

Two Cases of Chronic Pancreatitis with Pseudocyst Complicated by Obstructive Jaundice

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Abstract

We recently treated two cases of chronic pancreatitis with obstructive jaundice due to compression of the common bile duct by pancreatic pseudocyst. The two cases were males admitted with the complaint of icteric skin color. The first, a 46-year-old male, admitted with the complaint of icteric skin color. He was treated by operative cystojejunostomy after percutaneous drainage of the pseudocyst and percutaneous transhepatic biliary drainage. The other case was a 58 year-old male who admitted with the complaint of icteric skin color. He had an infected pseudocyst in the pancreas and was endoscopically treated. Both of them were discharged with favorable clinical course and normal laboratory findings after the treatment. The former patient remained well 11 months after treatment, but the latter patient died from necrotizing pancreatitis and septic shock 6 months after treatment. Most cases of obstructive jaundice associated with pseudocysts appear to be due to fibrotic stricture of the intrapancreatic portion of the common bile duct rather than due to compression of the bile duct by the pseudocyst. In a patient with secondary pancreatic infection or obstructive jaundice following pancreatic disease, differentiating between these two conditions is an important aspect of accurate diagnosis and therapy. Herein we report two unusual cases of chronic pancreatitis with pseudocyst complicated by obstructive jaundice.

Key Words: Obstructive jaundice, pseudocyst, chronic pancreatitis

INTRODUCTION

Jaundice occurs in 15–25% of patients hospitalized with pancreatitis.^{1,2} In patients with pancreatitis, the potential contributing causes are hepatocellular abnormalities or an obstruction of the common bile duct including gallstones, constriction of the intrapancreatic portion of the common bile duct by pancreatic edema, fibrotic stricture of the intrapancreatic bile duct, pancreatic cancer, or an abscess; any of the above acting alone or in combination.^{3,4} On the other hand, obstructive jaundice solely due to compression of the common bile duct caused by a pancreatic pseudocyst is rare.¹⁻⁴ In such a case, differentiation of the cause from biliary obstruction due to fibrotic stricture of the intrapancreatic portion of the common bile duct is an important aspect to assessing the need

for bilioenteric bypass. Herein we report one case of chronic pancreatitis with a large pseudocyst and another case of an infected pseudocyst complicated by obstructive jaundice.

CASE REPORT

Case 1

A 46-year-old alcoholic male was admitted to our hospital because of jaundice and abdominal distension. Jaundice and abdominal distension which had developed about 1 month before admission. He had been a heavy alcoholic drinker. On admission, he complained of general weakness, and showed signs of pruritus, abdominal distension, jaundice and dyspnea, but didn't complain of fever, diarrhea or constipation. Upon physical examination, his blood pressure was 180/100 mmHg, pulse rate 84/min, and body temperature 36.5°C. He appeared chronically ill looking and icteric. The examination of his abdomen revealed a markedly distended abdomen with a tender palpable mass on the epigastrium to the right of the

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midline without rebound tenderness or muscular guarding. His laboratory findings were as follow: white blood cell count $8,200/\text{mm}^3$, hemoglobin 10.7 g/dl, platelet $236,000/\text{mm}^3$, serum amylase 229 U/L, serum lipase 126 U/L, AST 59 IU/L, ALT 28 IU/L, alkaline phosphatase 558 IU/L, γ -glutamyl transpeptidase 350 IU/L, total bilirubin 22.5 mg/dl, direct bilirubin 12.5 mg/dl, and prothrombin time 35.6% of normal range. Ultrasonography and computed tomographic (CT) scan on admission demonstrated a large amount of ascites, the dilation of the biliary tract and the presence of a cystic mass, about 10 cm in diameter, located in the head of the pancreas with pancreatic parenchymal calcification (Fig. 1). Following a diagnosis of obstructive jaundice and pancreatic cyst, percutaneous transhepatic biliary drainage (PTBD) and percutaneous cyst drainage (PCD) were performed. Percutaneous transhepatic cholangiogra-

phy (PTC) and cystography revealed a long, smooth tapered stricture of the bile duct apparently curving around the cyst and compressed by it (Fig. 2). The aspirate obtained simultaneously with PCD was revealed to be slightly bloody in nature and the amylase 26,430 U/dl. Cytologic examination of the aspirate revealed no malignant cell. A follow-up cystography performed seven days after PCD revealed irregular stenosis and mild dilatation of the pancreatic duct communicating with the pancreatic cyst. After PTBD and PCD, the patient's symptoms and the laboratory findings were improved. However because the leakage of the cystic fluid through PCD persisted despite medical treatment, an exploratory cystojejunostomy was performed. After that, the patient showed a favorable clinical response with comparably normalized laboratory values. He remained well 11 months after treatment.

