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Case report

Transportal embolization for pancreatic arteriovenous malformation via a recanalized paraumbilical vein: A case report ☆,☆☆

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ABSTRACT

A 62-year-old man with liver cirrhosis presented with deterioration of liver function. Imaging studies revealed an arteriovenous malformation (AVM), with a dilated venous space, at the pancreatic head. Transarterial embolization of the AVM, using microcoils, was performed, although many feeding arteries remained. As the transarterial embolization was incomplete, further liver function deterioration was a possibility. In fact, 1 year after the procedure, the patient was referred back to our hospital for treatment of massive ascites and liver function deterioration. Transportal embolization of the dilated venous space was performed, using microcoils via the recanalized paraumbilical vein, with no enhancement of the AVM. No complications occurred. Based on our experience, we propose transportal embolization as an effective treatment option for pancreatic AVM.

Introduction

A pancreatic arteriovenous malformation (AVM) is a rare vascular anomaly that can cause various symptoms, such as gastrointestinal bleeding, pancreatitis, and portal hypertension [1]. While surgical resection is the most common treatment for pancreatic AVM, angiographic intervention is also an effective treatment option, as surgery can be risky, and technically challenging [1] . With regard to angiographic interventions, most previous studies have reported on the effectiveness of transarterial embolization [2-4], with only one case

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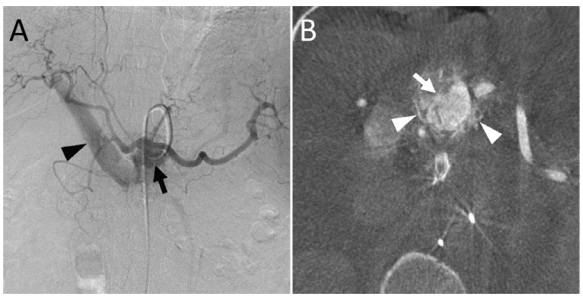

Fig. 1 – (A) Angiography of the celiac trunk, showing mass-like enhancement (arrow) at the pancreatic head, corresponding with the dilated venous space of the AVM. Early enhancement of the portal vein (arrowhead) is noted. (B) Axial cone-beam CT during celiac angiography, showing the dilated venous space (arrow) at the pancreatic head. Around the venous space, multiple small feeding arteries (arrowhead) are observed.

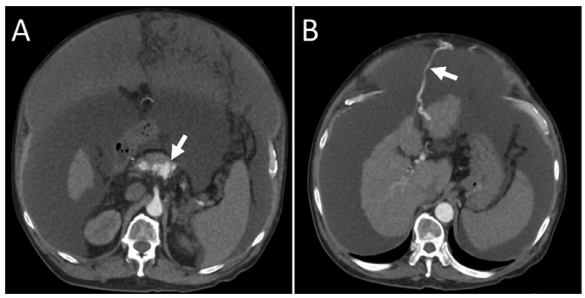

Fig. 2 – (A, B) Arterial-phase contrast-enhanced CT, obtained 1 year after the angiography results shown in Figure 1, demonstrating massive ascites. Enhancement of the dilated venous space (arrow in A) of the pancreatic AVM is shown and a patent paraumbilical vein (arrow in B) is observed.

report on the use of transportal embolization [5]. Herein, we report a case of pancreatic AVM treated using transportal embolization after incomplete transarterial embolization.

Case report

A 62-year-old man with liver cirrhosis caused by hepatitis C and alcoholism, presented with liver function deterioration. He had been treated with interferon-free direct-acting anti–viral therapy and sustained virologic response had been achieved before his presentation. Additionally, he had abstained from alcohol for more than 6 months. Thus, the reason for the deterioration was unclear. His Child-Pugh score was 8 (Child-Pugh class B). Angiography of the celiac trunk revealed a mass-like rapid enhancement at the pancreatic head, followed by early enhancement of the portal vein (Fig. 1A). Cone-beam computed tomography (CT), obtained during celiac angiography, revealed numerous small arteries converging into a dilated venous space draining into the portal vein (Fig. 1B). These findings confirmed the diagnosis of pancreatic AVM, with the mass-like enhancement corresponding to the dilated venous space. Since it was speculated that the pancreatic AVM caused portal hypertension and consequently deterioration of liver function, we decided to perform arterial embolization of the AVM. Several AVM feeding arteries were cannulated with a microcatheter and embolized using microcoils. However, after arterial embolization, many small feeding arteries remained, and additional embolization was considered ineffective; therefore, we ended the procedure. We planned to proceed with transportal embolization of the venous space on a subsequent day; however, the patient needed to be referred to a different hospital due to his employment.

One year after the procedure, he was referred back to our hospital for treatment of massive ascites refractory to conservative therapy. His Child-Pugh score was 10 (Child-Pugh class C), with a prothrombin time of 47%. Contrast-enhanced CT showed a large amount of ascites and enhancement of the dilated venous space of the pancreatic AVM (Fig. 2A). Recanalization of the paraumbilical vein was also observed (Fig. 2B). We decided to perform transportal embolization to improve liver function. Of note, the percutaneous transhepatic approach was not feasible owing to the large amount of ascites and coagulation abnormalities. Therefore, we planned to access the AVM via the paraumbilical vein.

