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Hepatic Encephalopathy Secondary to Intrahepatic Portosystemic Venous Shunt: Balloon-Occluded Retrograde Transvenous Embolization with n-Butyl Cyanoacrylate and Microcoils

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Abstract

We report a 70-year-old woman with hepatic encephalopathy due to an intrahepatic portosystemic venous shunt that was successfully occluded by percutaneous transcatheter embolization with n-butyl cyanoacrylate and microcoils.

Key words: Hepatic veins—Interventional procedures—Portal vein, therapeutic blockade—Shunts, portosystemic

Intrahepatic portosystemic venous shunt (IPSVS) is not common and requires closure when associated with hepatic encephalopathy [1]. We present such a case and its treatment by balloon occluded retrograde transvenous embolization with n-butyl cyanoacrylate (NBCA) and microcoils.

Case Report

A 70-year-old woman was referred to this department with a history of repeated episodes of unconsciousness. She had no history of trauma, liver biopsy, or liver disease. A review of the entire family tree revealed no inherited disorder. Laboratory studies showed no liver dysfunction with the exception of a mild increase in the serum ammonia level (90 μg/dl; normal range $<$79 μg/dl). Conventional enhanced CT showed an abnormal vessel in communication with a peripheral left portal branch at which point an aneurysm-like structure existed. From this, an anomaly of the portal venous system was suspected. Enhanced magnetic resonance imaging (MRI) performed subsequently led to the suspicion of an IPSVS between one of the left portal branches and the left hepatic vein (Fig. 1). Superior mesenteric arterial portography and celiac arteriography in the portal phase confirmed the presence of an IPSVS (Fig. 2). A small portal aneurysm existed between the IPSVS and the peripheral ventro-lateral left portal branch. ^{99m}Tc-pertechnetate per-rectal portal scintigraphy calculated the shunt ratio to be 14.8% (normal range $<$10% according to criteria used in our institution). Considering the absence of other pathology, these findings confirmed that the etiology of the repeated episodes of unconsciousness was hepatic encephalopathy caused by an IPSVS, which was considered to be congenital

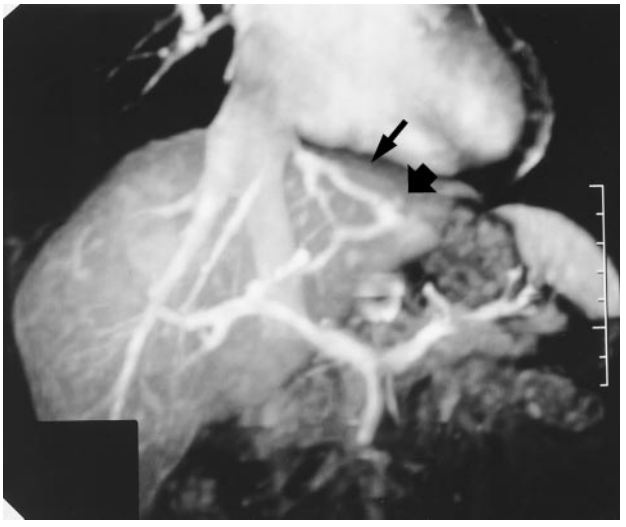

Fig. 1. MR angiography with contrast medium reveals one anomalous vessel (arrow) existing between the branch of the left portal vein and the left hepatic vein. Note that this vessel is connected with the portal venous branch via a small aneurysmal structure (arrowhead).

because there had been no episode to precipitate the anomalous shunt. Therefore, embolization of the IPSVS was attempted. Informed consent was obtained from the patient.

The right femoral vein was punctured, and a sheath (Terumo, Tokyo, Japan) to accommodate a 5-Fr catheter was inserted. A 5-Fr hook-shaped balloon catheter with a balloon 9 mm in diameter (Clinical Supply, Gifu, Japan) was introduced through the sheath into the left hepatic vein, followed by coaxial passage by a 0.018-inch infusion microcatheter (Renegade; Boston Scientific, Watertown, MA, USA) into the small portal aneurysm via the IPSVS. The branches of the portal vein were revealed by angiography from the microcatheter with the balloon inflated (Fig. 3). Six microcoils (Trufill; Cordis, Miami, FL, USA) were inserted from inside the small portal aneurysm to the IPSVS. However, the fact that slow blood flow through the IPSVS remained was determined by test injection of contrast medium via the microcatheter advanced over the proximal microcoil into the anomalous

