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Floating Pancreatic Duct Concrements in Chronic Pancreatitis

Pain Relief by Endoscopic Removal

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Summary

This report describes 3 patients with chronic relapsing pancreatitis, floating pancreatic duct concrements between 4 and 6 mm in diameter, moderate to advanced ductal changes, and repeated severe attacks of pain during acute relapses over a period of several months. Immediate relief of pain was achieved in all 3 patients by endoscopic papillotomy aimed at widening the main pancreatic duct and subsequent extraction or spontaneous passage of pancreatic duct concrements. On the basis of our experience with the patients presented here, endoscopic papillotomy widening the main pancreatic duct may be useful in some patients with chronic pancreatitis and floating pancreatic duct concrements.

Key-Words: Chronic pancreatitis, Endoscopic papillotomy, Endoscopic retrograde cholangio-pancreatography, Pancreatic duct concrements.

Introduction

Pancreatic calcification is one of the main morphological features in chronic pancreatitis (2, 13), presenting as pancreatic duct concrements, either alone or together with parenchymal calcifications.

Among the various conditions responsible for the development of acute relapses and pain in chronic pancreatitis, a build-up of pressure in the pancreatic duct behind a stenosis has been postulated (17). Pancreatic duct stenosis can be caused by inflammatory stricture, a tumor or pancreatic duct concrements (12), which can be reliably identified by endoscopic retrograde pancreatography (7). Pancreatic duct concrements differ considerably in size, varying between 2 and 8 mm in diameter (9) and may adhere to the pancreatic duct wall (6) or float around upon injection of contrast medium into the pancreatic duct system during ERP (12). Treatment of pain in chronic pancreatitis, caused by floating pancreatic duct concrements, should logically consist in their complete removal from the pancreatic duct system, as demonstrated in the following 3 patients.

Patient 1

A 38-year-old male, in whom chronic alcohol consumption was reliably excluded and who suffered repeated attacks of acute pancreatitis over a period of 4 years, was admitted for evaluation of upper abdominal pain and a weight loss of about 15 kg over the previous 12 months. Physical examination revealed tenderness of

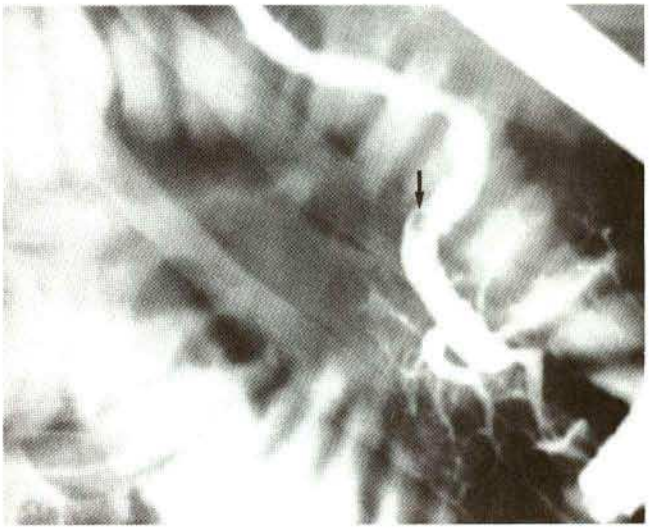

Fig. 1a

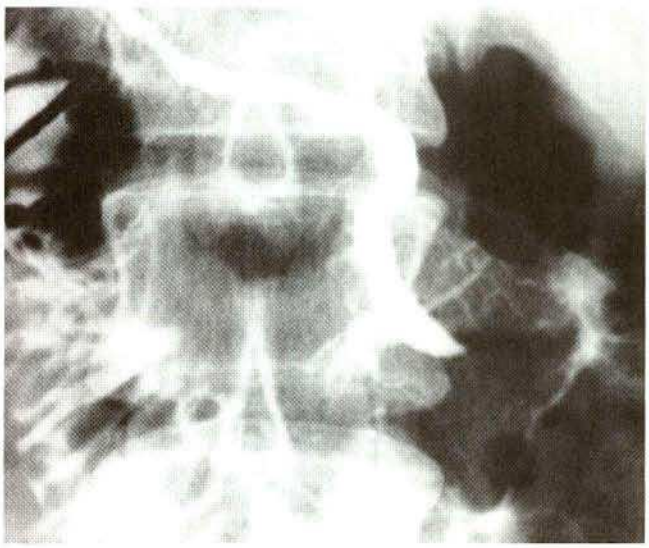

Fig. 1b

Fig. 1 Endoscopic retrograde pancreatogram in patient 1 with moderate chronic pancreatitis and a single pancreatic duct concrement (→) before (a) and after (b) endoscopic papillotomy to widen the main pancreatic duct and permit spontaneous passage of the concrement

