A Rare Case of Severe Acute Pancreatitis Complicated with Pancreatic Pseudocysts, Obstructive Jaundice and Intraperitoneal Hemorrhage

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A 58-year-old man visited our hospital because of back pain. Blood examinations revealed the presence of acute inflammation and an increase of pancreatic enzymes. Abdominal computed tomography indicated pseudocysts in the pancreas. The patient was diagnosed as having acute pancreatitis with pseudocysts formation. During the course of the disease, a newly formed pseudocyst in the pancreatic head compressed the common bile duct, leading to the obstructive jaundice. In addition, the rupture of a pseudocyst in the pancreatic tail caused intraperitoneal hemorrhage. This is an interesting case of acute pancreatitis with pseudocysts in which two rare complications developed.

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Introduction

Pancreatic pseudocysts generally develop in the course of pancreatitis or after traumatic episodes. Life-threatening complications may occur in some of these cases. We encountered a rare case with acute pancreatitis which was complicated by pseudocyst formation resulting in obstructive jaundice and intraperitoneal hemorrhage from a ruptured pseudocyst.

Case Report

A 58-year-old man with epigastralgia and back pain visited our hospital. There was no significant family history or past history. The patient had been drinking excessive amounts of alcohol for the previous two months, however he had no previous history of alcohol abuse. On August 16, 1994, he felt epigastralgia and radiating pain in the back. Since those symptoms persisted, the patient was admitted to our hospital. Laboratory findings on admission disclosed increases in all pancreatic enzymes and C-reactive protein. Computed tomographic study on admission (Fig. 1A–D) showed the dilatation of main pancreatic duct and the edema in the tissue surrounding the

pancreas. In addition, two thin-walled cystic lesions were found; one, 11 × 7 cm in size, in the pancreatic body, and the other one, 4 × 5 cm in size, in the pancreatic tail. From these findings, the patient was diagnosed as having mild acute pancreatitis with pseudocyst formation under the 1990 revised criteria for grading severity of acute pancreatitis from the Research Committee for Intractable Diseases of the Pancreas, Japanese Ministry of Health and Welfare. Treatment was started immediately with antibiotics, gabexate mesilate and total parenteral nutrition. In spite of the intensive medical treatment, abdominal pain with muscular defense and hypovolemic shock developed on the 18th hospital day. Findings of the arterial blood gas analysis showed a base excess of –10.4 mEq/l (≤ -3 mEq/l) and examination of the blood indicated a red blood cell count of $256 \times 10^4/\mu\text{l}$, hemoglobin of 8.8 g/dl, hematocrit of 26.7% ($\leq 30\%$, post infusion) and fasting blood glucose of 608 mg/dl (≥ 200 mg/dl) (Table 1). Computed tomographic study (Fig. 2A, B) revealed an increased density in the content of the cyst in the pancreatic tail and intraperitoneal fluid collection around the spleen, suggesting intracystic hemorrhage in the pancreatic tail and a resultant rupture of the cyst. From these findings, the patient was diagnosed as having severe acute

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Figure 1. Computed tomographic study on admission. Two thin-walled cystic lesions are noted; one in the pancreatic tail (A), and the other in the pancreatic body (B–D).

Table 1. Laboratory Data on the 18th Hospital Day

Peripheral blood		Blood urea nitrogen	11.8 mg/dl
White blood cell	16,570/ μ l	Creatinine	1.3 mg/dl
Red blood cell	256 $\times 10^4$ / μ l	Fasting blood glucose	608 mg/dl
Hemoglobin	8.8 g/dl	Ca	8.3 mg/dl
Hematocrit	26.7%	Amylase	852 IU//
Platelet	15.8 $\times 10^4$ / μ l	Lipase	235 U//
Biochemistry		Trypsin	3,000 ng/ml
Total protein	5.5 g/dl	Elastase I	1,995 ng/dl
Albumin	2.9 g/dl	PSTI	130.1 ng/ml
Total bilirubin	1.0 mg/dl	Serologic	
Aspartate aminotransferase	41 IU//	C-reactive protein	4.9 mg/dl
Alanine aminotransferase	46 IU//	Urine amylase	9,100 IU//
Lactate dehydrogenase	420 IU//	Blood gas analysis	
Alkaline phosphatase	243 IU//	pH	7.231
Leucine aminopeptidase	52 IU//	PaO ₂	79.5 mmHg
γ -glutamyl transpeptidase	51 IU//	PaCO ₂	38.8 mmHg
		Base Excess	-10.4 mEq//

Figure 2. Computed tomographic study on the 18th hospital day (A, B), and computed tomographic study with contrast enhancement on the 19th hospital day (C, D).

pancreatitis under the revised criteria for grading the severity of acute pancreatitis. Computed tomographic study with contrast enhancement on the following day (Fig. 2C, D) indicated the hemostasis of the intraperitoneal hemorrhage and newly formed cyst in the pancreatic head. The dosage of gabexate mesilate administration was increased to 1,000 mg/day. In addition, continuous infusion of ulinastatin 200,000 units/day and citicoline 1g/day was started. In spite of these treatments, the direct serum bilirubin concentration began to increase and reached 6.7 mg/dl on the 30th hospital day. Abdominal ultrasonography showed dilatation of the intrahepatic bile ducts and the proximal part of the common bile duct. Percutaneous transhepatic cholangio drainage and cholangiography were performed (Fig. 3). While the proximal half of the common bile duct was dilated, the intrapancreatic portion of the common bile duct was narrowed by the extramural compression.

