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Biliary Pancreatic Portal Fistula as a Complication of Chronic Pancreatitis. A Case Report with Review of the Literature

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Abstract

In this study we describe an unusual complication in a patient suffering from chronic calcifying pancreatitis. The patient had a fistula between the common bile duct, the pancreatic duct, and the portal vein. He received supportive medical treatment and achieved long-term survival. A review of the literature including diagnosis, treatment and outcome of this rare complication is presented.

Introduction

Portal vein involvement can be found in approximately 10% of the patients with chronic pancreatitis (1). Others have reported an overall incidence of vascular complication in pancreatitis about 1% but with an incidence three times higher in chronic than in acute pancreatitis (2). Vascular complication occurs almost exclusively in the presence of pancreatic necrosis, with or without associated infection. The development of these vascular complications is a result of the release and activation of pancreatic and bacterial enzymes such as elastases and collagenases. These enzymes cause digestion, weakening, and ultimately rupture of the blood vessels. It has also been described that previous operation of the pancreas may contribute to vascular complications (2).

In patients with pancreatitis vascular necrosis of blood vessels resulting in haemorrhage is relatively rare (3–5) and the development of a fistula between the portal venous system and the pancreas is definitely infrequent. Since 1966 thirty-three patients have been described in the literature with a fistula between the pancreatic duct or a pancreatic pseudocyst and the portal venous system (6–33). In addition, since 1964 twenty-seven patients with fistulas between the bile duct and a pancreatic pseudocyst or a pancreatic necrosis or in a pancreatic malignancy have also been reported (34–55). We report an unusual complication in a patient suffering from chronic calcifying pancreatitis in which a fistula between the common bile duct, the pancreatic duct, and the portal vein was verified by percutaneous cholecystography. The patient received supportive medical treatment and achieved long-term survival. The patients died three years later of acute respiratory failure due to chronic obstructive pulmonary disease.

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Table 1. Laboratory values at admission

Constituent	Value	Normal range
Haemoglobin	113	134–166 g/L
S-CRP	192	<10 mg/L
Leukocyte count	22.9	4–9 x 10^9/L
Platelets count	13	150–400 10^9/L
Prothrombin	60	70–130%
S-Bilirubin	257	4–21 μmol/L
S-Alkaline phosphatase	16.2	0.8–4.8 μkat/L
S-Asparate-aminotransferase (S-ASAT)	14.5	0.20–0.60 μkat/L
S-Alanine-aminotransferase (S-ALAT)	9.5	0.20–0.60 μkat/L
S-Amylase	0.9	1.4–6.0 μkat/L
S-Albumin	24	37–48 g/L
S-Calcium	2.41	2.20–2.60 mmol/L
S-Sodium	139	134–146 mmol/L
S-Potassium	3.5	3.6–4.8 mmol/L
S-Creatinine	260	60–106 μmol/L
S-Urea nitrogen (BUN)	31	2.9–9.0 mmol/l

Case Report

The patient is a 60-year-old male with chronic obstructive pulmonary disease and for more than ten years he had suffered from a chronic calcifying pancreatitis due to previous alcohol abuse. Exocrine and endocrine insufficiency was present and treatment consisted of enzyme supplementation and insulin. A pancreatic pseudocyst had previously been successfully treated with temporary percutaneous drainage. Two years prior to the present admission the patient underwent an operation (Nissen fundoplication) for gastro-oesophageal reflux and oesophagitis.

The patient was referred to our hospital due to severe weigh loss (>10% of body weight) and intractable abdominal pain. Abdominal computed tomography (CT) demonstrated obstruction and dilatation of the main pancreatic duct, a mass in the tail of the pancreas, and multiple calcifications within the pancreas. Several collateral veins in the hepatoduodenal ligament indicated portal hypertension. The patient underwent operation whereby the pancreatic tail was resected, the spleen removed, and the pancreatic duct opened and drained by means of a pancreaticojejunostomy. The resected specimen confirmed the diagnosis of chronic calcifying pancreatitis. The patient made an uneventful recovery and was discharged to home.

