

Pancreatic Head Enlargement Associated with a Pancreatitis- Induced Intrasplenic Pseudocyst in a Patient with Chronic Pancreatitis: Organ Preserving Surgical Treatment

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Summary

Intrasplenic pseudocyst is a rare form of a late complication of chronic pancreatitis. We report the case of a 30-year-old man with an intrasplenic pseudocyst associated with chronic alcoholic pancreatitis. The patient was admitted with the third acute phase of chronic relapsing pancreatitis. Abdominal US and CT showed a large cyst in the pancreatic tail with involvement of the spleen. ERCP revealed marked irregularities of the main pancreatic duct without communication to the large cyst and a narrowing of the distal common bile duct by chronic pancreatitis of the head of the pancreas. Organ preserving surgical treatment with duodenum-preserving resection of the head of the pancreas combined with distal pancreatectomy and splenectomy was performed. This procedure may be indicated in selected patients to preserve functional pancreatic tissue and prevent diabetes. It should be in the armamentarium of the specialized pancreatic surgeon.

Key words: chronic pancreatitis, pancreatic pseudocyst, intrasplenic pseudocyst, surgical treatment

Introduction

Late complications of acute pancreatitis, or of an acute phase of chronic relapsing pancreatitis, are diverse. The most common is formation of a pancreatic pseudocyst appearing in approximately 50% of acute and 25% of chronic pancreatitis cases (1). As the pancreas and the spleen are in close proximity, late complications may also involve the spleen in form of splenic vein thrombosis, splenic rupture and necrosis, or severe bleeding from eroded splenic vessels. The formation of a perisplenic or even intrasplenic pseudocyst in the course of acute or relapsing chronic pancreatitis is extremely rare. Since 1941, when Roton et

al. (2) first described an intrasplenic cyst due to an ectopic pancreas, approximately 40 cases have been reported in the international literature (3).

We report a case of chronic alcoholic pancreatitis with both, involvement of the head and tail of the pancreas associated with formation of an intrasplenic pseudocyst and describe the organ preserving surgical treatment.

Case report

A 30-year-old man was admitted to our hospital with the third phase of an acute relapsing chronic alcoholic pancreatitis. The diagnosis was made 6 years earlier by ERCP and elevated serum amylase levels with values up to fivefold greater than the normal range. Since then, he had reduced his alcoholic consumption and remained free of symptoms. In July 1994, the patient had a new, painful attack with left lateral discomfort and back pain and complained of a feeling of abdominal fullness. The physical examination showed a mild abdominal tenderness without any mass or organomegaly. Laboratory findings remarked a mild leukocytosis of 10700/mm³, serum amylase level of 770 U/I (-200), lipase 1930 U/I (-190), GOT 426 U/I, GPT 133 U/I, GGT 666 U/I, APH 133 U/I (-110) and total bilirubin of 29 µmol/l (-17) with normal electrolytes.

An abdominal ultrasound demonstrated pancreatitis with mild swelling of the pancreas and mild peripancreatic effusion. A large hypoechogenic area was detected in the left upper abdomen. CT scan was performed demonstrating a cyst measuring 15x13x10 cm compressing the spleen in the medio-caudal axis (**Figure 1**). In the head of the pancreas, a small pseudocyst of 1 cm in diameter was detected, but no pancreatic necrosis or calcifications were seen. The common bile duct was enlarged to 9 mm due to compression in the region of the papilla of Vater. An ERCP revealed ductular changes of the main pancreatic duct and side branches in the head, with small cystic cavities in the head of the pancreas. The common bile duct showed a marked compression. There was no communication found between the pancreatic main duct and the large cyst in the left upper abdomen (**Figure 2**).

To solve the problem of chronic pancreatitis of the head of the pancreas obstructing the common bile duct with cholestasis, a duodenum-preserving resection of the head of the pancreas was performed at the same time (4). At the end of the two resectional procedures, the remnant of the pancreatic corpus was 4 cm long with a macroscopically normal appearance and a normal pancreatic duct caliber.

