Ruptured Subcapsular Giant Hematoma of the Spleen as a Complication of Chronic Pancreatitis

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We present a 63-year-old man with chronic pancreatitis and the rare complication of giant subcapsular splenic hematoma. The hematoma showed no size reduction for 6 weeks. Then, the hematoma was infected with *Pseudomonas aeruginosa* after the recurrence of the pancreatitis, and it finally ruptured. This case suggested that in cases of giant subcapsular splenic hematoma with chronic pancreatitis reductive pressure treatment should be administered as early as possible. (Internal Medicine 34: 564–568, 1995)

Key words: pancreatic pseudocyst, reductive pressure treatment

Introduction

A subcapsular hematoma of the spleen is a rare hemorrhagic complication of pancreatitis (1–5). The treatment for subcapsular hematoma of the spleen with pancreatitis has not been established. We present a patient with a giant subcapsular splenic hematoma as a complication of chronic pancreatitis. The origin of the hematoma was thought to be rupture of small pancreatic pseudocysts in the tail of the pancreas.

Case Report

This 63-year-old Japanese man was referred to our hospital on November 2, 1992, due to a giant subcapsular cystic lesion of the spleen. He had a ten-year history of chronic pancreatitis. His spleen had been normal size on abdominal ultrasonography (US) performed for follow-up of chronic pancreatitis three or four times a year. He developed general malaise and anorexia 2 months before the present admission. Sudden severe pain occurred in the left upper quadrant of the abdomen on October 13, 1992, followed by dull and persistent pain in the same region. The giant subcapsular cystic lesion of the spleen was discovered on abdominal US at a local clinic and he was referred to our hospital for further evaluation.

On admission, his temperature was 36.4°C, the pulse rate was 76, and blood pressure 124/70 mmHg. Slight anemia of the conjunctiva was found on physical examination. Laboratory findings are presented in Table 1. Mild anemia was seen. Erythrocyte sedimentation rate (ESR) and C-reactive protein

were increased. Serum amylase was 1,390 U/l (normal, 115–360 U/l), and serum lipase 37 U/l (normal, less than 20 U/l). The serum levels of CA19-9, carcinoembryonic antigen (CEA) and DU-PAN-2 were all normal.

A chest X-ray (Fig. 1) showed an elevation of the left diaphragm with a slight pleural effusion. Abdominal US revealed a thin-walled cystic lesion measuring 10×10 cm in the antero-lateral part of the spleen, which compressed the spleen (Fig. 2A). No capsule was observed between the cyst and the splenic parenchyma (Fig. 2B). The cyst contained numerous fine echoic spots that were immobile, even on compression with the ultrasonographic probe. The splenic parenchyma showed no abnormality (Fig. 2C). The pancreas exhibited an irregular contour with a coarse internal texture that suggested chronic pancreatitis (Fig. 2D). Computed tomography (CT) of the abdomen (Fig. 3) showed the large cystic lesion covered with the splenic capsule. The cyst compressed the spleen medially. Small cystic spots were demonstrated in the tail of the pancreas. The cystic spots were not observed in the tail of the pancreas on US. Magnetic resonance imaging (MRI) (Fig. 4) showed high-intensity signals suggesting a bloody content on both the T1- and T2-weighted images of the subcapsular cystic lesion. Therefore, the cyst was regarded as a hematoma. Celiac angiography (Fig. 5) showed no evidence of encasement of the splenic artery or the formation of a pseudoaneurysm. The capsular branches of the splenic artery were stretched. No active bleeding site was observed and the splenic vein was patent. Endoscopic retrograde pancreatogram (ERP) (Fig. 6) revealed dilatation of the pancreatic duct. The hematoma did

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Table 1. Laboratory Data on Admission

Erythrocyte sedimentation rate	116 mm/h	Aspartate aminotransferase	21 U/l
C-reactive protein	3.1 mg/dl	Alanine aminotransferase	20 U/l
Red blood cell	323×10 ⁴ /μl	Alkaline phosphatase	168 U/l
Hemoglobin	10.3 g/dl	Lactate dehydrogenase	168U/l
Hematocrit	30.7%	γ-Glutamyltranspeptidase	53 U/l
White blood cell	5,900/μl	Total bilirubin	0.4 mg/dl
Platelet	27.8×10 ⁴ /μl	Blood urea nitrogen	16 mg/dl
Amylase	1,390 U/l	Creatinine	0.9 mg/dl
Lipase	164 U/l	Fasting blood sugar	94 mg/dl
Trypsin	1,200 ng/ml	CA19-9	7 U/l
Total protein	6.9 g/dl	Carcinoembryonic antigen	1.6 ng/ml
Albumin	3.7 g/dl	DU-PAN-2	<25 U/ml

Figure 1. Chest X-ray demonstrates the elevation of the left diaphragm (arrow) and small amount of pleural effusion in the left.

not communicate with the pancreatic duct. Small cystic lesions communicating with the pancreatic duct were observed in the tail of the pancreas. The small cystic lesions demonstrated in the tail of the pancreas on CT and ERP were considered as pseudocysts due to chronic pancreatitis. The hematoma was probably caused by the exudative pancreatic fluid due to the rupture of the small pseudocysts with chronic pancreatitis.