Under local anesthesia and conscious sedation, a 5-F sheath was inserted into the right common femoral artery and a 5-F balloon catheter was advanced. Angiography of the celiac trunk showed rapid enhancement of the dilated venous space of the pancreatic AVM (Fig. 3A). The paraumbilical vein was punctured percutaneously under ultrasound guidance using a 22-gauge coaxial needle. The 0.018-inch wire in a micropuncture kit (Cook Medical, Bloomington, USA) was advanced and the 4-F coaxial dilator in the same kit was placed (Fig. 3B). Through the dilator, a 0.014-inch guidewire, and a microcatheter were advanced into the portal vein. Using a 0.018-inch stiff guidewire, the dilator was exchanged with a 5-F guiding sheath (Parent Plus 45; Medikit, Tokyo, Japan), which was advanced to the left portal vein over a 4-F flexible catheter (Cerulean G; Medikit). A 5-F cobra guiding catheter was advanced from the guiding sheath. From the guiding catheter, the 4-F flexible catheter was advanced coaxially with the microcatheter into the dilated venous space through a draining vein. A dilated vein connected to the dilated venous space was located, which was actually the splenic vein, but misidentified as a part of the AVM and embolized with microcoils. The complex anatomy of the AVM and altered portal venous flow due to the AVM and portal hypertension led to this misidentification. The dilated venous space was then embolized using detachable microcoils. Flow control, via inflation of the catheter balloon at the celiac trunk, was not applied during embolization as the catheters and microcoils were stable. Our procedure is schematically illustrated in Figure 3C. After embolization, angiography of the celiac trunk showed no enhancement of the AVM (Fig. 3D). The mean portal venous pressure before and after embolization was 39 mm Hg and 31 mm Hg, respectively. After removing the guiding sheath placed in the paraumbilical vein, manual compression was applied for 20 minutes, followed by compression with gauze pads for 3 hours. There were no procedure-related complications.

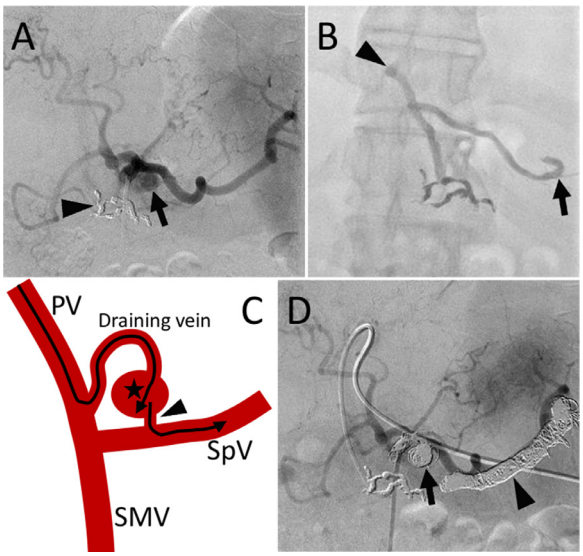

Fig. 3 – Transportal embolization of the pancreatic AVM. (A) Angiography of the celiac trunk, showing enhancement of the dilated venous space (arrow) of the pancreatic AVM. Metallic coils (arrowhead) placed in feeding arteries are visible. (B) Venography performed using a coaxial dilator (arrow), showing the patent paraumbilical vein. The proximal part (arrowhead) of the paraumbilical vein joins the left portal vein, not visualized on this image due to rapid hepatofugal flow. (C) Schematic drawing of the embolization procedure. The curved line with the arrow shows the route of access to the dilated venous space (star) and the splenic vein. The splenic vein was misinterpreted as an abnormal venous component of the AVM and embolized. The connection (arrowhead) between the dilated venous space and the splenic vein was not correctly recognized during the procedure. PV, portal vein; SMV, superior mesenteric vein; SpV, splenic vein. (D) Angiography of the celiac trunk, showing no sign of pancreatic AVM. Multiple coils placed in the dilated venous space (arrow) and splenic vein (arrowhead) are visible.

One month after the procedure, there was no improvement in ascites or liver function. Considering that the portal venous pressure was high after successful transportal embolization, it was assumed that the patient had already progressed to advanced liver cirrhosis due to the pancreatic AVM at the time of the transportal embolizaton and that the clinical effect of treating the AVM was therefore limited.

Discussion

Based on the 2006 AVM classification [6], AVM of the trunk and extremities are classified into 4 types: type I, II, IIIa, and IIIb. Type II AVM are also called AVM with a dominant outflow vein. The transarterial approach for type II AVM is usually difficult and, therefore, transvenous embolization is advocated [7,8]. In our case, the pancreatic AVM was classified as type II; consequently, we decided to perform transportal embolization after incomplete transarterial embolization. We suggest that pancreatic AVM should be classified before treatment, and that transportal embolization should be considered as a treatment option for type II AVM. The usefulness and safety of paraumbilical vein access have previously been reported [9,10]. Paraumbilical vein access is a reasonable approach to the portal vein in patients with a large amount of ascites or coagulation abnormalities.

In conclusion, we suggest transportal embolization as an effective treatment option for a pancreatic AVM, although the individual AVM vascular anatomy should be carefully evaluated before treatment.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee, and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study, formal consent is not required.

Patient Consent

Written and informed consent for publication was obtained from the patient.

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