. His weight had further decreased from 64 to 61 kg on August 15 and to 57 kg in September.

He was admitted to the hospital on September 19 due to suspected chronic GP with dysphagia causing failure to thrive. A CT scan of the abdomen and pelvis with contrast (non-pancreatic protocol) showed a diffusely dilated pancreatic duct with mild pancreatic atrophy consistent with chronic pancreatitis. He was found to have a gastric ulcer on endoscopy (confirmed as non-cancerous on biopsy) as well as pyloric stenosis (gastric outlet obstruction). He left the hospital on September 23 against medical advice. He was continued on supportive care for chronic pancreatitis as an outpatient.

He was readmitted to the hospital on October 20 due to continued failure to thrive. Due to the gastric outlet obstruction, he was placed on total parenteral nutrition (TPN). He underwent endoscopy and a nasojejunal tube was placed and advanced past the gastric outlet obstruction to begin tube feedings. However, due to worsening abdominal pain and nausea, he was unable to tolerate tube feedings. The nasojejunal tube was eventually removed and he was maintained on TPN. Magnetic resonance cholangiopancreatography showed an irregular main pancreatic duct dilation of 6 mm with abrupt

Table 1. Laboratory analysis from June 26, 2025 when the patient returned to the clinic with continued abdominal pain after being lost to follow up for more than 3 months.

Variable	Result	Normal range	Units
White blood cells	7.6	3.4-10.8	$\times 10^3/\mu\text{L}$
Red blood cells	4.77	4.14-5.80	$\times 10^6/\mu\text{L}$
Hemoglobin	16.1	13.0-17.7	g/dL
Hematocrit	47.9	37.5-51.0	%
Platelets	322	150-450	$\times 10^3/\mu\text{L}$
Blood urea nitrogen	10	8-27	mg/dL
Creatinine	0.65	0.76-1.27	mg/dL
Glucose	110	70-99	mg/dL
Glomerular filtration rate	104	$x > 59$	mL/min/1.73
Sodium	138	134-144	mmol/L
Potassium	4.5	3.5-5.2	mmol/L
Chloride	101	96-106	mmol/L
Carbon dioxide	21	20-29	mmol/L
Calcium	9.8	8.6-10.2	mg/dL
Albumin	4.6	3.9-4.9	g/dL
Total protein	6.7	6.0-8.5	g/dL
Bilirubin (total)	0.5	0.0-1.2	mg/dL
Alkaline phosphatase	101	47-123	IU/L
Aspartate aminotransferase (AST)	25	0-40	IU/L
Alanine aminotransferase (ALT)	40	0-44	IU/L
Amylase	48	31-110	U/L
Lipase	66	13-78	U/L

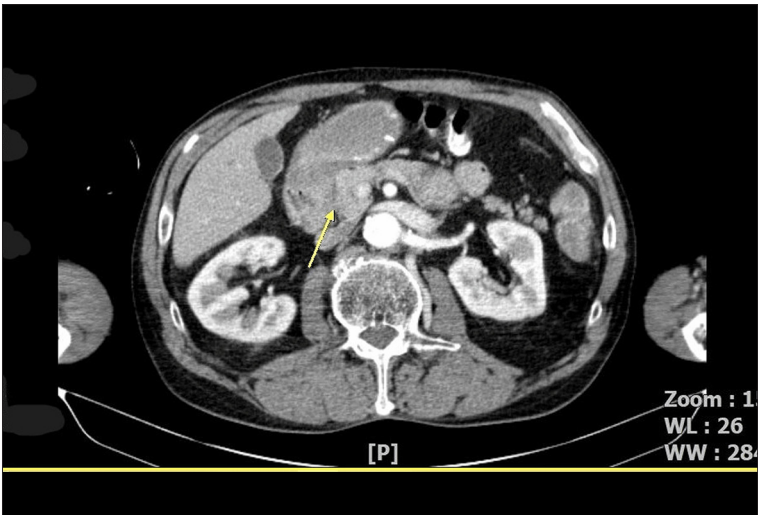


Figure 1. CT scan (with intravenous contrast, non-pancreatic protocol) from July 2025, axial view, showing fat stranding (loss of the fat plane) between the duodenal sweep and pancreatic head (yellow arrow) suggesting inflammation localized to the area.

