Treatment of pancreaticoportal fistula by endosonography-guided rendezvous

Pancreatic fistula may occur as a complication of chronic pancreatitis [1 – 3]. However, a communicating fistula between the pancreatic ductal system and the portal vein is a rare and difficult-to-treat complication [4]. We present the first report of endoscopic ultrasound (EUS)-guided main pancreatic duct rendezvous treatment of a symptomatic case of pancreaticoportal fistula. The case was complicated by a partial portal vein thrombosis, related to a pancreatic pseudocyst from chronic pancreatitis. A 69-year-old man presented with abdominal pain and weight loss during the previous 6 months. Laboratory tests revealed anemia (hemoglobin 7.7 g/dL) an increased amylase (7759 U/L) and an albumin of 3.4 g/L. Clinical history and abdominal magnetic resonance imaging (MRI) led to a diagnosis of pancreatic pseudocyst related to underlying alcoholic chronic pancreatitis. The MRI further showed a dilatation of the portal vein related to partial thrombosis (Fig. 1).

We performed an endoscopic retrograde pancreatography (ERP) that revealed a cystic collection in the head of the pancreas with stenosis and dilatation of the main pancreatic duct and secondary ducts. We were unable to pass a guidewire through the pancreatic duct stricture (Fig. 2). However, it was passed through a pancreaticoportal fistula, and the portal vein was visualized immediately after contrast injection. We then attempted an EUS-guided rendezvous maneuver. We accessed the main pancreatic duct through the stomach with a 19G needle, obtained pancreatography, and passed a 0.035-inch guidewire along the main duct through the minor duodenal papilla into the duodenum (Fig. 3). With the duodenoscope it was possible to grasp the guidewire and reposition it in the tail of the pancreas. We finally introduced a fully covered biliary self-expandable metallic stent (8 cm × 10 mm) across the pancreaticoportal fistula.

Fig. 1  A 69-year-old man underwent magnetic resonance imaging (MRI) 5 months after the onset of abdominal pain and weight loss. The MRI images showed portal vein thrombosis associated with increased caliber and parietal post-contrast enhancement: a, b coronal T2-weighted; c magnetic resonance cholangiography (MRC); d, e portal phase, post-gadolinium, fat-suppressed, T1-weighted; f late phase, post-gadolinium, fat-suppressed, T1-weighted. The pancreatic–portal communication, probably related to the pancreatic head pseudocyst is seen in part a (black arrows). The portal vein thrombus (white arrows, a – c) appears hyperintense on the T2-weighted and MRC images, most likely because of liquefaction caused by pancreatic juice. Lack of enhancement of the portal vein caused by thrombosis is shown on post-contrast T1-weighted images (white arrows, d – f), and parietal portal vein enhancement, probably related to inflammation, can be better seen on the late post-contrast phase image (f). The pancreatic head pseudocyst (*) is seen in parts b, c, and e. The dilated main pancreatic duct (arrowhead) is seen better in parts b and c. The normal biliary tree and gallbladder can be seen in part c, partially superimposed by portal vein thrombosis.

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creaticoportal fistula and the main pancreatic duct stenosis (Fig. 4).

Reported treatments for this condition range from conservative medical management to some variation of pancreatectomy [5]. Because of the rarity of the condition, there is a clear need for individualized treatment. In our patient, the insertion of the fully covered self-expandable metallic stent led to marked clinical improvement, reduction of serum amylase (389 U/L), and improvement of the abnormalities visible on MRI; these improvements were found to be persistent at 6-month follow-up.

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References

Fig. 4  a Endoscopic view of the guidewire exiting the minor duodenal papilla. b Fluoroscopy shows insertion of the fully covered self-expanding metal stent (SEMS). c, d Follow-up magnetic resonance imaging (MRI) after 1 month shows the SEMS inside the main pancreatic duct (white arrow), and persistence of portal vein thrombosis but with a marked decrease in the caliber of the portal vein (arrowheads).