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Case reports of interest

Right intrahepatic pseudocyst following acute pancreatitis: an unusual location after acute pancreatitis

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Abstract
The location of a pseudocyst (PC) in the liver is an exceptional event, and intrahepatic PCs are mainly located in the left lobe. We report here a case of right intrahepatic PC following acute pancreatitis associated with cystic (aberrant pancreatic) dystrophy of the duodenal wall (CDDW) and chronic pancreatitis. Morphological assessment (ultrasound, computed tomography [CT] scan, and cholangio-magnetic resonance imaging [MRI]) revealed a 10-cm right intrahepatic collection and rupture of the main pancreatic duct. Percutaneous puncture permitted us to detect a high level of amylase in the collection, confirming the diagnosis of intrahepatic PC. Surgical drainage concomitant with pancreatico-duodenectomy for the treatment of CDDW resulted in disappearance of the collection. The mechanism involved in this patient was rupture of the pancreatic duct in the retroperitoneal cavity and erosion reaching the right hepatic parenchyma. Although intrahepatic PCs are rare, the diagnosis of intrahepatic PC complicating acute pancreatitis can be confirmed by a high level of amylase in the collection. Asymptomatic intrahepatic PCs can be treated conservatively, and symptomatic intrahepatic PCs can be managed either transcutaneously or surgically.

Key words Intrahepatic · Pseudocyst · Acute pancreatitis

Introduction

Pseudocysts (PCs) as sequelae to acute pancreatitis or rupture of pancreatic ducts can occur at any site in the abdomen and even distant from the pancreas, such as in the mediastinum, but the intrahepatic location of a pancreatic PC is an exceptional event.[1,2] Fewer than 20 cases have been reported, and most of them are either located in the left lobe and/or are multiple.[1–3] We report here a case of right intrahepatic pseudocyst following acute pancreatitis associated with chronic pancreatitis and cystic dystrophy of the duodenal wall (CDDW). The mechanism was rupture of the pancreatic duct in the retroperitoneal cavity and erosion reaching the right hepatic parenchyma.

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Case report

In a 47-year-old man, CDDW with chronic pancreatitis had been diagnosed 2 years before his current hospitalization, following recurrent attacks of acute pancreatitis.

Two months after a recurrent attack, with inflammation and collection around the head of the pancreas, he was hospitalized because of acute upper right quadrant abdominal pain associated with a high serum amylase level (500 UI/l). Morphologic assessment (ultrasound [US], computed tomography [CT] scan, and cholangio-magnetic resonance imaging [MRI]) showed a right intrahepatic collection (10 cm, in segments 6,7,8), with possible partial cephalic rupture of the main pancreatic duct (Figs. 1 and 2). The alanine aminotransferase and glutamic pyruvic aminotransferase levels were normal (19 and 16 UI/l, respectively).

The collection was punctured, revealing a high level of amylase (3300 UI/l) and confirming the diagnosis of intrahepatic PC. Because the PC was symptomatic and a pancreatico-duodenectomy (PD) was planned for treatment of his CDDW, the patient had preoperative percutaneous drainage, with partial (50%) regression of the PC, and was referred 10 days later for surgery. During the PD, a pericephalic collection in the head of the pancreas secondary to partial rupture of the main pancreatic duct was found, with a communication to the intrahepatic PC via the right suprarenal space. The

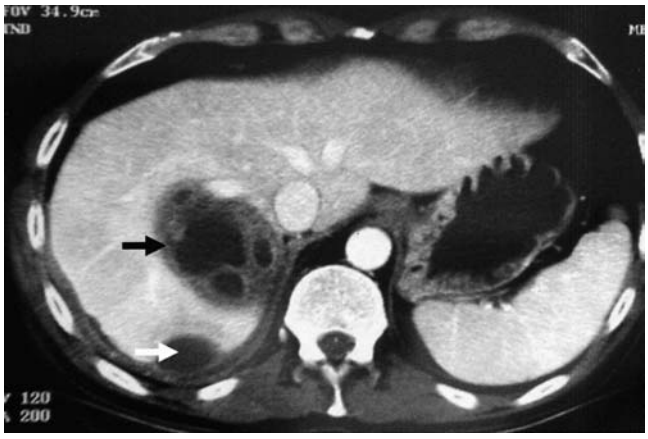

Fig. 1. Computed tomography (CT) scan, showing the right intrahepatic pseudocyst (*black arrow*) and the collection in the right prerenal space (*white arrow*)

