SUCCESSFUL TREATMENT OF DISCONNECTED PANCREATIC TAIL SYNDROME WITH PERCUTANEOUS AND ENDOSCOPIC DRAINAGES

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Abstract

Disconnected pancreatic tail syndrome (DPTS) is characterized by the lack of ductal continuity between upstream viable pancreatic tissue and the downstream. It is an increasingly recognized complication of severe acute pancreatitis and in association with a persistent pancreatic fistula or pancreatic-fluid collection. A 59-year-old man developed necrotizing acute pancreatitis after endoscopic retrograde cholangiopancreatography. Computed tomography scan showed that the viable pancreatic tissue was about 20% of the pancreas and mainly in the pancreatic tail, and a huge fluid collection along the location of the original pancreas was found. Endoscopy showed that the pancreatic duct orifice was atrophied and stenting through the nature orifice was impossible. Although debridement and reconstruction are generally the recommended treatments for DPTS, we do not consider operation since it will keep even less viable pancreatic tissue. Trying to preserve the residual pancreatic tissue, we treated the patient with combination of percutaneous drainage and endoscopic stenting through a duodenostomy that was created by a needle knife at an optimal timing. Abdominal pain subsided after the therapy and the percutaneous drainage tubes were removed uneventfully. Though diabetes did occur, the serum glucose could be well controlled with low dose oral anti-glycemic drug.

Key words: Disconnected pancreatic duct syndrome, Percutaneous drainage, Endoscopic drainage, Endoscopic retrograde cholangiopancreatography, Pancreatic cyst

Introduction

Disconnected pancreatic tail syndrome (DPTS) is characterized by complete disruption of main pancreatic duct (P-duct) resulting in a variable portion of the upstream pancreatic parenchyma to disconnect from main P-duct downstream¹². This phenomenon is also called disconnected pancreatic duct syndrome³⁴. DPTS is an increasingly recognized complication of severe acute pancreatitis (SAP). The upstream viable pancreatic tissue from the transection will continue to

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Disconnected pancreatic tail syndrome

Secrete pancreatic juice through the point of disruption without access to the duodenum, resulting in a persistent non-healing pancreatic fistula or pancreatic-fluid collection. Therefore, patients with DPTS represent an extremely difficult therapeutic dilemma and a multidisciplinary approach is mandatory to optimize outcomes. We describe a patient with DPTS that is unique by the only viable tissue in the pancreatic tail and without a P-duct connected to the duodenum. He is successfully treated with combination of percutaneous and endoscopic drainages.

Case report

A 59-year-old man underwent endoscopic retrograde cholangiopancreatography (ERCP) for choledocholithiasis. He developed SAP after ERCP. Eight days later, computed tomography (CT) scans revealed pancreatic necrosis (about 80%) in the head, most of the body, and some of the tail of the pancreas. Ten days later, the patient started with continuous jejunal feeding via a naso-jejunal tube. Twenty-one days later, CT scans showed complete disappearance of the pancreatic head and most of the body. Instead, a 20-cm pancreatic cyst with multiloculated portion near the duodenum formed along the original pancreatic axis (Fig. 1). Complete obstruction of the splenic vein resulted in left-sided portal hypertension was also noted. Percutaneous drainage (PD) was performed because of abdominal pain caused by the expanding cyst. The amylase and lipase concentrations in the cystic fluid were 63781 U/L and 52645 U/L, respectively. The daily drainage volume was almost constant (190 ± 10 mL/day) after PD. Follow-up of two serial CT scans revealed that the cyst had regressed but the size remained constant (Fig. 2). Therefore, endoscopic therapy was carried out 21 days after PD. Transpapillary drainage could not be performed since the P-duct orifice was completely atrophied (Fig. 3). Transgastric drainage could not be performed because the cyst had reduced in size after PD. Although there was no bulge into the duodenum marking the location of the

Fig. 1. Computed tomography scan obtained 21 days after the onset of pancreatitis showed the huge pancreatic cyst and the little viable tissue in the pancreatic body and tail (arrows). Note complete disappearance of the pancreatic head due to necrosis.

Fig. 2. Computed tomography scan obtained 19 days after percutaneous drainage (arrowhead) showed that the pancreatic cyst had regressed, but persisted as a residual cyst (C), with a multiloculated part (c). Note the cyst was next to the duodenal C-loop. Arrows indicated the nasojejunal tube.
cyst, we realized that the residual cyst was beside the duodenal C-loop by CT (Fig. 2) and endoscopic ultrasonography. After insertion of a biliary stent (as a biliary marker under fluoroscopy), a diathermy needle (Microknife, Boston Scientific) was used to puncture the duodenal wall presumed to be the original P-duct orifice directly and to contact the residual cyst successfully. This approach allowed us to insert a catheter into the multiloculated area of the cyst and perforate the septa by using a 6-mm balloon dilator (Hurricane; Boston Scientific) (Fig. 4). Two double pigtails and one nasocystic catheter were placed in the pancreatic cyst. The cyst disappeared gradually and the PD catheter was successfully removed 10 days after the endoscopic therapy.