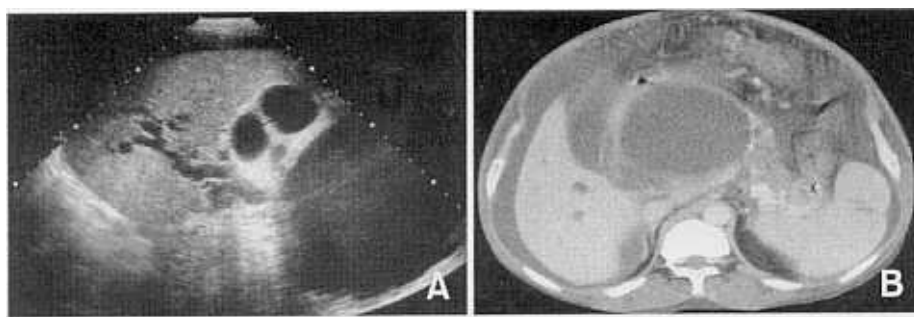


Fig. 1. Ultrasonography (A) and CT scan (B) on admission, demonstrating a large amount of ascites, dilation of the biliary tract and a cystic mass, about 10 cm in diameter, located in the head of the pancreas with pancreatic parenchymal calcification.

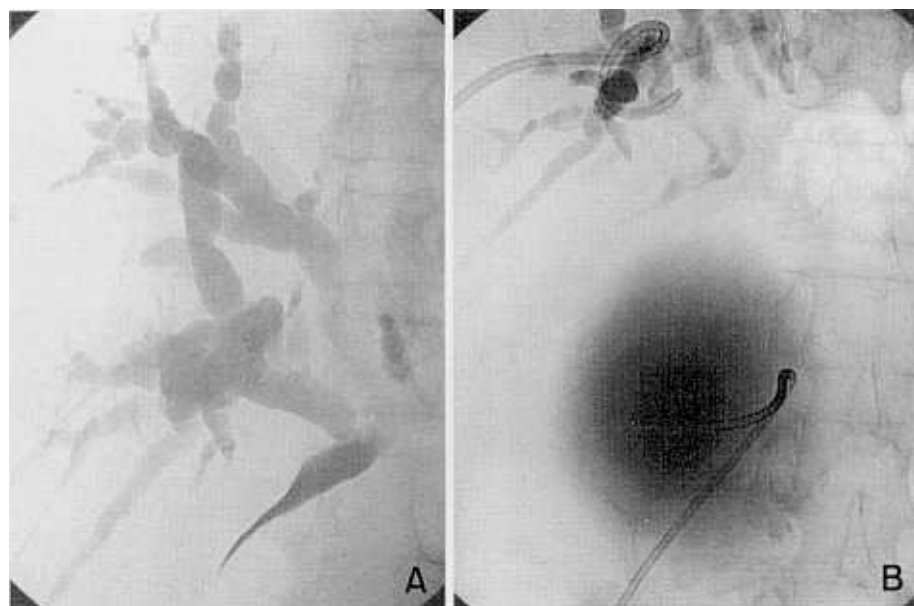


Fig. 2. PTC (A) and cystogram (B), revealing a long, smooth tapered stricture of the bile duct apparently curving around the cyst and compressed by it.

