A Case of Pancreatic Ascites and Pleural Effusion: Confirmation of a Pancreatic Duct Contrast Leakage Using Computed Tomography after Endoscopic Retrograde Cholangiopancreatography

Matsumoto, Kojiro; Tanigawa, Ken; Nakao, Hideto; Okudaira, Sadayuki; Yamada, Masashi; Eguchi, Katsumi

Acta medica Nagasakiensia. 2003, 48(1-2), p.73-76
Case Report

A Case of Pancreatic Ascites and Pleural Effusion: Confirmation of a Pancreatic Duct Contrast Leakage Using Computed Tomography after Endoscopic Retrograde Cholangiopancreatography

Kojiro Matsumoto1, Ken Tanigawa2, Hideto Nakao2, Sadayuki Okudaira3, Masashi Yamada3, Hiroki Kishikawa3, Katsumi Eguchi1

1) The First Department of Internal Medicine, Nagasaki University School of Medicine
2) Internal Medicine, Kohseikai Hospital
3) Surgery, Kohseikai Hospital

A seventy-two year old Japanese man with chronic alcoholism was admitted with increasing epigastric pain and abdominal fullness. He gave a history of bouts of epigastric pain radiating to the back for the past year. At admission, abdominal ultrasonography and computed tomography (CT) demonstrated massive ascites and a pseudocyst in the pancreatic body. A chest X-ray showed bilateral pleural effusion, and the level of amylase was elevated in the serum, urine, ascitic fluid and pleural effusion. First, the patient was treated with nothing by mouth but with intravenous hyperalimentation, however, no improvement was noted after 2 weeks. Then, the patient underwent endoscopic retrograde cholangiopancreatography (ERCP) and abdominal CT after ERCP. They showed irregular dilatation of the pancreatic main duct and branch, and an extravasation of contrast media from the pancreatic duct into the peritoneal cavity, after which the patient underwent surgery. Because no fistula was found during surgery, drainages were retained into the pseudocyst and peritoneal cavity. Due to marked elevation of amylase and protein levels in ascitic fluid and pleural effusion and findings from ERCP and CT after ERCP, pancreatic ascites and pleural effusion was diagnosed. The diagnosis of chronic pancreatitis is due to his history, laboratory data, and irregular dilatation of the pancreatic duct on ERCP. After surgery, his clinical status improved rapidly. We thus described a case of pancreaticoperitoneal fistula demonstrated by CT scan subsequent to ERCP which was treated successfully by surgery.

Address Correspondence: Kojiro Matsumoto, M.D.
The First Department of Internal Medicine, Nagasaki University School of Medicine, 1-7-1 Sakamoto, Nagasaki, 852-8501, Japan
TEL: +81-95-849-7262 FAX: +81-95-849-7270, E-mail: koujirou708@yahoo.co.jp

Key Words: pancreatic pleural effusion, pancreatic ascites

Introduction

Although complications of chronic pancreatitis are commonly pseudocyst formation, diabetes mellitus and malabsorption, pancreatic ascites and pleural effusions are being reported with increasing frequency because of the growth of alcoholism in Japan. In the case of the massive hemorrhagic ascites and pleural effusion, we should consider chronic pancreatitis as a differential diagnosis. Adequate information about the pathological anatomy by ultrasonography, computed tomography (CT), endoscopic retrograde cholangiopancreatography (ERCP), CT scan subsequent to the ERCP (ERCP-CT scan) and magnetic resonance imaging (MRI) are useful for diagnosis and success of therapy.

Case Report

A seventy-two year old Japanese man with chronic alcoholism was admitted to hospital complaining of a two week history of increasing epigastric pain and abdominal fullness. He gave a history of bouts of epigastric pain radiating to the back for the past year. His past history included hypertension and diabetes mellitus, but his family history was unremarkable. On admission, physical examination revealed marked distress, slight abdominal tenderness and fluctuation. The liver and spleen were not palpable. Laboratory findings: serum amylase was elevated up to 1,480 SU/l, urine...
amylase to 12,740 SU/l and C-reactive protein (CRP) to 7.1 mg/dl. Serum protein 5.3 g/dl was decreased. The ascitic fluid and pleural effusion were hemorrhagic and showed raised amylase (ascites 21,460 SU/l, pleural effusion 22,900 SU/l), lipase (32,920 SU/l, 34,470 SU/l) and protein (2.6 g/dl, 2.5 g/dl) values (Table 1). A chest X-ray showed bilateral pleural effusion (Fig. 1). Abdominal US and CT demonstrated massive ascites and a pseudocyst in the pancreatic body.