Three months later the patient was admitted to the local hospital with abdominal pain and fever. Preoperative investigations revealed a right subphrenic abscess and a pelvic abscess. Both abscesses were surgically drained. Postoperatively the patient developed sepsis and multiple organ failure involving liver, kidney, and heart. He was referred to our institution and was initially treated in the ICU. Laboratory results at admission are shown in Table 1. The patient was highly jaundiced. He developed an acute acalculous cholecystitis with high fever, severe right upper quadrant pain, and rebound tenderness. Due to his severe general condition

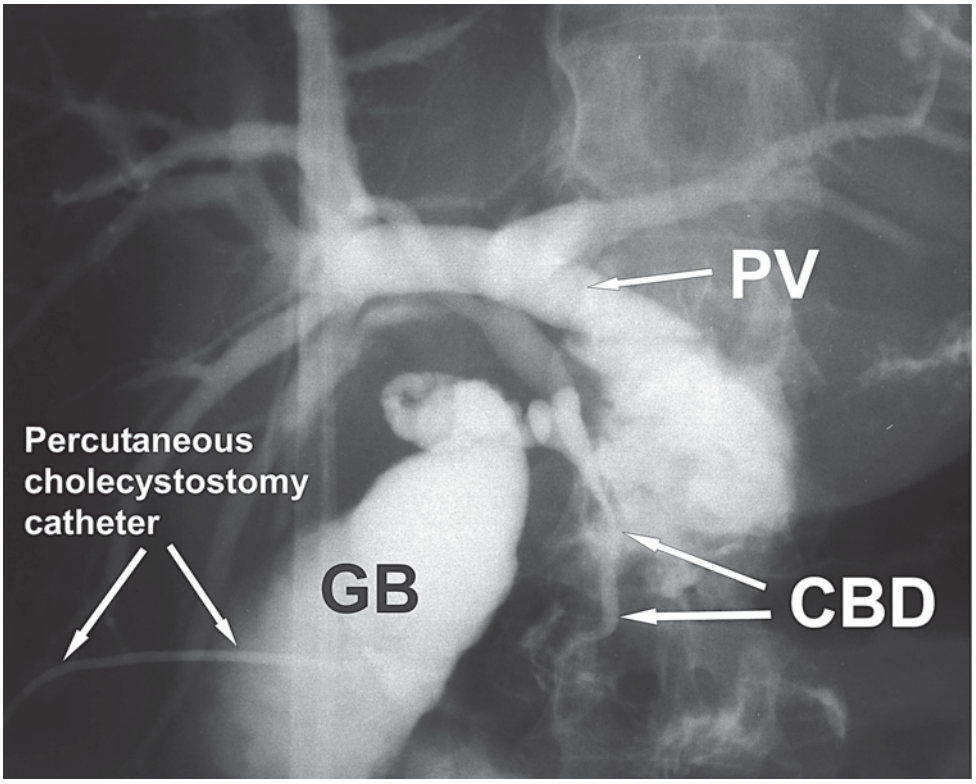

Figure 1. After injection of water-soluble contrast fluid via the percutaneous cholecystomy catheter simultaneous filling of the gallbladder (GB), the common bile duct (CBD) and the portal vein (PV) can be seen.

a percutaneous cholecystostomy (56) was used for treatment of the cholecystitis. In association with the procedure administration of contrast medium resulted in concurrently filling of the gallbladder, the intra- and extra-hepatic bile ducts, the portal vein and the main pancreatic duct (Figure 1). The portal venous blood flow was low but hepatopetal and no sign of thrombosis was found. However, there was no sign of contrast medium in the superior mesenteric vein. A CT scan showed an inflammatory process in the head of the pancreas but no sign of a pseudocyst. The patient improved and he was transferred to the general ward. A newly developed intrahepatic abscess of the left liver lobe was successfully drained percutaneously. The patient was operated once more because of abdominal wound dehiscence. After five weeks he was discharged to the local hospital and after a further two weeks of rehabilitation the patient could return to home.