On the 55th hospital day, the repeated percutaneous transhepatic cholangiography (Fig. 4) showed the complete obstruction of the common bile duct. Computed tomography taken on that day (Fig. 5A, B) revealed not only the enlargement of the cyst in the pancreas head but also swelling of the pancreas around the cyst, with resulting obstruction of the common bile

duct. Consequently, surgical treatment was performed on the 75th hospital day for the obstructive jaundice. The common bile duct was compressed and obstructed by the cyst in the pancreatic head. Another cyst 4 × 5 cm in size was found in the tail of pancreas. The content of the cyst in the tail was a brown colored fluid, indicating old intracystic hemorrhage. In addition, approximately 400 ml of pale blood-colored ascites was observed in the abdominal cavity. Choledocho-ileostomy and gastropancreatic tail cyst anastomosis were performed.

After the surgery, jaundice and pancreatitis temporarily subsided. However, the levels of pancreatic enzymes increased, with the aggravation of pancreatitis. The patient developed disseminated intravascular coagulation, and eventually died on the 126th hospital day.

Discussion

We report a case of acute pancreatitis with pancreatic pseudocysts, one of which compressed the common bile duct, leading to obstructive jaundice and in the other hemorrhage occurred. Pancreatic pseudocysts typically occur following acute pancreatitis and 2–20 cases are found in 100,000 hospital

Figure 3. Percutaneous transhepatic cholangiography on the 30th hospital day showed the narrowing of the common bile duct extramural compression (arrows) and leakage of contrast medium into the duodenum.

admissions (1). The major complications of pancreatic pseudocysts are rupture, hemorrhage, and abscess formation. Although a pancreatic pseudocyst, when present in the head of the pancreas, may compress the common bile duct and cause obstructive jaundice, the reports of such cases with this complication are extremely rare (2–9). Pancreatitis is frequently complicated by biliary stenosis and obstructive jaundice as a consequence of fibrotic stricture of the intrapancreatic portion of the common bile duct. Therefore, to confirm that the biliary obstruction is due to the direct compression by pancreatic pseudocysts, four criteria have been proposed (10); I) presence of biliary obstruction, II) surgical demonstration of the compressed common bile duct by a pseudocyst, III) relief of biliary obstruction by drainage of the pseudocyst, and IV) complete

Figure 5. Computed tomography on the 55th hospital day revealed increased size of the cyst and swelling of the pancreas around the cyst (A, B).

Figure 4. Percutaneous transhepatic cholangiography on the 55th hospital day. The common bile duct was completely obstructed at its middle portion and the contrast medium injected into the common bile duct did not leak into the duodenum.

Table 2. Cases of Pancreatic Pseudocyst Complicated with Intraperitoneal Hemorrhage in Japan

No.	Year	Age/sex	Chief complaint	Therapy	Prognosis	Reference
1	1983	31/M	up. abd. pain	Operation	Alive	Ishizaki et al (21)
2	1984	62/M	low. abd. pain	Conservative	Dead	Harigane et al (22)
3	1986	34/M	up. abd. pain	Conservative	Alive	Yamamoto et al (23)
4	1987	51/M	up. abd. pain, fever	Hemostasis	Alive	Miyake et al (24)
5	1988	58/F	abd. pain, abd. distension	Hemostasis of pseudoaneurysm	Alive	Kobayashi et al (25)
6	1989	69/M	epigastralgia	Drainage	Alive	Matsui et al (26)
7	1989	30/M	lt. hypochondralgia	Distal pancreatectomy and splenectomy	Alive	Hayashi et al (27)
8	1991	43/F	epigastralgia, back pain	Conservative	Dead	Sekibe et al (28)
9	1992	50/M	up. abd. pain	Distal pancreatectomy and splenectomy	Alive	Fujii et al (29)
10	1993	51/M	hematoemesis	Conservative	Dead	Ishitodani et al (30)
11	1994	47/M	abd. pain	Conservative	Dead	Ito et al (31)

M: male, F: female, up: upper, abd: abdominal, low: lower, lt: left.

disappearance of jaundice during the post operative period. The clinical course of the present case satisfied the first two of the criteria but did not entirely satisfy the others. In this present case, choledochostomy was performed because the organization around the pancreas was too prominent to drain the cyst. However, it is conceivable that the obstructive jaundice was caused by the direct compression of the common bile duct by the pancreatic pseudocyst based on the following two reasons. Firstly, the direct serum bilirubin concentration showed a parallel increase with the enlargement of the pancreatic pseudocyst. Secondly, percutaneous transhepatic cholangiography revealed a long smooth-tapered obstruction of the common bile duct which is usually observed in cases with extramural compression by a pseudocyst.

In the present case, intracystic hemorrhage also occurred, followed by its rupture into the intraperitoneal cavity. Although the intracystic hemorrhage with resulting rupture is the most important cause of death in patients with pancreatic pseudocysts (11), the incidence of spontaneous hemorrhage into a pancreatic pseudocyst appears to be a rare phenomenon and is observed only in several percent of all the pancreatic pseudocysts (12–18). Furthermore, rupture of the pancreatic pseudocyst caused by an intracystic hemorrhage is also reported to be an extremely rare phenomenon (14, 19, 20). Indeed, in Japan, there have been only 11 cases over the past 15 years to the best of our knowledge (Table 2) (21–31).

In summary, we reported a case with pancreatic pseudocysts with two rare complications; obstructive jaundice caused by a pseudocyst compression and intracystic hemorrhage with resultant rupture of the pseudocyst. Thus, the present case emphasizes the importance of careful observation and intensive treatment for the patients with pancreatic pseudocysts because life threatening complications may occur.

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