Table 1. Laboratory Findings on Admission

<table>
<thead>
<tr>
<th>WBC</th>
<th>5660 /mm³</th>
<th>Serum Amylase</th>
<th>1480 SU/l</th>
<th>Urine Amylase</th>
<th>1480 SU/l</th>
<th>Ascites Amylase</th>
<th>21,480 SU/l</th>
<th>S-U</th>
<th>Urine Amylase</th>
<th>21,480 SU/l</th>
<th>S-U</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>3.47 x 10⁷/mm³</td>
<td>Lipase</td>
<td>399 IU/L</td>
<td>Protein</td>
<td>2.6 g/dl</td>
<td>Culture Class</td>
<td>I</td>
<td></td>
<td>Lipase</td>
<td>32,920 SU/l</td>
<td>S-U</td>
</tr>
<tr>
<td>Hb</td>
<td>11.4 g/dl</td>
<td>Glucose</td>
<td>105 mg/dl</td>
<td>Creat</td>
<td>1.3 mg/dl</td>
<td>Pleural Effusion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ht</td>
<td>33.7 %</td>
<td>T.P</td>
<td>5.3 g/dl</td>
<td>BUN</td>
<td>25.9 mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pit</td>
<td>3.07 x 10⁴/mm³</td>
<td>T.Bill</td>
<td>1.1 mg/dl</td>
<td>Na</td>
<td>139 mEq/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T.P</td>
<td>5.3 g/dl</td>
<td>AST</td>
<td>26 IU/L</td>
<td>K</td>
<td>3.7 mEq/l</td>
<td>AmYLase</td>
<td>22,900 S-U</td>
<td>S-U</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT</td>
<td>13 IU/L</td>
<td>ALP</td>
<td>97 IU/L</td>
<td>Ca</td>
<td>8.4 mg/dl</td>
<td>Lipase</td>
<td>34,470 S-U</td>
<td>S-U</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDH</td>
<td>287 IU/L</td>
<td>ALP</td>
<td>97 IU/L</td>
<td>CRP</td>
<td>7.1 mg/dl</td>
<td>Protein</td>
<td>2.5 g/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>r-GTP</td>
<td>30 IU/L</td>
<td>ESR</td>
<td>22 mm/hr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

First the patient was treated with nothing by mouth but with intravenous hyperalimentation, however, no improvement was noted after 2 weeks. Then, the patient was underwent ERCP and abdominal CT after ERCP. They showed irregular dilatation of the pancreatic main duct and branch, and an extravasation of contrast media from the pancreatic duct into the peritoneal cavity (Fig. 2 and Fig. 3). Then, the patient was operated upon. There was a cyst behind the stomach and an abscess in front of the pancreas. Because no fistula was found during surgery, we thought it may be undetectably small. Only drainages were kept in the pseudocyst and peritoneal cavity. Due to marked elevation of amylase and protein levels in ascitic fluid and pleural effusion and findings from ERCP and CT after ERCP, pancreatic ascites and pleural effusion was

Clinical course: There were markedly elevated amylase and protein levels in the ascitic fluid and pleural effusion. We thought that the ascites and pleural effusion were caused by chronic alcoholic pancreatitis.

Figure 1. The chest X-ray showed bilateral pleural effusion

Figure 2. a. Endoscopic retrograde cholangiopancreatography showed extravasation of the contrast media from the pancreatic duct in the pancreatic body.
b. The main duct (large arrow) and branch (small arrow) in the body of the pancreas are dilated irregularly. These findings are observed in chronic pancreatitis.
Kojiro Matsumoto et al: A Case of Pancreatic Ascites and Pleural Effusion

Figure 3. Computed tomography subsequent to the endoscopic retrograde cholangiopancreatography demonstrated extravasation of the contrast media from the pancreatic duct into the peritoneal cavity.

diagnosed. The diagnosis of chronic pancreatitis is due to his history, laboratory data, and irregular dilatation of the pancreatic duct on ERCP. We did not make a secretin test and histology.