Two months later the patient was readmitted to our institution due to a right subphrenic abscess and a cutaneous fistula in the right upper quadrant of the abdomen. The subphrenic abscess was drained percutaneously. The cutaneous fistula was derived from the gallbladder. Contrast medium filled the gallbladder but no contrast medium reached the extra- or intra-hepatic bile ducts. For treatment a pigtail cath-

eter was inserted through the fistula into the gallbladder. A CT scan showed sign of chronic pancreatitis but no sign of pancreatic abscesses or pseudocysts. Three weeks later the patient left the hospital in good condition but with the catheter in place.

Subsequently the patient was able to work part time. Several attempts to remove the drainage resulted in empyema of the gallbladder and new insertion of a catheter was necessary. The overall medical condition of the patient represented a contraindication to cholecystectomy. Thus, the catheter was changed routinely every six to eight weeks. The patient died of acute respiratory failure due to chronic obstructive pulmonary disease three years after his initial admission.

The autopsy revealed sign of chronic inflammation in the hepatoduodenal ligament and at the hilum of the liver. However, no fistula between the bile ducts, the pancreatic duct, or the portal vein was found. There was no pathological finding in the extra- or intrahepatic bile ducts. The portal vein was patent and without any sign of old or new thrombosis. The liver showed no sign of cirrhosis. The remaining pancreas showed sign of chronic pancreatitis with extensive fibrosis and multiple calcifications.

Discussion

Various vascular complications may occur in patients with chronic pancreatitis (1, 2, 57–60). Since 1966 thirty-three patients with pancreatico-portal fistulas have been reported in the literature (Table 2) (6–33). The male/female ratio was 26/7 and the mean age was 49 years. Thirty-one patients suffered from chronic pancreatitis most often alcohol related. Two patients suffered from acute pancreatitis (25, 30). One patient suffered from both chronic pancreatitis and a pancreatic adenocarcinoma (13).

Several symptoms were observed in the patients (Table 2). Most of the patients had severe abdominal pain and hyperamylasaemia. Ten patients had subcutaneous fat necrosis – Weber-Christian disease (6, 7, 9, 12, 14, 17, 19, 20, 30). The association of pancreatitis with disseminated fat necrosis includes subcutaneous nodules, polyarthritis, polyserositis, necrotic bone lesions, and peripheral eosinophilia. The pathogenesis of fat necrosis in distant tissues associated with pancreatic disease remains unclear. However, high levels of circulating proteolytic and lipolytic enzymes are most commonly implicated. Six patients had recently had pancreatic surgery.

The diagnosis was obtained by several methods (Table 2). Endoscopic retrograde cholangiopancreaticography (ERCP) was frequently used and are recommended as the most useful method by several authors. However, in seven cases the diagnosis was established first at the autopsy. Furthermore, one method – ultrasonographic guided pancreatic duct cannulation – has been described as useful especially if contrast fluid is injected in the pancreatic duct prior to a CT scan (8, 10). Recently, magnetic resonance tomography (MR) has been described as an effective diagnostic tool (22, 33).

Table 2. General description, underlying disease, symptom, and main diagnostic procedure in patients with pancreatico-portal fistulas documented in the literature (6–33)

General description	
Number of Studies	29
Number of patients	33
Male/female	26/7
Mean age (range)	49 yrs. (29–82)

	Aetiology			
Underlying disease	Alcohol	Gallstones	Unknown	Number of patients (female)
Chronic pancreatitis*	25 (5)	0	6 (2)	31 (7)
Acute pancreatitis	1	1	0	2
Total	26 (5)	1	6 (2)	33 (7)

*) One patient had also pancreatic adenocarcinoma.

Symptom	Number of symptoms in 33 patients
Abdominal pain	25
Hyperamylaemia	20
Anaemia	12
Subcutaneous fat necrosis	10
Gastrointestinal bleeding	8
Sepsis	7
Jaundice	5
Previous abdominal operation	6

Main diagnostic procedure	Number of patients
ERCP	14
Autopsy	7
Portography	3
Operation	3
CT	3
Ultrasongraphic guided pancreatic duct cannulation	2
MRI	1
Total	33

Twenty-nine of the patients had a pancreatic pseudocyst most often located in the head of the pancreas. In 19 patients there was a communication between the pseudocyst and the portal venous system or the main pancreatic duct and the portal venous system. The specific vessels involved are described in Table 3. In most cases there was thrombosis of the portal venous system.

