Title

Two cases of pancreatic abscess associated with penetration to the gastrointestinal tract during treatment using endoscopic ultrasound-guided drainage

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TWO CASES OF PANCREATIC ABSCESS ASSOCIATED WITH PENETRATION TO THE GASTROINTESTINAL TRACT DURING TREATMENT USING ENDOSCOPIC ULTRASOUND-GUIDED DRAINAGE

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Abstract: Of pancreatic pseudocysts, approximately 30% are complicated by abscess formation, perforation into the abdominal cavity, penetration to the gastrointestinal tract, or bleeding. We report two cases of pancreatic abscess complicating severe acute pancreatitis in which the abscess penetrated to the gastrointestinal tract during the course of treatment with endoscopic ultrasound (EUS)-guided pseudocyst drainage. In these cases, neither aggravation nor recurrence of the pancreatic abscess has been identified since the event occurred. The EUS-guided treatment was effective for improvement of severe inflammation of the pseudocyst as an initial treatment. However, drainage tube placement limitations pertained because the pseudocyst was present with multilocular infection. Penetrations eventually contributed to their resolution because the fistulas were used as wide drainage routes. It is important to understand the courses of these cases for preparation of therapeutic strategies to treat pancreatic pseudocyst/abscess.

Key words: pancreatic pseudocyst, penetration, EUS-guided drainage

INTRODUCTION

Pancreatic pseudocysts, which contain pancreatic juice and effusion, are formed by pancreatic inflammation. A secondary pseudocyst develops in 10–15% of
patients with acute pancreatitis and chronic pancreatitis\(^1\). Of those, approximately 30% are complicated by abscess formation, perforation into the abdominal cavity, penetration to the gastrointestinal tract, or bleeding (gastrointestinal bleeding, intra-abdominal bleeding, intracystic bleeding\(^2,3\)). About 20-60% of pancreatic pseudocysts will disappear spontaneously. However, that rate falls to less than 10% for pseudocysts of 6 cm diameter or greater. Therefore, if a cyst is not reduced, even after the sixth week when the cyst wall starts to organize and adhere to surrounding organs, cyst drainage is indicated\(^4,5\). On the other hand, a pancreatic pseudocyst associated with infection or pancreatic abscess might cause sepsis and multi-organ failure. It might penetrate into the gastrointestinal tract, cause bleeding, and form a pseudo-aneurysm if inflammation spreads to surrounding organs. Consequently, for conservative treatment of uncontrolled intracystic infection, immediate drainage is required, even if in the early phase of pseudocyst formation.

We have recently experienced two cases of pancreatic abscess complicating severe acute pancreatitis in which the abscess penetrated to the gastrointestinal tract during treatment with endoscopic ultrasound (EUS)-guided pseudocyst drainage. In these cases, penetrations eventually contributed to their resolution because the fistulas were used as wide drainage routes. The courses of these cases should be understood for preparation of therapeutic strategies of pancreatic pseudocyst/abscess.

**CASE REPORT**

**Case 1**

A 65-year-old man was admitted to another hospital because of severe acute pancreatitis. Although the pancreatitis was relieved using various treatments, pancreatic pseudocyst developed around the pancreas. After eating was restarted, he displayed a high fever (39°C); infection of the pseudocyst was suspected. He was then referred to our hospital, to which he was admitted for treatment. Laboratory tests performed on admission indicated a severe inflammatory reaction with WBC of 21,200/\(\mu l\) and CRP of 12.7 mg/dl. Mild anemia, mild thrombocytopenia, liver dysfunction, and marked hypoalbuminemia were also indicated. Renal functions, blood glucose, serum AMY, and tumor markers were normal (Table 1). Abdominal CT showed a 12-cm-diameter abscess around the pancreatic tail and another abscess around the pancreatic head (Fig. 1). It was considered that immediate drainage for abscess should be done. Therefore, EUS-guided drainage was performed using a 19-gauge puncture needle (Echotip19G; Wilson–Hirata, Osaka, Japan) guided by ultrasound using a convex type echoendoscope (GF-UCT240AL-5; Olympus Medical Systems. Co., Tokyo, Japan) (Fig. 2a) with a transgastric approach. After puncture of the abscess, the needle stylet was removed. The aspirated fluid, viscous, white, and slightly bloody, was collected. A 0.025-inch guide wire (Jagwire; Boston Scientific Japan, Tokyo) was inserted into the needle.
Table 1. Laboratory tests of Case 1
(on admission)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>WBC</td>
<td>21,200/μl</td>
<td>TP</td>
<td>6.2 g/dl</td>
</tr>
<tr>
<td>RBC</td>
<td>3.49×10^6/μl</td>
<td>ALB</td>
<td>1.6 g/dl</td>
</tr>
<tr>
<td>Hb</td>
<td>10.9 g/dl</td>
<td>BUN</td>
<td>18 mg/dl</td>
</tr>
<tr>
<td>Hct</td>
<td>32.9%</td>
<td>CREA</td>
<td>0.6 mg/dl</td>
</tr>
<tr>
<td>PLT</td>
<td>13.8×10^9/μl</td>
<td>Na</td>
<td>137 mEq/l</td>
</tr>
<tr>
<td>TB</td>
<td>1.6 mg/dl</td>
<td>Cl</td>
<td>102 mEq/l</td>
</tr>
<tr>
<td>DB</td>
<td>1.1 mg/dl</td>
<td>AMY</td>
<td>82 IU/l</td>
</tr>
<tr>
<td>AST</td>
<td>162 IU/l</td>
<td>BS</td>
<td>128 mg/dl</td>
</tr>
<tr>
<td>ALT</td>
<td>73 IU/l</td>
<td>CEA</td>
<td>1.5 ng/ml</td>
</tr>
<tr>
<td>LDH</td>
<td>391 IU/l</td>
<td>CA19-9</td>
<td>29.1 U/ml</td>
</tr>
<tr>
<td>y-GTP</td>
<td>153 IU/l</td>
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Fig. 1. Abdominal CT showed a 12-cm-diameter abscess around the pancreatic tail and another abscess around the pancreatic head in case 1.