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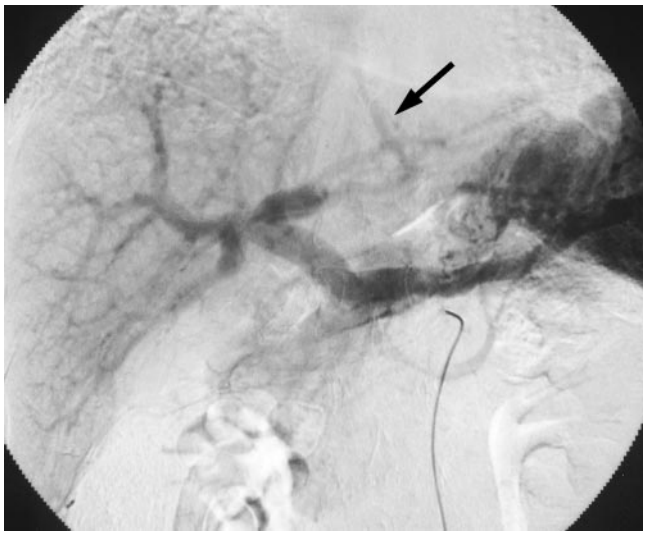

Fig. 2. The celiac arteriography in the portal phase shows one connection between one of the left portal branches and the left hepatic vein (arrow).

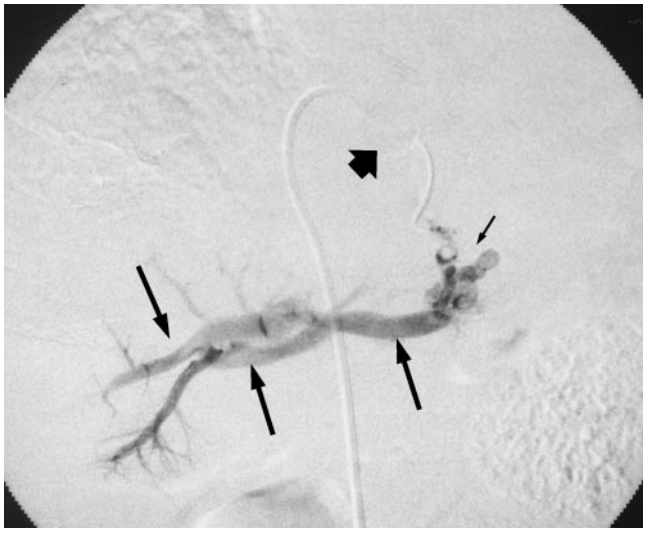

Fig. 3. Retrograde injection of the contrast medium via the microcatheter advanced into the portal aneurysm (small arrow) after balloon *(arrowhead) occlusion of the origin of the left hepatic vein shows branches of the left portal vein (large arrows).

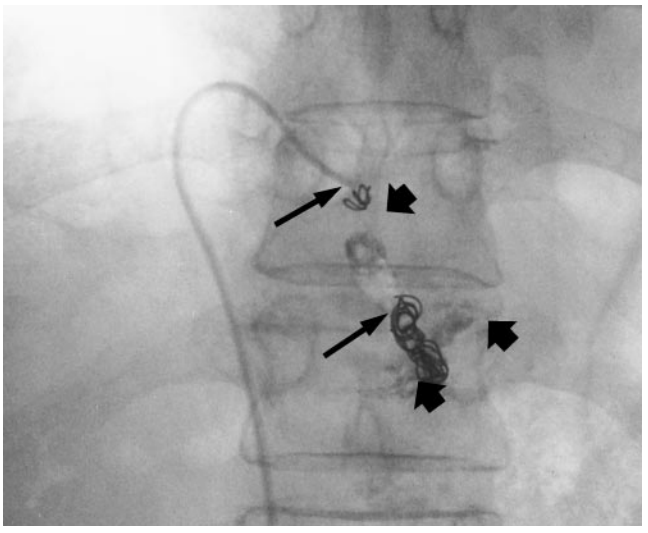

Fig. 4. Plain X-ray film after all embolization procedures shows that the portal aneurysm and the IPSVS are completely occupied by the radiopaque NBCA-Lipiodol mixture (arrowheads) and microcoils (arrows).

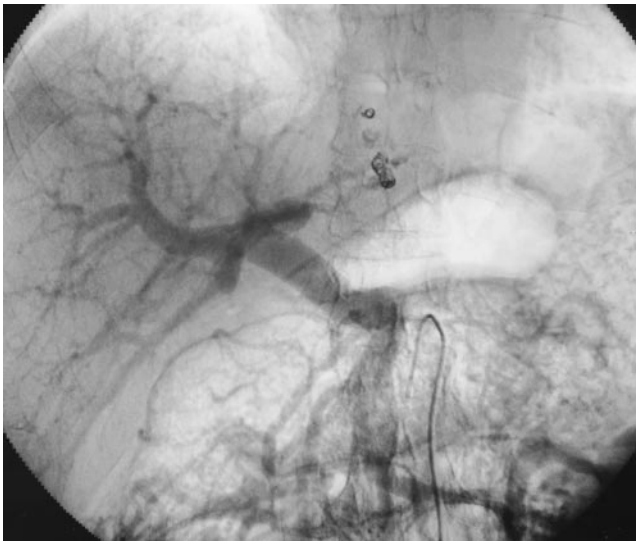

Fig. 5. Superior mesenteric arteriography in the portal phase after the procedure showed no portal aneurysm or IPSVS.