Case 2

A 58-year-old alcoholic male was admitted to our hospital for the evaluation of jaundice and the right epigastric pain. Jaundice and right epigastric pain which had developed about 10 days before admission. He admitted being a light alcoholic drinker. He had been treated under the diagnosis of acute pancreatitis and diabetes mellitus in our hospital about 2 years previously. On admission, he complained of general weakness, and showed signs of pruritus, jaundice and the right epigastric pain, but didn't complain of fever, diarrhea or constipation. Upon physical examination, his blood pressure was 160/90 mmHg, pulse rate 110/min, and body temperature 37.2°C. He appeared chronically ill looking and icteric. The examination of his abdomen revealed direct tenderness on the right epigastrium without rebound

tenderness or muscular guarding. His laboratory findings were as follow: white blood cell count 13,700/mm³, hemoglobin 12.9 g/dl, platelet 146,000/mm³, serum amylase 86 U/L, serum lipase 840 U/L, AST 398 IU/L, ALT 94 IU/L, alkaline phosphatase 387 IU/L, γ -glutamyl transpeptidase 840 IU/L, total bilirubin 16.5 mg/dl, direct bilirubin 8.6 mg/dl and prothrombin time 94.9% of normal range. Ultrasonography and CT scan on admission demonstrated the dilation of the extrahepatic bile duct and the presence of a cystic mass, about 4 cm in diameter, located in the head of the pancreas (Fig. 3). Following a diagnosis of obstructive jaundice and pancreatic cyst, endoscopic retrograde cholangiopancreatography (ERCP) was performed. ERCP revealed a smooth tapered stricture of the distal common bile duct around the cystic mass, and irregular stenosis and mild dilatation of the pancreatic duct (Fig. 4). The duodenoscopic

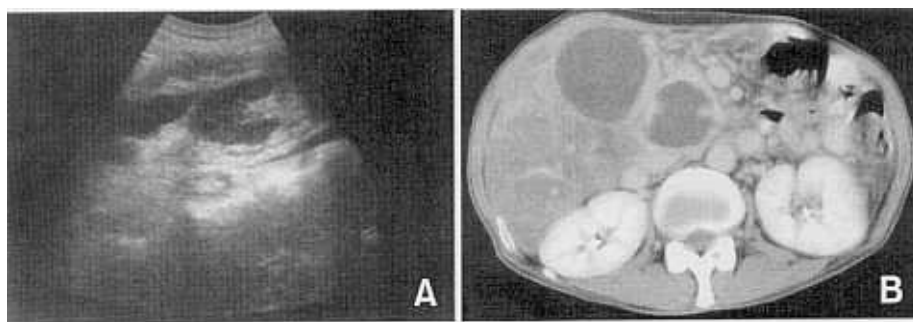


Fig. 3. Ultrasonography (A) and CT scan (B) on admission, demonstrating dilation of the extrahepatic bile duct and a cystic mass, about 4 cm in diameter, located in the head of the pancreas.

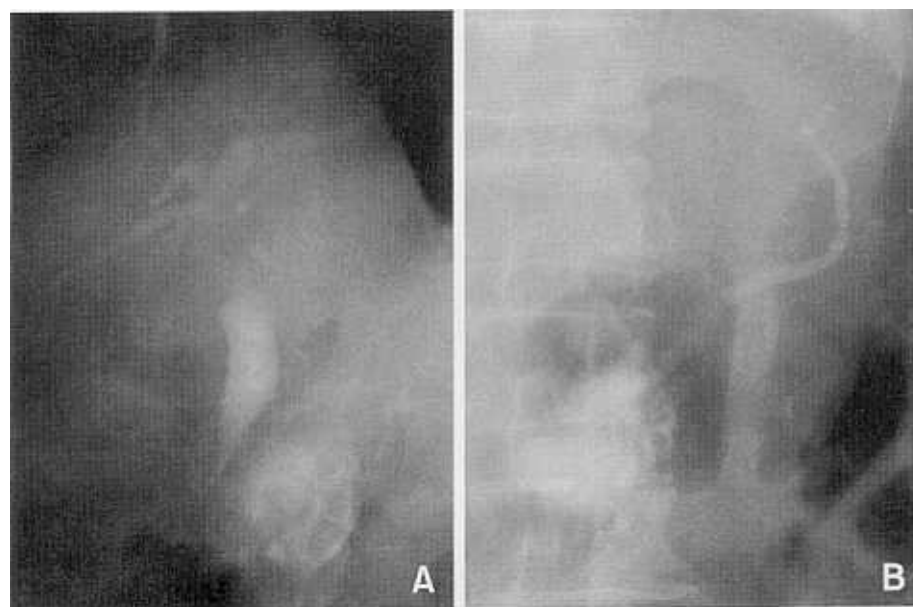


Fig. 4. ERCP, revealing a smooth tapered stricture of the distal common bile duct around the cystic mass, and irregular stenosis and mild dilatation of the pancreatic duct.