the upper abdomen as the sole pathological finding. Laboratory analysis, including repeated determination of pancreatic isoamylase and pancreatic lipase, revealed normal results except for fasting serum glucose (271 mg%; normal 70–100). A plain film of the abdomen and ultrasonography failed to show any pancreatic calcifications. The secretin-pancreozymin test (15) indicated moderate exocrine pancreatic function impairment with a 50% reduction in stimulated bicarbonate and enzyme output. Endoscopic retrograde pancreatography (Fig. 1a) revealed moderate chronic pancreatitis (7, 8) with dilatation of the main pancreatic duct and rarefication of the side branches, especially in the body and tail of the pancreas. In addition, ERP identified a single pancreatic duct concrement, 5 mm in diameter, floating in the pancreatic duct upon injection of contrast medium. Treatment comprised endoscopic papillotomy to open the main pancreatic duct and subsequent spontaneous passage of the pancreatic duct concrement (Fig. 1b).

Patient 2

A 40-year-old male with an average daily alcohol intake of about 80 g over 5 years and two attacks of acute pancreatitis in 1981 and 1983, presented with upper abdominal pain of varying intensity. Physical examination detected tenderness in the middle and right upper abdomen, and meteorism. Besides a slightly elevated γ-GT (33 U/l; normal 6–28), total amylase and pancreatic isoamylase, together with leucocyte counts were repeatedly elevated 2–3 fold following oral alimentation. A plain film of the abdomen, ultrasonography and computed tomography demonstrated parenchymal calcifications in the head of the pancreas. Exocrine pancreatic function (Secretin-pancreozymin test) was not impaired. Endoscopic retrograde cholangio-pancreatography (Fig. 2a and b) detected moderate chronic pancreatitis (7) with dilatation of the main pancreatic duct and rarefications of the side branches, mainly in the head and body of the pancreas. Furthermore, ERP showed several pancreatic duct concrements, between 4 and 6 mm in diameter, in the head of the pancreas, floating upon injection of contrast medium. Endoscopic papillotomy performed to widen the main pancreatic duct permitted subsequent partial extraction and spontaneous passage of the residual pancreatic duct concrements (Fig. 2c), resulting clinically in immediate and complete relief of pain.

Patient 3

A 35-year-old male with an average daily alcohol consumption of about 200 g over a period of 6 years was admitted for repeated severe epigastric pain over the last 9 months prior to hospitalization. Physical examination revealed a poor nutritional state (180/59 kg) and extensive tenderness of the upper abdomen. Laboratory findings showed elevated serum levels of fasting glucose (143 mg%), SGOT (35 U/L; normal 0–18), SGPT (116 U/L; normal 0–22) and alkaline phosphatase (245 U/L; normal 33–105). Total amylase (1640 U/L, normal 230–2700) and leucocyte count 7.900/cmm; normal 4.000–9.400) were in the normal range. A plain film of the upper abdomen revealed excessive and diffuse pancreatic calcification. Pancreatic pseudocysts were excluded by ultrasonography. ERCP indicated advanced chronic pancreatitis (7, 8) and a single pancreatic duct concrement with a diameter of 5 mm in the head of the pancreas, which again floated upon injection of contrast medium. Following endoscopic papillotomy that opened the main pancreatic duct, the ductal concrement passed spontaneously, resulting in immediate and complete relief of pain.

Discussion

Complete and lasting relief of pain is claimed to occur spontaneously in the late course of chronic pancreatitis closely related to severe impairment of pancreatic function (1, 5).