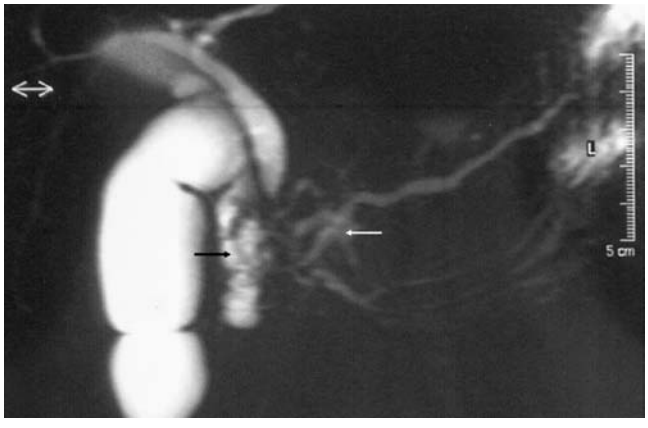

Fig. 2. Cholangio-magnetic resonance imaging (MRI) reconstruction, showing cystic dystrophy of the duodenal wall (CDDW; *black arrow*) and the ruptured main pancreatic duct (*white arrow*)

remaining intrahepatic PC was drained after the PD, under intraoperative US control. The postoperative course was uneventful. Postoperative CT scan revealed that the PC had disappeared on day 7. Pathological findings confirmed the diagnosis of CDDW and the cephalic pancreatic duct rupture.

Discussion

Most pseudocysts (PCs) occur near the pancreas, arising from the action of pancreatic secretions containing proteolytic enzymes, but they can occur at any site within the abdomen, even in the mediastinum.[3] The intrahepatic formation of a PC associated with acute pancreatitis is a very rare event, with fewer than 20 cases reported.[1–12]

The intrahepatic formation of PCs can be explained by two mechanisms[1,4,5] (1) The fluid present in the lesser sac spreads along the hepatogastric ligament into the liver. This mechanism explains why localization in the left lobe is more frequent and can consist only of erosive surface changes or alternatively, of intraparenchymal collection, caused by digestion of the capsule facing the lesser sac. (2) The pancreatic fluid spreads from the head of the gland into the hepatoduodenal ligament and the porta hepatis along the portal vein, leading to left or right intrahepatic PCs.[3] The mechanism of splenic and renal collections is similar: in these locations, pancreatic fluid enters along the major blood vessels into the parenchyma with no direct erosion. In the present patient, the PC was located deep in the parenchyma of segments 6 and 7 and communicated with a collection in the right anterior pararenal space. The mechanism was probably similar to mechanism 1 described above, i.e., leakage of pancreatic fluid into the posterior face of the pancreatic head, reaching the pararenal space and then perforating, by enzymatic action, the parietal peritoneum and finally penetrating the hepatic parenchyma. The preoperative and anatomical-pathological findings confirmed a rupture of the main pancreatic duct that communicated with the intrahepatic formation.

The differential diagnosis of intrahepatic PCs can be challenging.[1,3,5,13] Hepatic abscess or mass can have similar radiological findings. In the presence of signs of acute pancreatitis, the diagnosis of hepatic PC is not difficult by imaging. The pancreatic manifestation can be moderate, and then elevated serum and urinary amylase levels are useful. The serum levels of aminotransferases may not be elevated despite the digestion of liver cells,[3] as occurred in our patient. The content of the intrahepatic PC is homogeneous, with low echoic findings on ultrasound (US) and low density on computed tomography (CT), whereas, in liver abscess, the content is denser and the contours less demarcated.[1,4,5] Finally, amylase levels in the fluid obtained by puncture, as performed in the present patient, are elevated and are the most useful tool for the diagnosis of pancreatic origin. More rarely, intrahepatic PCs can mimic biliary dilatation when the pancreatic fluid spreads into the hepatoduodenal ligament. In these cases, the differential diagnosis comprises biliary obstruction, both malignant and benign. Intracystic hemorrhage, which can occur in 10% of all pancreatic PCs, was reported in two patients with intrahepatic PCs.[14]

No specific treatment is needed in the majority of intrahepatic PCs. Puncture can be used for diagnosis and repeated for treatment.[2,3] Percutaneous drainage is necessary when the collection persists or if it is infected. In the patient we reported here, percutaneous drainage was performed preoperatively, because the patient

was symptomatic, and surgical treatment of CDDW and cephalic chronic pancreatitis was planned. After 10 days of drainage he was asymptomatic and the diameter of the PC was reduced by 50%. A pancreaticoduodenectomy was performed, together with surgical drainage of the associated hepatic PC.

Conclusion

Although they occur rarely, intrahepatic PCs must be suspected when an intrahepatic collection is found in a patient with acute or chronic pancreatitis. The diagnosis is not easy in the absence of evident signs of pancreatitis, and a high level of amylase in the collection can be useful for the diagnosis. Spontaneous regression of the collection can occur with no specific treatment. Symptomatic intrahepatic PCs can be managed percutaneously. Surgery is mainly indicated for the treatment of the underlying pancreatic lesions responsible for the PC.

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