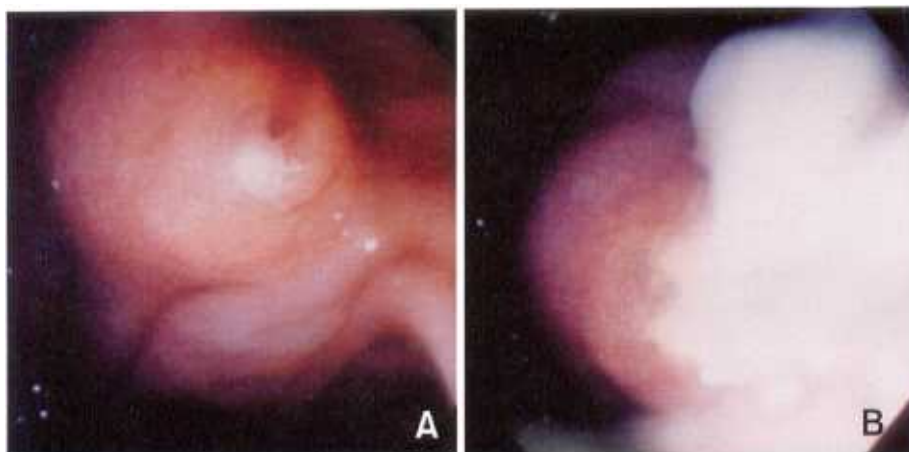


Fig. 5. The duodenoscopic findings showing protruding ampulla (A). After the pancreatic duct sphincterotomy, pus gushing out from the pancreatic duct was shown (B).

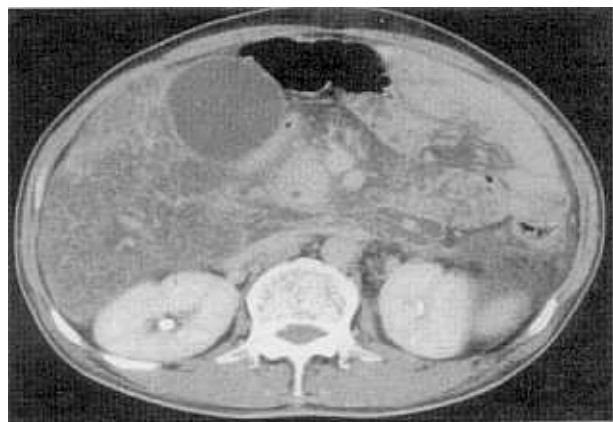


Fig. 6. An abdominal CT scan performed on readmission showing no evidence of the pancreatic cyst 6 months after the treatment.

findings showed protruding ampulla (Fig. 5A). A pancreatic duct sphincterotomy was performed and the pus was seen gushing out from the pancreatic duct (Fig. 5B). A follow-up ultrasonography performed five days after the pancreatic duct sphincterotomy showed a decrease in the size of the pancreatic cyst, reduced to 2 cm in diameter. The total bilirubin level obtained after 13 days after the pancreatic duct sphincterotomy was 2.0 mg/dl. After that, the patient showed good clinical response with improved laboratory findings. He remained well for 6 months after treatment, but then was readmitted due to necrotizing pancreatitis and septic shock, and died on remission. An abdominal CT scan performed on readmission showed no evidence of the pancreatic cyst (Fig. 6).