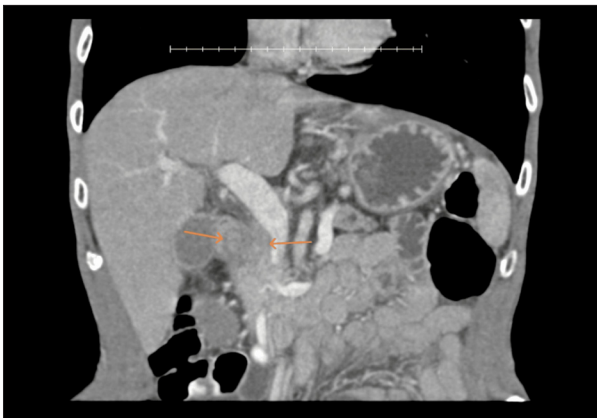


Figure 2. Coronal CT scan (with intravenous contrast, pancreatic protocol) from October 2025 showing an ill-defined 1.9-cm mass at the head of the pancreas (orange arrows). This was biopsied and confirmed as pancreatic adenocarcinoma.

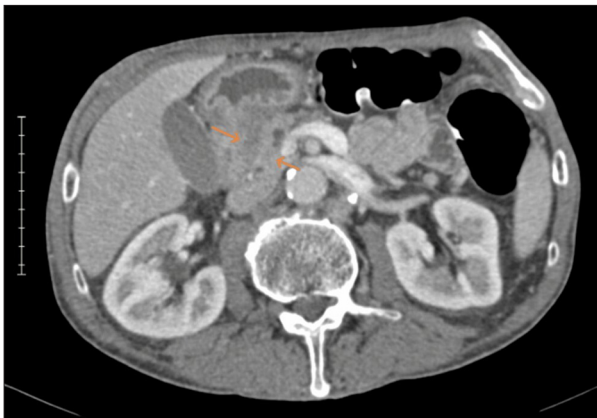


Figure 3. An axial view from the same CT scan from October 2025 showing the pancreatic head mass (orange arrows).

Table 2. Lab values for carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA-19-9) tumor markers on initial presentation to the hospital on September 22, 2025 and October 20, 2025.

Variable	Result	Normal range	Units	Date
CA-19-9	4244	x < 37	U/mL	September 2025
CA-19-9	7309	x < 37	U/mL	October 2025
CEA	4.7	x < 3 x < 5 in smokers	ng/mL	September 2025

cutoff in the pancreatic head. A short-segment narrowing of the mid common bile duct with mild upstream biliary dilation was also seen. These findings were thought to be due to a pancreatic head mass. A CT scan of the abdomen and pelvis with intravenous contrast (triple-phase pancreatic protocol) (**Figures 2, 3**) showed a 1.9 × 1.5 × 1.6 cm, ill-defined mass in the region of the pancreatic head with dilation of the pancreatic duct (measuring up to 6 mm with abrupt cutoff at the pancreatic head) and mild dilation of the common bile duct. There was no vascular involvement and no metastatic or nodal disease was seen. The carbohydrate antigen-19-9 (CA-19-9) tumor marker was markedly elevated at 4244 U/mL in September 2025 and 7309 U/mL in October 2025 (normal range less than 37 U/mL) and the carcinoembryonic antigen (CEA) was normal at 4.7 ng/mL (normal range typically less than 3 ng/mL in non-smokers and less than 5 ng/mL in smokers) (**Table 2**). The mass was core-needle biopsied via endoscopic ultrasound and confirmed as pancreatic adenocarcinoma (**Figure 4-6**). Immunohistochemical staining showed abnormal insulin-like growth factor II mRNA-binding protein 3 (IMP3) positivity (**Figure 7**) and retained SMAD-4 expression (**Figure 8**).

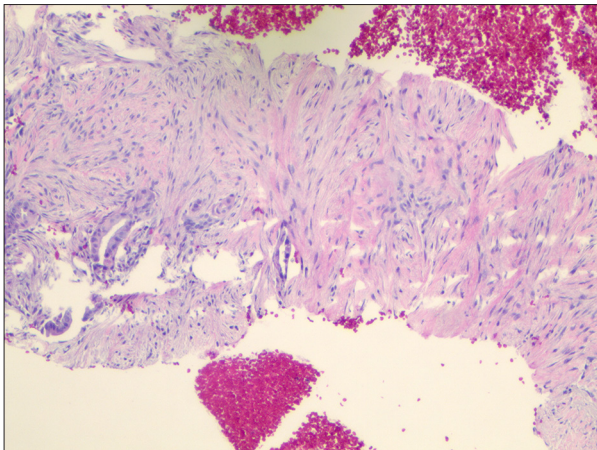


Figure 4. Pancreatic head core-needle biopsy showing pancreatic adenocarcinoma, hematoxylin and eosin (H&E staining). The histology shows sparse tubular/ductal structures among a background of desmoplasia (an abnormal growth of dense stromal connective tissue). 10 × magnification.