and was advanced into the abscess. Then a 6 Fr endoscopic naso-biliary drainage (ENBD) tube (Flexima; Boston Scientific Japan, Tokyo) was placed in the abscess over the guide wire (Fig. 2b). The abscess cavity was washed every day with saline mixed with amikacin sulfate. After this treatment, although the inflammatory reaction decreased and the patient’s general condition improved, follow-up CT revealed residual abscess cavities. Two weeks after the drainage, hematochezia was observed. Emergent colonoscopy revealed two ulcerative lesions with mucosal edema in the transverse colon; penetrations of pancreatic abscesses into the colon were suspected (Fig. 3). Images obtained by CT scanning revealed an abscess containing air near the pancreatic tail. Pseudo-aneurysm was not seen in the locations of the fistulas. Fortunately, apparent anemia and an inflammatory reac-
Fig. 2.  

a: EUS-guided drainage was performed using a 19-gauge puncture needle guided by ultrasound using a convex type echoendoscope with the transgastric approach.

b: An endoscopic naso-biliary drainage (ENBD) tube was placed in the abscess.

Fig. 3. Colonoscopy revealed two ulcerative lesions with mucosal edema in the transverse colon. Penetrations of pancreatic abscesses into the colon were suspected.

tion were not identified. Therefore, conservative treatments were performed. An abdominal CT scan performed 2 weeks after the hematochezia showed communication between the abscesses and the transverse colon, as well as the reduced abscesses (Fig. 4). Since the event occurred, neither aggravation nor recurrence of the pancreatic abscess has been identified.
Case 2

A 53-year-old man with a history of choledocholithiasis was admitted to another hospital with a diagnosis of severe acute alcoholic pancreatitis. The pancreatitis was relieved through treatment with protease inhibitors and antibiotics under a fasting condition. Nevertheless, the patient developed a fever of 39°C and abdominal pain. Abdominal CT images suggested infection of a pancreatic pseudocyst associated with acute pancreatitis. For treatment, he was referred to our hospital. On admission, laboratory tests showed WBC of 9,900/µl, CRP of 11.8 mg/dl, mild anemia, thrombocytosis, and mild liver dysfunction. Renal function

<table>
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<tr>
<th>Table 2. Laboratory tests of Case 2 (on admission)</th>
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<tbody>
<tr>
<td>WBC 9,900/µl</td>
</tr>
<tr>
<td>RBC 376×10^6/µl</td>
</tr>
<tr>
<td>Hb 11.9 g/dl</td>
</tr>
<tr>
<td>Hct 33.7%</td>
</tr>
<tr>
<td>PLT 51.7×10^6/µl</td>
</tr>
<tr>
<td>TB 0.5 mg/dl</td>
</tr>
<tr>
<td>DB 0.3 mg/dl</td>
</tr>
<tr>
<td>AST 23 IU/l</td>
</tr>
<tr>
<td>ALT 25 IU/l</td>
</tr>
<tr>
<td>LDH 251 IU/l</td>
</tr>
<tr>
<td>ALP 479 IU/l</td>
</tr>
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and serum AMY were normal; a marked increase of blood glucose was not observed (Table 2). Abdominal CT revealed extensively developed abscesses around the pancreas (Fig. 5). It was considered that immediate drainage should be performed. Therefore, EUS-guided drainage was performed using the same procedure as that used in “case 1” (Fig. 6a). Yellow and white pus was aspirated from the abscess. At that time, a 6 Fr ENBD tube (Flexima; Boston Scientific Japan, Tokyo) was indwelled in the abscess (Fig. 6b). The abscess cavity was washed through the tube daily with saline mixed with amikacin sulfate. Improvement of fever and the inflammatory reaction were apparent after the drainage. Two weeks later, X-ray fluoroscopic examination was done by injection of contrast medium via
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Fig. 7. On the fluoroscopic image, the small intestine was visualized in images taken inside of the abscess cavity (arrow), suggesting that the pancreatic abscess had penetrated into the small intestine.