After surgery, the patient was relieved of abdominal pain and the ascites and pleural effusion did not reaccumulate. After 4 months, ERCP and abdominal CT demonstrated a disappearance of the cyst. The patient was discharged in good condition.

Discussion

A small amount of reactive pleural effusion and ascites commonly complicates acute pancreatitis. Although massive pleural effusion and ascites are rarely associated with chronic pancreatitis, they are being reported increasingly in connection with chronic alcoholic pancreatitis which is increasing in Japan.

Pancreatic pleural effusion and ascites share a common pathophysiology.\textsuperscript{1,3} Disruption of the pancreatic duct secondary to inflammatory pancreatic disease can result in an internal pancreatic fistula into the peritoneal or pleural cavities. If the duct disrupts anterior, pancreatic ascites results. In case of a posterior disruption of the duct, pancreatic secretions will generally track through the retroperitoneum into the mediastinum via the aortic or esophageal hiatus. If the secretions break through the mediastinal pleura, a fistula into pleural cavities can result. Recently the anatomical demonstration of a fistula can be shown by US, CT, MRI and ERCP.

We reviewed 158 cases of pancreatic pleural effusion complicated by chronic pancreatitis reported in Japanese literature between 1972 and 1999.\textsuperscript{4,6} One hundred fifty five cases were male and the cause in 97% of cases was chronic alcoholism. Though the main symptoms were reported dyspnea and/or chest and back pain, abdominal symptoms of epigastralgia was only 20%. Effusion was into the left pleural space (58%), the right space (26%) or into both spaces (16%). The volume of the fluid varied between 1000 and 3000ml. The pleural fluid was bloody in 80% of cases.

We reviewed 65 cases of pancreatic ascites reported in Japanese literature between 1975 and 1999.\textsuperscript{5,9,10} Fifty seven cases were male and the cause of 97% of cases was chronic alcoholism. Reported symptoms were abdominal fullness, epigastralgia and back pain, but there were patients who had no complaints. The volume of fluid varied between 1000 and 8000ml. The ascitic fluid was bloody in 70% of cases.

The diagnosis was confirmed biochemically by paracentesis of pancreatic pleural effusion and ascites, which demonstrated a markedly elevated amylase content and a protein level generally above 2.5g/dl. Our case also exceeded the normal level of amylase and protein level in ascites and effusion.

Anatomical diagnosis is confirmed by demonstration of a pancreatic pseudocyst and/or fistula. Ultrasonography, ERCP, and CT have been performed commonly and can be useful diagnostic tools. ERCP is the best method for diagnosis and to decide therapy because it can show the formation of the pancreatic duct, pseudocyst, fistula and contrast media leaking into pleural and peritoneal cavities. Furthermore, CT scan subsequent to the ERCP (ERCP-CT scan) can be useful for diagnosis.\textsuperscript{5,11} Recently, magnetic resonance imaging (MRI) has been reported more commonly and has been able to demonstrate internal pancreatic fistula.\textsuperscript{12}

Several authors recommend a conservative approach
for 2-3 weeks and then consider surgical therapy if there is no improvement. Conservation therapy is defined as the use of one or more of the following treatments: elemental diet, parenteral nutrition, paracentesis, or the more recently used somatostatin analogues. The aim of these treatments is to reduce pancreatic exocrine secretion, or evacuate ascites. First, our case was treated with nothing by mouth but with intravenous hyperalimentation, however, no improvement was noted after 2 weeks. So he underwent surgery. Lipsett et al have reported that 83% of patients were managed successfully by surgery. The selection of surgical procedure depends on information provided by an image, for example, pancreatic duct catastasis, the location and size of pseudocyst and/or fistula, adhesions with ambient organs and general condition. Therefore, adequate information of the pathological anatomy by US, CT, ERCP and MRI is important for success of diagnosis and management of surgical therapy.

In conclusion, we reported a case of pancreatic pleural effusion and ascites with a pancreaticoperitoneal fistula caused by chronic alcoholic pancreatitis. The fistula was demonstrated by CT scan subsequent to the ERCP, and the disease was treated successfully by surgery.

References