

Pancreatic pseudocyst rupture into the portal vein diagnosed by magnetic resonance imaging

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Case

A 38-year-old man presented to the United Christian Hospital, Hong Kong, with acute epigastric pain in October 2014. He was a chronic drinker and

had experienced intermittent abdominal pain for 6 months. His serum amylase level was elevated (454 IU/L), and a diagnosis of acute-on-chronic pancreatitis was made. The patient was treated conservatively.

Magnetic resonance cholangiopancreatography performed 3 months after hospital discharge showed a 5.8-cm-diameter unilocular cystic mass over the pancreatic head (Fig 1). The main and right portal veins showed a signal intensity identical to that of the cystic pancreatic lesion on all phases, without any contrast enhancement (Fig 2). There was communication between the main portal vein and the cystic mass (Fig 3). The presence of multiple collateral veins in the hepatic hilum was consistent with cavernous transformation (Fig 2c). Features were suggestive of a pancreatic head pseudocyst that had ruptured into the main portal vein.

The patient presented again 1 month later with recurrent pancreatitis. Contrast computed tomography (CT) showed that the pancreatic pseudocyst had enlarged, to 7.6 cm in diameter (Fig 4). Pancreatic cystojejunostomy and cholecystectomy were performed. Intra-operatively, a 10-cm cystic

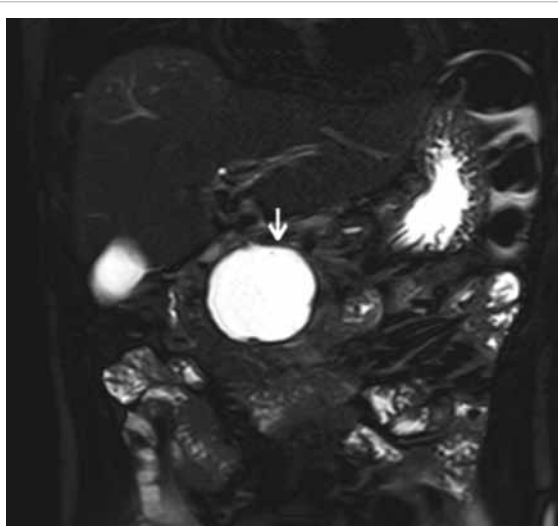


FIG 1. Coronal T2-weighted magnetic resonance image showing a cystic lesion at the pancreatic head (arrow)

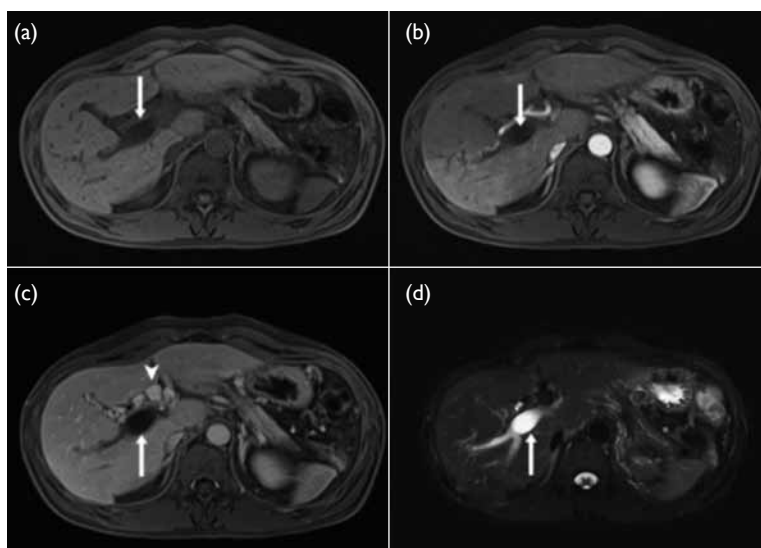


FIG 2. Axial magnetic resonance images: (a) T1-weighted pre-contrast image and (b) T1-weighted post-contrast arterial phase image showing low signal in the main and right portal veins without contrast enhancement (arrows); (c) T1-weighted post-contrast portal venous phase image showing low signal in the main and right portal veins without contrast enhancement (arrow), and with collateral veins at the hepatic hilum (arrowhead) suggesting cavernous transformation; and (d) T2-weighted image showing high signal intensity within the right and main portal veins (arrow)



FIG 3. Two-dimensional slab magnetic resonance cholangiopancreatogram showing a pancreatic head cystic lesion with communication (arrow) with the main portal vein, both as high signal intensity (a normal portal vein with flowing blood should be of low signal intensity); the non-dilated biliary tree (arrowheads) and the gallbladder are also visible



FIG 4. Reformatted coronal computed tomographic image in portal venous phase showing fluid attenuation in the main and right portal veins (PV), absence of contrast enhancement, and communication between the portal veins and the pancreatic cystic lesion (arrow)

lesion at the pancreatic head was found, and 200 mL of clear fluid was aspirated. Intra-operative ultrasonography showed the lack of flow in the main portal vein.

The patient had a few more episodes of recurrent pancreatitis thereafter. The last CT examination, performed 2 years after surgery, showed a reduction in the size of the pseudocyst, to 2 cm. The patient remains on regular follow-up.

Discussion

Rupture of a pancreatic pseudocyst into the portal vein is an uncommon complication; only a handful of cases have been reported in the literature.¹ It has been postulated that portal vein thrombosis occurs first, followed by erosion of the portal vein by pancreatic enzymes present in the pseudocyst, and subsequent lysis of the thrombus and filling of the portal vein with fluid.^{1,2} It has also been reported that rupture of the pseudocyst into the portal vein may be the initial event, followed by the development of portal vein thrombosis.^{3,4}

Previously reported cases have used various diagnostic techniques. Invasive methods include endoscopic retrograde cholangiopancreatography and portography with surgery. Non-invasive methods include ultrasonography, CT, and magnetic resonance imaging (MRI). In all reported cases in

which MRI was performed, the signal intensity of fluid in the portal vein matched that of the pancreatic pseudocyst.¹⁻³ Direct communication between the portal vein and the pancreatic pseudocyst was clearly seen in most cases. The presence of residual thrombus or concomitant existence of complete thrombosis of the portal vein has also been reported.¹

There is no well-established treatment protocol. Options include conservative management, endoscopic or percutaneous procedures, or surgery. The patient's clinical condition and symptoms, patency of the portal vein, communication between the pseudocyst and pancreatic duct, size of pseudocyst, and any other complicating factors should be considered in treatment planning.³

In summary, rupture of a pancreatic pseudocyst into the portal vein is an uncommon complication. On MRI, demonstration of fluid signal in the portal vein that matches the signal intensity of a pancreatic pseudocyst allows the diagnosis to be confidently made, obviating the need for more invasive investigations.

Declaration

The authors have no conflicts of interest to disclose.

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