Table 3. Diagnostic findings in patients with pancreatico-portal fistulas documented in the literature (6–33)

	Pseudocyst present (n=29)	Pseudocyst not present n=4
Location:		
Head	22	
Body	3	
Tail	4	
Relation to the main pancreatic duct:		
Yes	19	
No	6	
Not stated	4	

	Number of patients		
Ruptured vessel	Pseudocyst present	Pseudocyst not present	Total
Portal vein	21	3	24
Splenic vein	6	0	6
Superior mesenteric vein	1	1	2
Arterial-portal fistula*)	1	0	1
Total	29	4	33

*) (26)

Thrombosis of the portal venous system	Number of patients
Portal vein	27
Splenic vein	2
Superior mesenteric vein	1
No sign of thrombosis	3
Total	33

Twenty-two patients underwent medical supportive treatment that included percutaneous puncture and drainage of pseudocysts, ERCP with or without insertion of an endoprosthesis in the common bile duct and/or in the pancreatic duct, and endoscopic cystogastrostomy. In one case with an arterial-portal fistula the treatment was angiography and embolisation (26). Ten patients underwent surgical treatment including pancreaticoduodenectomy, left sided pancreatic resection including splenectomy, pancreaticojejunostomy, partial pancreatic resection with pancreaticojejunostomy or cystojenunostomy, and exploration and drainage (Table 4).

Nine patients died, three of these were women, and all were treated non-surgically. Three of the 10 patients with subcutaneous fat necrosis died. The cause of death is shown in Table 4. In one patient the outcome was not stated.

It is not possible to give any general guidelines for the medical or surgical treatment because the patients and the severity of their disease evidently differ. Thus,

Table 4. Treatment, surgical procedure, and cause of death in patients with pancreatico-portal fistulas documented in the literature (6–33)

Treatment	Number of patients	Mortality, female ( )
Medical supportive	22	9 (3)
Surgical	10	0
Not stated	1	–
Total	33	9 (3)

Surgical procedure	Number of patients
Pancreaticoduodenectomy	2
Left sided pancreatic resection + splenectomi	3
Pancreaticojejunostomy	2
Partial pancreatic resection and cystojenunostomy	1
Partial pancreatic resection and pancreaticojejunostomy	1
Laparotomy + drainage	1
Total	10

Cause of death	Number of patients
Gram negative sepsis	1
Generalised fat necrosis	3
Oesophageal varices bleeding	1
Intrathoracic abscess	1
Pulmonary embolism	1
Intestinal perforation (colonic volvolus)	1
Renal insufficiency	1
Total	9

the management must be individualised and medical and surgical treatment alone or in combination may be the treatment of choice.

Previous case reports concerning pancreatico-portal complications have also involved patients with pancreatic carcinoma without a pre-existing fistula (45, 50, 61–63). Furthermore, a fistula between the bile duct and a pancreatic pseudocyst or pancreatic necrosis has also been reported (34–44, 46–49, 51–55).

The patient in this report suffered from chronic calcifying pancreatitis. Before the development of the fistula the patient had an operation including a distal pancreatic resection and a pancreaticojejunostomy. The development of a fistula between the pancreatic duct, the common bile duct, and the portal vein was probably due to an episode of acute aggravation of the chronic pancreatitis in the head of the pancreas. In addition, the previous surgical intervention may also have been a contributing factor (2). The development of the liver abscess could have been a result of the fistula between the area of inflammation in the pancreas and the portal vein. Bacteria, endotoxins, pancreatic enzymes, and inflammatory active substances

could thereby reach the liver. Similarly, an impaired portal venous blood supply could be a contributing factor for the abscess formation due to focal liver ischemia. On the other hand, the patient had a well-developed collateral blood flow to the liver that helped to restore the liver function and subsequently contributed to his survival. The patient did not suffer from subcutaneous fat necrosis – Weber-Christian disease. Except for the percutaneous cholecystostomy he received no specific treatment for resolving the fistula. At autopsy the fistula had totally disappeared, the portal vein was patent and the liver was without sign of cirrhosis.

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