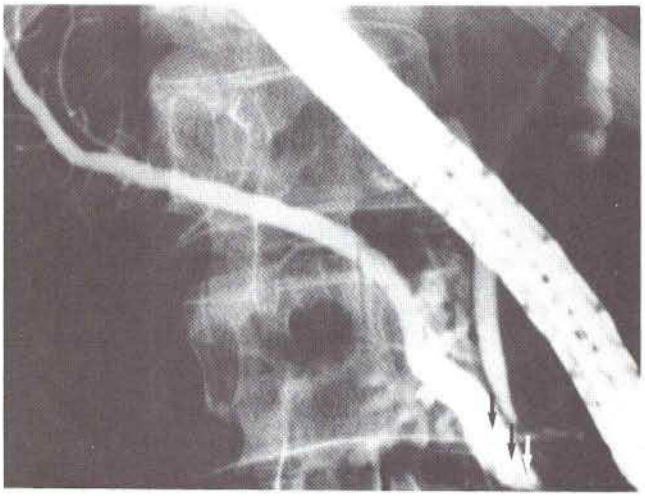

Fig. 2a

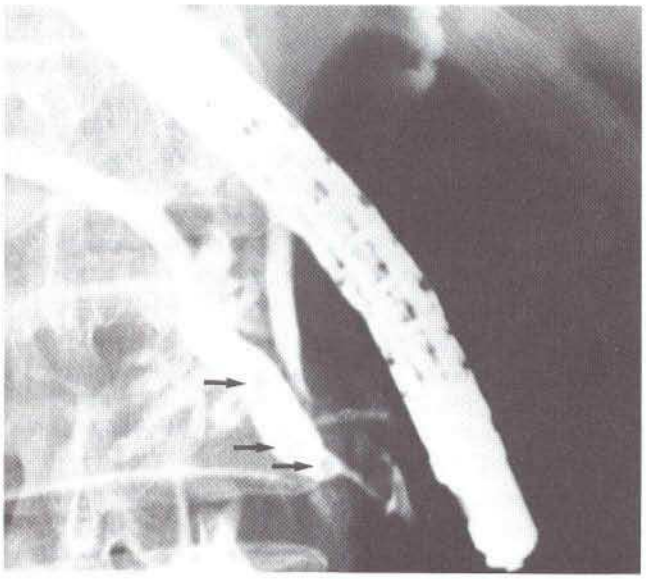

Fig. 2b

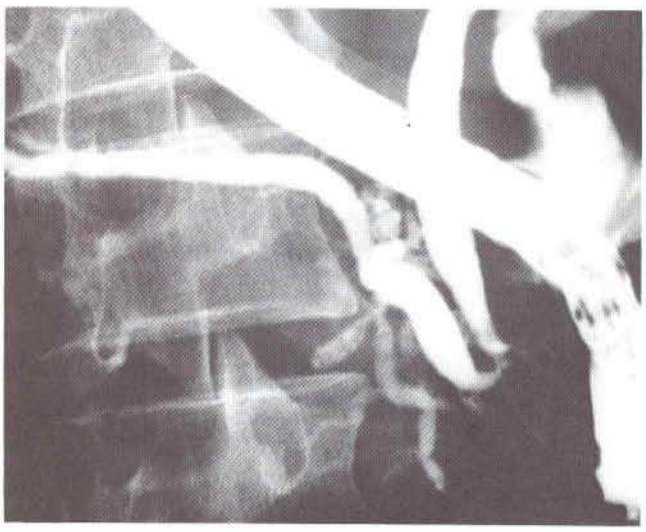

Fig. 2c

Fig. 2 Endoscopic retrograde pancreatogram in patient 2 with moderate chronic pancreatitis and pancreatic duct concrements (→), located in the head of the pancreas (a), floating upon injection of contrast medium (b) and completely removed (c) by partial extraction and spontaneous passage, following endoscopic papillotomy

Nevertheless, some patients with advanced chronic pancreatitis and extremely impaired exocrine function, require surgical therapy for repeated severe attacks of pain. However, immediate and complete relief of pain can obviously be achieved in selected patients with chronic pancreatitis and floating pancreatic duct concrements by endoscopic papillotomy that widens the main pancreatic duct, and subsequent extraction or spontaneous passage of the concrements, as shown in the 3 patients described here. Acute relapses of chronic pancreatitis seem to be due to intermittent pancreatic duct obstruction by pancreatic duct concrements, with a subsequent increase in intraductal pressure. Thus, removal of floating concrements represents causal therapy.

Among the various therapeutic strategies for removing pancreatic duct concrements, pancreatolithotomy (11, 16) is not widely applied, because it has to be combined with permanent pancreatic duct drainage (10), for which the main pancreatic duct has to be larger than 8 mm in diameter. Partial pancreas resection in Whipple's operation, combined with intraoperative Ethibloc® occlusion of the remaining pancreatic duct (4) includes intraoperative removal of pancreatic duct concrements, but the procedure is limited to chronic pancreatitis with severe pain complicated by large pseudocysts, bleeding from the pancreatic duct and/or jaundice. Chemical dissolution of pancreatic duct concrements by prolonged oral citrate administration is theoretically attractive (3, 9). However, in clinical use, it has been rather disappointing, with only 35% of chronic pancreatitis patients responding with a clear decrease, both in number and volume, of pancreatic duct concrements and of pain (14).

Compared with surgery, endoscopic papillotomy with subsequent extraction or spontaneous passage of ductal concrements is less invasive; compared with chemical dissolution it is more successful in removing at least floating concrements and in relieving pain.

In contrast to floating concrements, no therapeutic effect of endoscopic papillotomy may be expected in patients with advanced morphological ductal changes and large concrements, embedded in the pancreatic duct wall. In conclusion, particular attention should be drawn to the detection of floating pancreatic duct concrements during endoscopic retrograde cholangio-pancreatography in patients with chronic relapsing pancreatitis and pain.

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