After the onset of SAP, the patient was in hyperglycemia all the time and lost 10 kg of his body weight. He discharged 58 days after the onset of SAP with the pancreaticoduodenal stents and was prescribed oral hypoglycemic agent (acarbose) for hyperglycemia. He regained 6 kg of body weight 3 months after discharge. In the one year of follow-up, there was no recurrence of the pancreatic cyst or pancreatitis.

Discussion

Methods of managing the persistent non-healing pancreatic-fluid collection resulting from DPTS are redirection of pancreatic secretion endoscopically, resection of the disconnected pancreas and conservative treatment until the disconnected pancreas undergoing atrophy. However, preserving the little viable pancreatic tissue in the body and tail is important in terms of maintaining the endocrine and exocrine function (at least partially). Therefore, in the third week after the onset of SAP, we faced a therapeutic dilemma when the abdominal pain worsened due to the expanding cyst. Although surgery is generally recommended for the treatment of DPTS, it is associated with high morbidity and mortality. More importantly, surgery may reduce volume of the viable pancreatic tissue.

Endoscopic drainage was not performed at
Disconnected pancreatic tail syndrome

first because (1) the cyst is immature. Late organized necrosis (at least 4 weeks after SAP) is an important prerequisite for successful endoscopic treatment (2). In our experience, the effect of early endoscopic drainage for a multiloculated pancreatic cyst is often not adequate. Although there is no literature to support the latter point of view, Venu et al. reported that the success of endoscopic drainage is less for multiloculated pancreatic abscess.

Therefore, we performed PD for the symptomatic pancreatic cyst. In general, PD cannot be successfully used for DPTS as it was in this case. However, PD might have some role on the success of overall treatment: (1) PD acted as a bridge to endoscopic therapy, preventing early endoscopy. (2) Combined therapies may improve the outcome of DPTS. Santvoort et al. recently reports a step-up approach with followed PD by minimally invasive retroperitoneal necrosectomy to reduce the rate of major complications and deaths compared with open necrosectomy in patients with infected necrotizing pancreatitis, and emphasizes the important role of PD. Furthermore, PD may reduce size of the cyst and that would make manipulation of endoscopic transmural drainage easier.

Endoscopic drainage is eventually performed to treat the persistent pancreatic cyst and fistula after PD. The failure rate is 23% for endoscopic drainage of DPTS related fluid collection and the recurrence rate is 43.8% for patients with DPTS undergoing successful endoscopic treatment. In this case, we show that endoscopic transduodenal drainage could be safely and successfully performed from the site nearby the original P-duct orifice if the pancreatic cyst is in next to the duodenal loop. This approach is somewhat like the endoscopic transpapillary drainage, and allows us to perforate the intracystic septa within the cyst for better drainage. The perforation of the septa might be part of the successful endoscopic procedures. Furthermore, we did not remove the pancreaticoduodenal stents to prevent recurrence of the pancreatic cyst. The high recurrence rate reported by Lawrence et al. may due to removing the transmural stents once resolution of the fluid collection is documented. Because the viable pancreas will continue to secret, long-term placement of transmural stents would be better to prevent the recurrence of pseudocyst.

Some may question the diagnosis of DPTS in this patient because there was no pancreatography to document complete disruption of the main P-duct. However, giving the fact that complete atrophied P-duct orifice and disappearance of pancreatic tissue in head on endoscopy and CT scans, one can not expect that there was still a viable and patent P-duct connected to the duodenum.

In conclusion, although surgery is generally recommended for DPTS, this case shows that DPTS can be treated with non-surgical methods which at least partially maintained the exocrine and endocrine functions in this patient.

References

patients with disconnected duct syndrome after severe acute pancreatitis. Surgery 2001;130:714-719.
合併經皮引流術及内視鏡成功治療胰尾不連續症候群

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摘要

胰尾不連續症候群（Disconnected pancreatic tail syndrome）乃指上行主胰管斷裂所致之臨床表現。此症候群與嚴重型急性胰腺炎所併發之胰周管或胰液滲漏有關。本文報告一59歲男性於接受經內視鏡逆行性胰管造影術後，併發壞死性急性胰腺炎；其電腦斷層攝影發現原有之胰臟多為液體所取代，胰臟組織僅剩20%，且集中於尾部。內視鏡下發現胰管開口萎縮，經此直接由內視鏡置入支架無法達成。現有報告之治療方式較多採手術清創及重建，惟本案例所剩胰臟組織過少，手術可能造成剝除胰臟組織更為不足，故吾人仍採用合併經皮引流術與內視鏡治療，期可解決患者之臨床症狀與保存剝除之胰臟組織。內視鏡支架乃於適當之時機下，藉由針刀所創之造口置入；患者接受此療法後，解除腹痛，且最終成功拔除經皮引流管，雖然仍產生糖尿病，但可以低劑量口服降血糖藥控制。

關鍵詞：胰尾不連續症候群，經皮引流術，內視鏡引流術，經內視鏡逆行性胰腺管造影術，胰臟囊腫

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