Fig. 8. Abdominal CT showed the reduced pancreatic abscess. Contrast medium was recognized in the small intestine and the abscess cavity.

an ENBD tube to decrease the pancreatic abscess. Then, the small intestine was visualized in images taken inside the abscess cavity, which suggested that the pancreatic abscess had penetrated into the small intestine (Fig. 7). Subsequently, CT showed the reduced pancreatic abscess (Fig. 8). During the course of the case, abdominal pain, melena, inflammatory reaction, and anemia were not seen. Since that event occurred, neither aggravation nor recurrence of the pancreatic abscess has been identified.
DISCUSSION

Pancreatic pseudocysts are classifiable into three types depending on their etiology: type I, pseudocyst complicating acute pancreatitis; type II, pseudocyst complicating the aggravation of chronic pancreatitis; or type III, chronic pseudocyst [retention cyst]. Both type I and type II described above are conditions of post-necrotizing pseudocyst. The space of the omental sac between the front of the pancreas and the stomach becomes a cyst cavity when the inflammation spreads from the pancreas. The exudate is retained in that space, and a pseudocyst is formed because Winslow's foramen is closed by severe inflammation. Type III is a cyst within the pancreas. Although the cyst wall might adhere to the gastrointestinal tract because of repeated inflammation in chronic pancreatitis, the cyst wall is usually separated from the wall of the gastrointestinal tract. Generally, type I and type II cysts, which develop secondary to acute pancreatitis and acute aggravation of chronic pancreatitis, tend to be complicated by bleeding, infection, and penetration into the gastrointestinal tract. In both cases described in this report, pancreatic pseudocysts complicating severe acute pancreatitis (above-mentioned type I) were infected, consequently forming pancreatic abscesses. They were at high risk of sepsis and might have bled severely by prolonged inflammation. Therefore, immediate drainage was necessary. As noted previously, the cyst wall might include the gastric wall itself, and it was considered that endoscopic retrograde pancreatography-guided transpapillary drainage might aggravate the acute pancreatitis. For those reasons, EUS-guided trans-gastrointestinal drainage was chosen. In both cases, inflammatory reactions improved gradually after drainage, showing the effect of washing through the drainage tube. Our treatment was inferred to be effective as an initial treatment.

Pancreatic pseudocyst is known to perforate into the abdominal cavity and the retroperitoneum, and to penetrate into the gastrointestinal tract. The frequency of penetration into the gastrointestinal tract is approximately 15-20% among pancreatic pseudocysts. In the case of a pseudocyst complicating acute pancreatitis, the partial wall of the pseudocyst was composed of the wall of the gastrointestinal tract, and adhered to the surrounding gastrointestinal tract because of severe inflammation. The pancreatic enzymes in the pseudocyst weaken the gastrointestinal tract wall. Consequently, the pseudocyst might penetrate into the gastrointestinal tract and naturally generate an internal fistula. Moreover, internal pressure of the pseudocyst caused by hemorrhage might involve penetration into the gastrointestinal tract. To our knowledge, there are some such cases of penetration into other organs in the literature. Of those, the most frequently penetrated organ was the stomach (9 cases); other organs including the duodenum (4 cases), transverse colon (4 cases), spleen (4 cases), aorta (1 case), and bile duct (1 case) were also penetrated (two or more organs were penetrated in some
cases). No penetration has ever been recorded into the small intestine, as occurred in case 2. The fistula which was formed by penetration into the gastrointestinal tract imparted a drainage effect that might have eliminated some pseudocysts naturally. In both cases reported here, naturally generated internal fistulas as a complication eventually contributed to elimination of the pseudocyst.

Pseudocysts complicating acute pancreatitis often form multilocular cysts because of widely expanded inflammation. In recent years, EUS-guided drainage has been performed widely as a treatment for pancreatic pseudocyst including abscess and infectious pancreatic necrosis. Many studies have examined the usefulness of the technique\(^{20,21}\). As an initial treatment for advanced inflammation, EUS-guided drainage is effective. On the other hand, limitation of the drainage tube placement in the case of multilocular infection is known. In such cases, multiple drainage tubes might have been required to be punctured from other sites. Additionally, because of the large amount of necrotic debris contained in the abscess, debris removal might be distressful, even with larger drainage tubes, and sufficient treatment might be difficult. In such cases, an endoscopic approach into the pseudocyst through the gastrointestinal tract wall (endoscopic necrosectomy) should be performed\(^{22,23}\).

In our case 1, pancreatic abscesses penetrated into the transverse colon and caused bleeding. Further massive bleeding in the cyst and the abdominal cavity might occur if inflammation involves major vessels near the pseudocyst and forms a pseudo-aneurysm\(^{24,25}\). Therefore, as noted above, trans-gastrointestinal endoscopy-guided necrosectomy should be considered early when EUS-guided drainage is not effective.

In conclusion, EUS-guided treatment was effective for improvement of severe inflammation of a pseudocyst as an initial treatment. However, limitations of the drainage tube placement pertained in these cases of pseudocyst with multilocular infection. For treatment of pseudocyst/abscess complicating acute pancreatitis, a therapeutic strategy should include consideration of the pathogenic mechanism and configuration, with particular attention to the potential secondary complications.

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