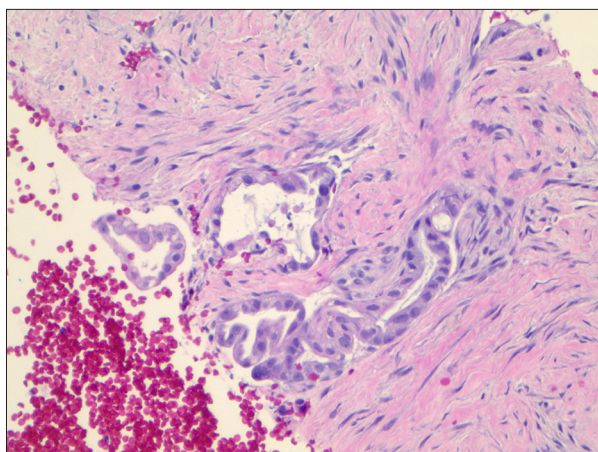


Figure 5. Pancreatic head core-needle biopsy showing pancreatic adenocarcinoma (H&E staining), showing a cluster of ductal structures among a dense stromal desmoplastic background showing fibrosis. 20 × magnification.

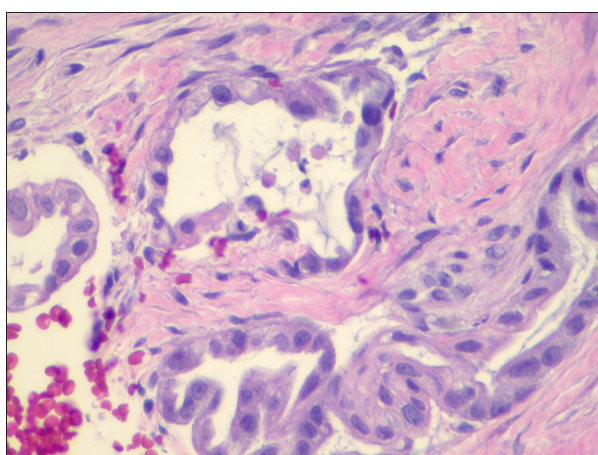


Figure 6. Pancreatic head core-needle biopsy showing pancreatic adenocarcinoma (H&E staining), a magnified view of one of the ductal elements showing some dysplasia. 40 × magnification.

The patient was unable to tolerate enteral nutrition and on November 5 he underwent an open gastrojejunostomy and cholecystectomy for the gastric outlet obstruction/pyloric stenosis as well as chronic cholecystitis. Peritoneal washings during the gastrojejunostomy showed diffuse peritoneal metastasis (peritoneal carcinomatosis). He was able to tolerate only small amounts of clear liquids orally after the gastrojejunostomy and he was maintained on TPN. The hospitalization was further complicated by a small bowel obstruction. Due to the diffuse intra-abdominal metastatic disease (peritoneal carcinomatosis), he was deemed not to be a surgical candidate for a pancreaticoduodenectomy (Whipple procedure). The final staging of the pancreatic head adenocarcinoma was stage IV (T1N0M1, with peritoneal metastasis).

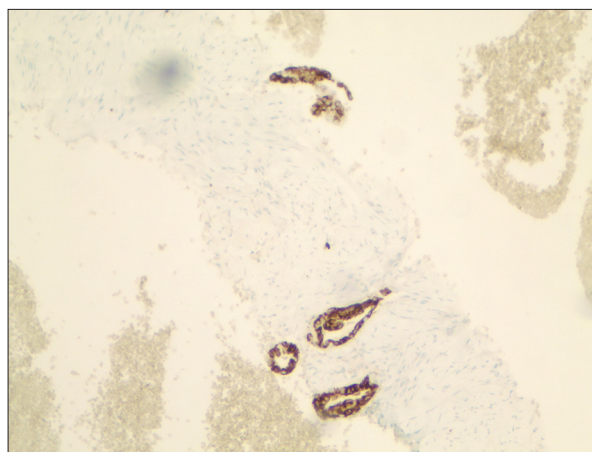


Figure 7. Pancreatic head core-needle biopsy showing abnormal IMP3 expression on immunohistochemical staining. Numerous areas of patchy IMP3 expression (dark brown staining) are seen in pancreatic ductal cells. 10 × magnification.