shunt vessel after the balloon was slightly deflated. Thus, approximately 1.0 ml of NBCA (Histoacryl-Blue; Braun, Melsungen, Germany) mixed with Lipiodol (Laboratoire Guerbet, Roissy, France) at a 1:1 ratio was additionally infused to completely embolize the anomalous shunt vessel after re-inflation of the balloon (Fig. 4). Then the balloon was slowly deflated under careful observation to be sure that the NBCA-Lipiodol cast did not move or become attached to the balloon. Superior mesenteric arterial portography after the procedure showed that the portal aneurysm and the IPSVS were completely occluded (Fig. 5). There were no procedural complications, and liver function was consistently normal after embolization. Laboratory data from the day following the procedure showed that the serum ammonia level had normalized to 18 μg/dl. Two weeks after embolization, the shunt ratio calculated by ^{99m}Tc-pertechnetate per-rectal portal scintigraphy had decreased to 6.2%.

Currently, 5 months after this interventional procedure, there are no symptoms of hepatic encephalopathy and the clinical condition of the patient is good.

Discussion

From an extensive review of the literature on IPSVS, Park et al. [1] categorized IPSVSs into four different morphologic types: 1) a single large tube of constant diameter that connects the right portal vein to the inferior vena cava (the most common type); 2) a localized peripheral shunt in which single or multiple communications are found between peripheral branches of portal and hepatic veins in one hepatic segment; 3) a connection between peripheral portal and hepatic veins through an aneurysm; and 4) multiple

communications between peripheral portal and hepatic veins diffusely in both lobes. The present case shows features of a mixture of Park's types 2 and 3.

The cause of IPSVS has been controversial except for cases occurring after liver biopsy or injury. Some believe that it is congenital [2–5], and others have postulated that it is acquired secondary to portal hypertension due to liver cirrhosis [6, 7]. Because there was no history of liver biopsy, injury, or chronic liver damage in the present case, we considered the cause to be congenital.

IPSVS had been thought to be uncommon [1], but recent advances in imaging modalities such as ultrasound (US), computed tomography (CT), and MRI have revealed even asymptomatic intrahepatic shunts in an increasing number of patients [6–8]. In patients with symptoms of hepatic encephalopathy after increased blood flow through the shunt, treatment is required [1]. Traditionally, surgical removal of the shunt was used to alleviate symptoms [2, 4]. In recent years, however, less invasive treatments using interventional techniques such as transcatheter embolization have been widely accepted [3, 9–14] in parallel with the rapid development of various interventional instruments such as microcatheters, imaging modalities, and embolic agents. For multiple IPSVSs, the mesenteric venous route through a small abdominal incision has often been selected [9–11], whereas for single or a few IPSVSs the less invasive percutaneously trans-hepatic [12] or trans-caval approach [3, 13, 14] has been chosen. In the present case, we chose the trans-caval route because it seemed to be the most easily performed. Regarding the embolic agents, stainless steel coils or microcoils have been widely used [3, 9–14]. In general, however, embolization with coils alone necessitates many coils [9–11, 13, 14], making the procedure expensive and time-consuming.

Considering the shortcoming of coils alone as embolic agents for IPSVS, we additionally used NBCA mixed with Lipiodol. NBCA is a liquid acrylic surgical adhesive material that is also widely used as a permanent embolic agent, especially in the field of intracerebral interventional radiology such as embolization of arteriovenous malformation [15, 16]. By adding Lipiodol to the NBCA, the embolized vessel can be visualized. Also, the adhesion time can be regulated according to the ratio of the mixture of Lipiodol and NBCA. Thus, migration of embolized agents or hepatic infarction, which are potential complications, may be avoided as was the case in our patient.

In conclusion, the management of hepatic encephalopathy due to IPSVS by embolization with the combination of microcoils and NBCA should be considered as an effective method for treatment of this condition. To our knowledge, there have been no reports of an IPSVS successfully embolized with NBCA. In addition, per-rectal portal scintigraphy performed before and after therapy was useful for evaluating therapeutic effectiveness, although the shunt ratio before therapy was relatively lower than in some previous reports of IPSVS with hepatic encephalopathy in which ratios ranged from 24.3% to 77% [17, 18].

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