DISCUSSION

Jaundice in patients with pancreatitis may result from hepatocellular abnormalities or obstruction of the common bile duct. In acute pancreatitis with or without common duct stones, transient edema surrounding the distal common bile duct may cause a biliary obstruction. In chronic pancreatitis, a biliary obstruction occurs when fibrosis of the pancreas encroaches upon the lumen of the intrapancreatic portion of the common bile duct. The intrapancreatic portion may be narrowed by fibrosis without producing evidence of obstruction until there is additional inflammation and edema associated with acute pancreatitis. In this case, a bile duct obstruction that requiring an operation usually represents less than 5 percent of overall cases.^{1,4,5}

On the other hand, obstructive jaundice solely due to compression of the common bile duct caused by a pancreatic pseudocyst is rare.^{3,4,6} Obstruction of the common bile duct by external compression can result from a combination of etiologic factors including the presence of fibrosis and compression by the pseudocyst.⁷

Sidel and colleagues⁸ suggested four criteria for confirming that jaundice is caused by a pancreatic pseudocyst: 1) the presence of a biliary obstruction; 2) demonstration that the common bile duct is compressed by the pseudocyst; 3) relief of the biliary obstruction by drainage of the pseudocyst; 4) the complete disappearance of jaundice during the post-operative period. In recent years, advances in various diagnostic imaging and therapeutic modalities have facilitated the detection and treatment of pancreatic

pseudocysts.

The clinical course of our two patients satisfied these above mentioned criteria. In the former case, after the cystojejunostomy, he remained well 11 months after treatment. In the latter case, an abdominal CT scan performed 6 months after the pancreatic duct sphincterotomy showed no evidence of the pancreatic cyst. In these cases, the cyst is generally a pseudocyst sequela of chronic pancreatitis. In the normal pancreatic parenchyma, compression of the common bile duct may be compensated for a shift of the duct in the opposite direction and relative dilation caused by increased luminal pressure, and therefore jaundice does not arise. However, in chronic pancreatitis, these compensatory mechanism may not function effectively because of pancreatic fibrosis, and jaundice may eventually develop per se.⁶

Our cases could be diagnosed as chronic pancreatitis with pseudocyst because of the presence of the following findings. In the former case, the patient had a slightly elevated level of serum amylase and a normal range of serum lipase, pancreatic calcification was revealed on an abdominal CT scan and, irregular stenosis and mild dilatation of the pancreatic duct communicating with the cyst was revealed on a follow-up cystography performed 7 days after PCD, and the presence of the pseudocyst was confirmed pathologically. In the latter case, the patient had diabetes mellitus, a normal range of serum amylase and a slightly elevated level of serum lipase, irregular stenosis and mild dilatation of the pancreatic duct communicating with the cyst was revealed on an endoscopic retrograde pancreatography, and the presence of the pseudocyst was diagnosed clinically.

The incidence of infection complicating pancreatic pseudocyst is reported to be approximately 16–34%.^{9,10} Secondary infection can occur in the pseudocyst after chronic pancreatitis, and bacteria may be present in the pseudocystic fluid without revealing any clinical signs of infection.^{11,12} In our case, the patient had jaundice, direct tenderness on the right epigastrium and leucocytosis, but he had no fever, rebound tenderness or muscular defense on the abdomen.

Differentiation of the condition of an infected pseudocyst from that of a pancreatic abscess is an important aspect of treatment and prognosis. A pancreatic abscess is a localized collection of pus caused by infection as a result of liquified necrotic

tissue of the pancreatic or peripancreatic tissue. This abscess is well demarcated from the surrounding tissue, but has only a poorly defined wall. It usually develops two or more weeks after the onset of pancreatitis, after the subsidence of the acute phase. Treatment must include surgical debridement and closed drainage, and the mortality rate is 15–20%. An infected pseudocyst results from the infection of a pancreatic pseudocyst with a well-demarcated wall consisting of fibrous tissue. Treatment is based on external drainage, and the mortality rate is very low.¹³⁻¹⁵

We think that the latter case is described above was an infected pseudocyst, not a pancreatic abscess, because an abdominal CT performed on admission showed a well-demarcated cystic wall, he had no predisposing acute phase of pancreatitis and his clinical signs were mild. We think that the internal drainage of the infected pseudocyst by use of endoscopic pancreatic duct sphincterotomy was very effective because the cyst reduced in size and the infection disappeared after the treatment.

Obstructive jaundice solely due to compression of the common bile duct caused by an infected pseudocyst in patients with chronic pancreatitis is very rare.⁶ Herein, we report two unusual cases, one of obstructive jaundice solely due to compression of the common bile duct caused by a large pseudocyst which was successfully treated by surgical therapy and another of an infected pseudocyst in a patient with chronic pancreatitis successfully treated by endoscopic therapy.

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