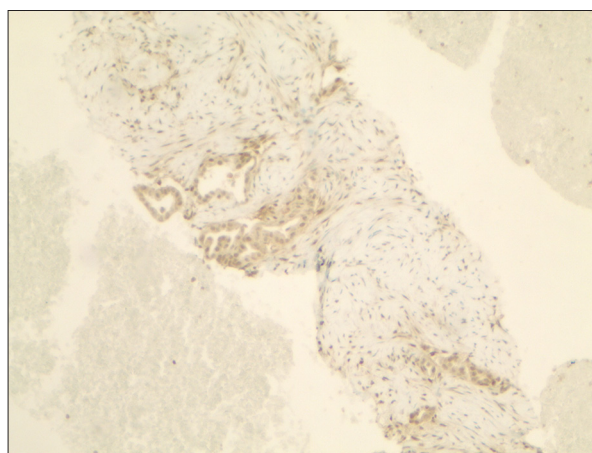


Figure 8. Pancreatic head biopsy showing retained SMAD-4 expression on immunohistochemical staining. Retained SMAD-4 expression seen as light brown staining in pancreatic ductal cells. 10 × magnification.

An implanted venous port was placed for planned palliative chemotherapy. The patient was maintained on TPN for nutrition. He was discharged from the hospital in December of 2025 with palliative care started. A percutaneous gastrostomy tube was maintained for comfort purposes to alleviate symptoms of small bowel obstruction (which developed in November 2025) due to the pancreatic cancer. The gastrostomy tube alleviated abdominal discomfort by venting air proximal to the obstruction. The patient and family declined palliative chemotherapy. The patient entered hospice and died in January 2026. **Figure 9** documents the patient's weight along a timeline showing key clinical symptoms, imaging findings, surgical procedures, and hospitalizations.