Initially, we contemplated either percutaneous drainage of the hematoma or splenectomy. On US, however, the hematoma was found to contain numerous fine echoic spots that were immobile, thus it was determined impossible to perform percutaneous drainage. After admission, his serum pancreatic enzymes as well as anemia and serum level of C-reactive protein all improved. His subjective symptoms ameliorated, so that we

continued a conservative treatment. On US performed after 4 weeks (Fig. 7), the hematoma changed to be more echogenic with high echoic spots, which suggested that the content of the hematoma was undergoing coagulation.

On December 16, 1992, he complained of fever and dull pain in the left upper quadrant of the abdomen with sudden onset. The white blood cell increased to 10,400/μl, and serum amylase reached 471 U/l; a relapse of the pancreatitis was suspected. The symptoms and laboratory findings were improved by conservative therapy. On December 18, the white blood cells decreased to 6,700/μl, and serum amylase went down to 288 U/l. However, on the morning of December 24, he complained of severe abdominal pain accompanied by muscle guarding. Abdominal CT (Fig. 8) showed reduction of the hematoma size and ascites of the same density as the bloody content of the hematoma. Panperitonitis due to rupture of the hematoma was suspected, and an emergency operation was performed.

Laparotomy showed massive (about 800 ml) bloody, and colloidal fluid with clots in the splenic hematoma, and ascites (about 600 ml) of the same nature. Neither splenectomy nor splenectomy with partial resection of the pancreatic tail could be performed, because the pancreatic tail adhered so tightly to the spleen and peritoneum. Two drainage tubes were set into the hematoma and two additional drainage tubes were placed in the intraperitoneum. The amylase level of the fluid in the hematoma was 20,400 U/l. *Pseudomonas aeruginosa* was detected in the fluid. Fortunately, his postoperative clinical course was uneventful.

Discussion

Although the tail of the pancreas touches the hilus of the spleen, and the anterior surface of the pancreas is covered with the peritoneum enclosing the splenic vessels, hemorrhagic complications of pancreatitis involving the spleen are rare.

The present patient had abdominal US at a local clinic three or four times a year for examination of chronic pancreatitis, but his spleen appeared consistently normal size. The US revealed no abnormality three months before the discovery of the hematoma. Thus, the enlargement of his hematoma seemed to

Figure 2. Ultrasonography (US) shows the thin-walled cystic lesion is seen in the antero-lateral part of the spleen. The spleen compresses the cyst medially (A). No capsular formation between the cyst and the splenic parenchyma is shown (B). The cyst contains numerous fine echoic spots. The splenic parenchyma shows a normal texture (C). The pancreas exhibits an irregular contour suggestive of chronic pancreatitis (D).

Figure 3. Computed tomography (CT) of the abdomen demonstrates a large cystic lesion covered with the capsule of the spleen. It compresses the spleen laterally. Small pseudocysts are observed in the tail of the pancreas.

be very rapid. The hematoma was probably caused by the exudative pancreatic fluid due to the rupture of the small pseudocysts demonstrated on CT and ERP.

Despite the huge hematoma, we adopted conservative medical management. The size of the hematoma did not change during a 6-week period. After a relapse of the pancreatitis, the hematoma finally ruptured. The cause was considered to be infection.

The treatment for subcapsular hematoma of the spleen with pancreatitis is not established. It is reported that pseudocysts of the pancreas 5 cm or larger rarely resolve spontaneously. In the case of spontaneous resolution, it usually occurs within 6 weeks (6, 7). Bradley et al stated that the prolonged observation of pancreatic pseudocysts of more than 7 weeks after their onset in the anticipation of a spontaneous resolution exposes the risk of complications, such as rupture or abscess and emphasized that the length of such observation should not exceed 6 weeks (8). It is generally acknowledged that the spleen should be conserved whenever possible. Quinn et al reported two cases of intrasplenic pseudocysts due to pancreatitis and two cases of traumatic splenic hematoma undergoing percutaneous drainage (9). In the two cases of traumatic splenic hematoma and one case of intrasplenic pseudocysts, the drainage was successful,

Figure 4. Magnetic resonance imaging (MRI) of the abdomen shows high-intensity signals both T1 (A) and T2 (B)-weighted images in the subcapsular cystic lesion. The giant cyst is regarded as a hematoma.

but in the remaining case of intrasplenic pseudocysts sterile pleural effusion occurred by transpleural catheter position. Vyborny et al reported one case of a large splenic hematoma with chronic pancreatitis in which rupture of the splenic capsule was prevented and splenectomy was obviated by percutaneous drainage performed during the subacute phase (10).

From the clinical course of the present case, we concluded that a large splenic hematoma of larger than 5 cm as a complication of pancreatitis should be subjected to pressure reduction by percutaneous drainage at an early stage or laparotomy even if the clinical course has temporarily improved.

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Figure 5. Celiac angiogram demonstrates no evidence of encasement of the splenic artery, or the formation of a pseudoaneurysm. The capsular branches of the splenic artery are stretched.

Figure 6. Endoscopic retrograde pancreatogram (ERP) demonstrates the dilated pancreatic duct. The duct does not communicate with a cyst. Small pseudocysts are observed in the pancreatic tail.

Figure 7. Ultrasonography (US) shows the texture of the hematoma with various intensity of echogenicity (A, B), suggesting coagulation.

Figure 8. Computed tomography (CT) demonstrates the reduced hematoma and ascites (arrowheads).

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