Reconstruction after resection of the head of the pancreas was initiated by dissecting the uppermost jejunal loop 40 cm distally to the ligament of Treitz. The aboral jejunal loop was brought up, and a two-row, layered, end-to-side anastomosis was carried out between the proximal part of the remnant pancreas and the jejunal loop (**Figure 4**). A side-to-side anastomosis between the jejunal loop and the remnant of the pancreatic head (approx. 1 cm large) at a distance of approximately 6 cm followed in the same technique (**Figure 4**). To restore bile flow in the upper intestine, an additional anastomosis between the prestenotic common bile duct and the jejunal loop was necessary because of incomplete decompression of the intrapancreatic segment of the common bile duct. The end-to-side anastomosis between the common bile duct and the jejunum was sutured using a single-layer technique. As mandatory in these patients, cholecystectomy followed this procedure. To prevent possible pancreatic fistula on the resection surface of the tail of the pancreas and at the same time to enhance the drainage of pancreatic secretion, a third end-to-side, two-row anastomosis between the distal part of the remnant pancreas and the end of the jejunal loop was performed (**Figure 4**). Finally, a typical Roux-en-Y anastomosis was carried out 40 cm distally to the pancreatico-jejunal anastomosis. Two intestinal drains were placed for anastomosis drainage. To prevent postoperative complications such as peripancreatic fluid collection, leakage and fistula formation, perioperative inhibition of exocrine pancreatic secretion was provided by administering octreotide (Somatostatin, Sandoz, Basel, Switzerland) 200 µg/tid for 6 days.

The postoperative course was uneventful. No leakage was found by normal amylase levels in the drains, which were taken out 3 days after operation. The laboratory follow up tests were within normal limits, including the liver function tests and amylase and lipase. There was no diabetes induced by regular diet. The glucose levels were between 4-5.5 µmol/l (3.66-5.55 µmol/l). Ten days after surgery, the patient was discharged free of symptoms.

Discussion

The formation of pseudocysts is one of the most frequent complications of acute or relapsing chronic pancreatitis. Pancreatic pseudocysts are known to expand along the tissue planes into areas far away from the pancreas. So Hamm et al. (5) found a frequency of 22% of atypically located pancreatic pseudocysts in the liver, spleen, stomach wall or even mediastinum. However, formation of

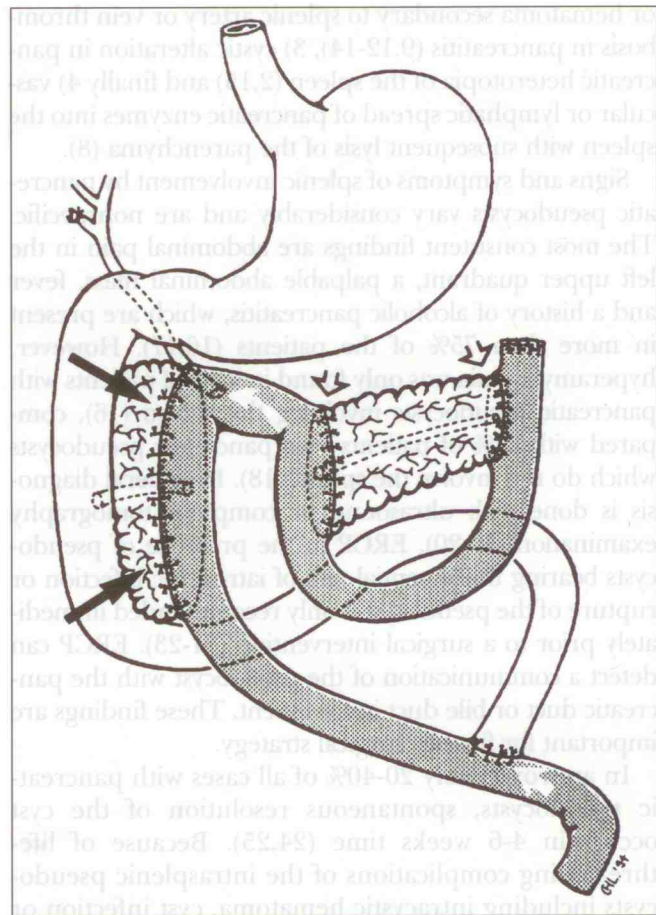


Figure 4 Schematic drawing of postoperative reconstruction in duodenum-preserving resection of the head of the pancreas, splenectomy and distal pancreatectomy. Side-to-side pancreatico-jejunal anastomosis (black closed arrows). Choledocho-jejunal anastomosis (white bended arrow). End-to-side pancreatico-jejunal anastomosis (black open arrows). Jejunoo-jejunal anastomosis Roux-en-Y (white closed arrow).

an intrasplenic pancreatic pseudocyst is rare (1,3,6). The small incidence of pancreatic pseudocyst extension into the spleen is approximately 1% of patients with acute or relapsing pancreatitis (6). Intrasplenic pseudocysts are seen to be pancreatitis-induced if 1) there is pancreatic tissue histologically proved inside the cyst or in direct contact with the pseudocapsule of the cyst, 2) if puncture of the cyst shows a higher amylase level as in the serum or 3) as in our case, there is a spontaneous formation of an intrasplenic cyst in a patient with well known pancreatitis without having suffered a previous trauma or a primary affection of the spleen (7,8). There are four possible formation mechanisms suggested: 1) direct dissection by pancreatic enzymes along the splenic hilum (9-11), as suspected in our case, 2) liquefaction of a splenic infarct