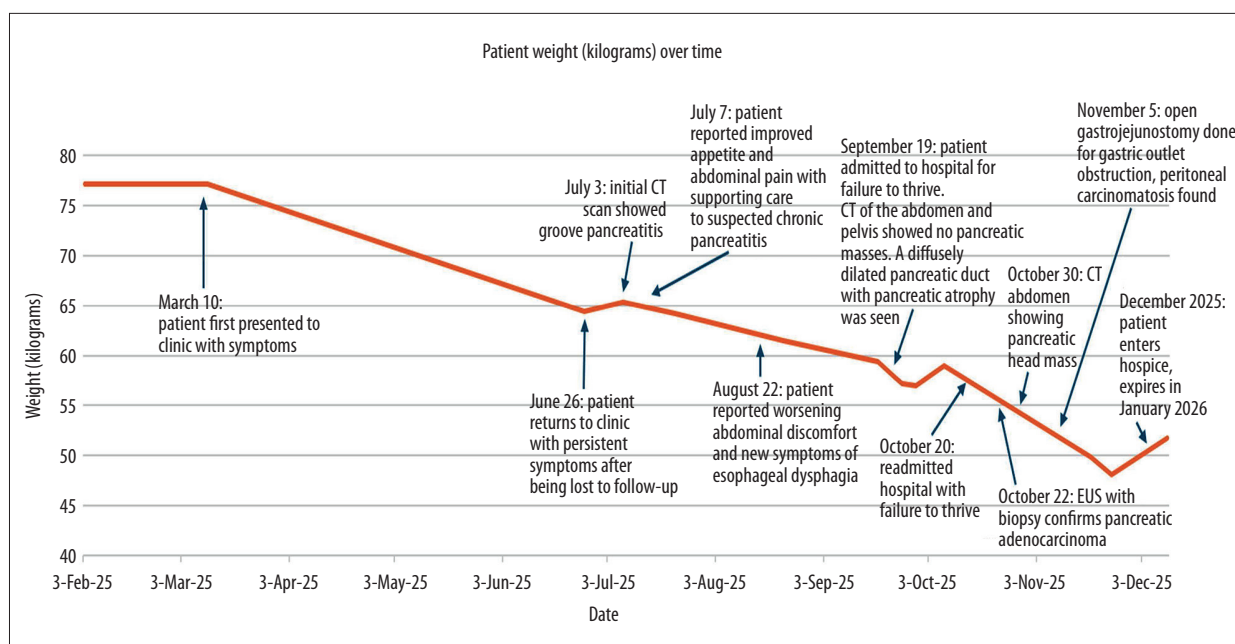


Figure 9. Patient's weight in kilograms plotted from the time of initial presentation on March 10, 2025 to death in January 2026. Arrows indicate significant clinical events, imaging tests, and surgical procedures.

Discussion

This patient initially presented with signs, symptoms, imaging findings, and clinical course suggestive of an exceedingly rare form of chronic pancreatitis (groove pancreatitis). As his condition worsened, and further diagnostic imaging and tumor markers were obtained, it was determined that the initial diagnosis of GP was instead pancreatic head adenocarcinoma. This was complicated by malignant gastric outlet obstruction and obstruction of the main pancreatic duct. The patient had metastatic disease (peritoneal carcinomatosis) at the time pancreatic adenocarcinoma was diagnosed and it was determined that he was not a candidate for curative surgery (pancreaticoduodenectomy). This case illustrates the complexities in differentiating GP from pancreatic cancer. Indeed, in those with GP, excluding cancer of the pancreatic head is challenging, especially in the segmental subtype in which the head of the pancreas is involved [5,6]. Advanced imaging specific to pancreatic adenocarcinoma such as pancreatic protocol CT scan, endoscopic ultrasound, magnetic resonance imaging or cholangiopancreatography can be used to differentiate pancreatic adenocarcinoma from GP [13]. Early use of endoscopic ultrasound may aid in the differentiation of pancreatic adenocarcinoma from groove pancreatitis. Marked duodenal wall thickening or stenosis, cystic lesions of the pancreatic groove area, and a smoothly tapering intrahepatic common bile duct are all specific to GP rather than adenocarcinoma on endoscopic ultrasound [2]. In a population-based study in southern Finland, cysts in the duodenal wall at the c-loop or groove area, duodenal wall thickening greater than 10 mm, and a visible mass

in the groove area were all specific to the radiological diagnosis of GP [1]. In that population-based study, cystic lesions in the groove area were seen in most patients with GP [1]. These findings were not seen in the initial CT scan in the present patient, who only had loss of the fat plane between the pancreatic head and the c-loop of the duodenum.

The present patient had many risk factors associated with chronic pancreatitis, including heavy chronic alcohol use, tobacco use, and male sex. Alcohol and tobacco are known to have a synergistic effect with the development of chronic pancreatitis [7-9]. He initially had a moderate improvement of his symptoms (but not resolution) with supportive care. However, after an initial improvement 3 months after presentation, an improvement believed to be aided by alcohol cessation, reducing tobacco use, and symptomatic treatment with NSAIDs, his symptoms then began to worsen again, which prompted investigation of alternative diagnoses. Indeed, chronic pancreatitis (or groove pancreatitis) that does not improve with supportive care (as in this case) should prompt exploration of alternative diagnoses.

The final diagnosis was pancreatic head adenocarcinoma, which likely explains the continued worsening course, which was ultimately complicated by failure to thrive and severe malnutrition. GP (without pancreatic cancer) can also be complicated by duodenal obstruction, biliary obstruction, or pancreatic duct obstruction, which can also lead to failure to thrive [1-3]. However, the present patient's clinical course continued to worsen despite conservative treatment (alcohol cessation, smoking

reduction, pain management, pancreatic enzyme supplementation) prompting the diagnostic re-evaluation.

Elevations in pancreatic enzymes including lipase and amylase are possible in patients with GP, or they may be normal, as in the present patient [4]. However, he was lost to follow up for 3 months after initially presenting with symptoms, and pancreatic enzymes were not immediately checked, so there is a possibility the enzymes may have been elevated early in the disease course. Early utilization of tumor markers CA-19-9 and CEA may also differentiate pancreatic adenocarcinoma from GP, but these markers are sometimes elevated in cases of GP without pancreatic adenocarcinoma [1]. In a population-based study, 33% of patients with GP (without pancreatic cancer) had elevations in CA-19-9 and CEA [1]. In another small population-based study, elevated levels of CA-19-9 and a mass on CT scan near the pancreatic head were both associated with pancreatic cancer more than with GP [12]. The present patient had elevations in CA-19-9 but not CEA. CA-19-9 is not sufficiently sensitive on its own to be used as a screening test for pancreatic cancer [13]. In symptomatic patients, elevated levels of CA-19-9 have a sensitivity of 79% to 81% and a specificity of 82% to 90% for diagnosing pancreatic cancer [14]. Elevations in CA-19-9 combined with a pancreatic mass on imaging, weight loss greater than 9 kg and elevations in bilirubin have a nearly 100% specificity for pancreatic cancer [14]. However, early in the pancreatic cancer disease course, CA-19-9 levels are associated with a false positive rate of 10% to 15% and may also be elevated in some other conditions, including acute or chronic pancreatitis, cholangiocarcinoma, and obstructive jaundice [14].

The present patient had risk factors for pancreatic adenocarcinoma, including regular tobacco use (the strongest risk factor) and chronic alcohol use [13]. The risk factors for chronic pancreatitis and pancreatic cancer strongly overlap. It is also notable that chronic pancreatitis is itself a risk factor for developing pancreatic cancer [13].

Immunohistochemical staining was done on the tissue biopsy to aid in diagnosis and provide prognostic information. Immunohistochemical staining showed abnormal patchy IMP3 and retained SMAD-4 expression in the tumor. IMP3 staining had a sensitivity of 92% and specificity of 100% in differentiating pancreatic adenocarcinoma from chronic pancreatitis in a small case series of 50 patients [15]. SMAD-4 is a tumor suppressor gene whose inactivation is associated with worse prognosis and higher risk of metastasis in pancreatic cancer [16]. SMAD-4 is highly involved in transforming growth factor β (TGF- β) signaling, and loss of SMAD-4 results in TGF- β becoming tumorigenic by impairing its ability to inhibit the cell cycle or promote apoptosis [16]. The present patient had a tumor with IMP3 positivity, which is strongly associated with pancreatic adenocarcinoma and retained SMAD-4 expression,

which is associated with less risk of metastasis and a better prognosis (however, the patient did have metastatic disease at the time of diagnosis). Early biopsy with IMP3 immunohistochemical staining can be valuable for differentiating pancreatic adenocarcinoma from chronic pancreatitis.

Conclusions

This case report discusses the case of a patient with symptoms and imaging findings originally believed to be due to a rare type of chronic pancreatitis (groove pancreatitis). He was later found to have adenocarcinoma of the pancreatic head with gastric outlet obstruction and pancreatic duct obstruction. The patient had metastatic disease in the form of peritoneal carcinomatosis at the time of definitive diagnosis, and his 3-month survival after diagnosis was less than the median survival of those with stage IV pancreatic cancer, which is about 6 months. This case report illustrates the importance of diagnostic re-evaluation in suspected chronic pancreatitis when symptoms progress despite conservative management or when complications such as failure to thrive and gastric outlet obstruction develop. The patient's continued weight loss and refractory symptoms led to diagnostic reassessment, which yielded a definitive diagnosis of pancreatic head adenocarcinoma.

The early use of tumor markers such as CA-19-9 or CEA or use of specialized imaging such as endoscopic ultrasound, pancreatic protocol CT scans, or magnetic resonance cholangiography (MRCP) can aid in differentiating pancreatic adenocarcinoma from suspected GP early in the disease course. The use of IMP-3 histopathologic staining can also aid in differentiating pancreatic adenocarcinoma from chronic pancreatitis. Research is needed to prospectively evaluate the role of EUS, CA-19-9, or CEA levels, or IMP-3 staining in patients with GP who fail conservative management, particularly those with high-risk features such as male sex, tobacco use, and progressive weight loss.

Institution Where Work Was Done

Outpatient clinic at Dedicated Senior Medical Center, Warren, Michigan, USA.

Patient Consent

Patient informed consent and permission were obtained.

Declaration of Figures' Authenticity

All figures submitted have been created by the author who confirms that the images are original with no duplication and have not been previously published in whole or in part.

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