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CASE REPORT

Successful portal-systemic shunt occlusion of a direct shunt between the inferior mesenteric vein and inferior vena cava with balloon-occluded retrograde transvenous obliteration following recanalization after placing a covered stent in the portal and superior mesenteric veins

Sadao Hayashi · Yasutaka Baba · Terutoshi Senokuchi
Kazuto Ueno · Masayuki Nakajo

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Abstract Extrahepatic portal-systemic shunts cause portal-systemic encephalopathy. Direct communication between the inferior mesenteric vein (IMV) and the inferior vena cava (IVC) is a relatively rare pathway among the variety of portal-systemic shunts. This report describes a case of successful occlusion of an IMV–IVC shunt. Based on laboratory data and computed tomography findings, a 69-year-old woman with liver cirrhosis was diagnosed with portal-systemic encephalopathy due to a shunt between the IMV and the IVC. Her hepatic coma had not been adequately controlled by oral or intravenous pharmacotherapy. First, we placed a covered stent in the main trunk of the portal vein and the superior mesenteric vein (SMV) to block the SMV hepatofugal flow and splenic vein hepatopetal flow, but this therapy showed only a transient therapeutic effect due to recanalization. Next, we performed balloon-occluded retrograde transvenous obliteration (BRTO) of the portal-systemic shunt. After the BRTO, she has had no episodes of portal-systemic encephalopathy for 2 years.

Key words BRTO · Covered stent · Hepatic encephalopathy · Portal-systemic shunt

Introduction

Portal-systemic venous shunts are usually formed because of portal hypertension due to liver cirrhosis, and they lead to hepatic encephalopathy.[1–3] Chronic hepatic encephalopathy, which is sometimes refractory to pharmacotherapy, impairs the patient's quality of life.[2] Obliteration of portal-systemic shunts by surgical ligation, transhepatic or transvenous embolization, or balloon-occluded retrograde transvenous obliteration (BRTO) is effective for intractable portal-systemic encephalopathy.[4–6]

Direct communication between the inferior mesenteric vein (IMV) and the inferior vena cava (IVC) is relatively rare.[7–10] We were asked to treat a patient with a direct communication between the IMV and the IVC that had caused refractory hepatic encephalopathy. First, we inserted a covered stent in the main trunk of the portal vein and the superior mesenteric vein (SMV) to block the hepatofugal flow from the SMV and the hepatopetal flow from the splenic vein. This therapy resulted in only a transient therapeutic effect, as recanalization occurred, once again producing hyperammonemia. Next, we performed BRTO for a large shunt between the IMV and the IVC. This therapy alleviated her hepatic encephalopathy for a longer period.

The institutional review board at our hospital does not require approval for a retrospective case report.

Case report

A 69-year-old woman consulted a doctor at a nearby hospital complaining of multiple episodes of unconsciousness during the last year. She had been treated at the same hospital for diabetes mellitus (with insulin) as well as liver cirrhosis due to hepatitis C virus for the past 13 years. Her medical doctor discovered that she had high serum ammonia levels, and computed tomography

S. Hayashi (✉) · Y. Baba · T. Senokuchi · K. Ueno · M. Nakajo
Department of Radiology, Kagoshima University Graduate School of Medical and Dental Sciences, 8-35-1 Sakuragaoka, Kagoshima 890-8520, Japan
Tel. +81-99-275-5417; Fax +81-99-265-1106
e-mail: hayashi@m.kufm.kagoshima-u.ac.jp

(CT) revealed a large portal-systemic shunt between the IMV and the IVC.

She was treated by an intravenous drip infusion of branched-chain amino acid solution (Aminoleban; Otsuka Pharmaceutical, Tokyo, Japan) 500 ml/day, and her conscious state improved. She was then given oral branched-chain amino-acid supplement and lactitol hydrate (Portolac; Nippon Shinyaku, Kyoto, Japan). However, her encephalopathy was refractory to the oral and intravenous pharmacotherapy. Therefore, she was referred to our department for further evaluation and treatment of her encephalopathy. She was admitted to our department on January 4, 2006.

On admission, she had no signs of anemia, icterus, cutaneous stigmata of chronic liver disease, ascites or peripheral edema. Her past history included pylorogastrectomy for gastric cancer 6 years ago. She had no habit of drinking alcohol.

The laboratory data were normal except for pancytopenia: leukocyte count 3000 μ/l, normal 4500–8500 μ/l; erythrocyte count (RBC) 344 $\times$ 10^4/μl, normal 380–480 $\times$ 10^4/μl; hemoglobin 9.0 g/dl, normal 12–16 g/dl; hematocrit 29.1%, normal 35.0–48.0%; platelet count 9.9 $\times$ 10^4 /μl, normal 13–32 $\times$ 10^4/μ. She also had high serum ammonia (180 μg/dl, normal <66 μg/dl); low serum albumin (3.4 g/dl, normal 4.1–5.5 g/dl); and a low cholinesterase (150 IU/l, normal 176–388 IU/l). The indocyanine green excretion test showed 36% retention at 15 min (normal <15%). The Child-Pugh score was 9, and Child-Pugh classification was class B.

The endoscopic examination revealed that there was a single, small, straight esophageal varix (F1) and no evidence of the red color sign (RC–).[11] The maximum intensity projection (MIP) image constructed by contrast-enhanced 16-detector CT (Aquilion 16; Toshiba, Tokyo, Japan) scans showed a markedly tortuous mesenteric varix in front of the left kidney. The varicose IMV was dilated and drained directly into the left side of the caudal IVC (Fig. 1). The diameter of the intrahepatic portal vein was smaller than that of the IMV.

Abdominal arteriography was performed to evaluate portal venous flow. The portal venous phase of the celiac arteriography revealed that most of the splenic venous flow drained into the IVC via the dilated IMV. The portal venous phase of the superior mesenteric artery arteriography revealed that most of the superior mesenteric vein (SMV) flow drained into the IVC via the splenic vein and the dilated IMV. Based on these findings, this shunt was deemed the main cause of the portal-systemic encephalopathy.

During hospitalization, she had an episode of encephalopathy. Considering the risk of further recurrent attacks of coma due to portal-systemic encephalopathy, we recommended an endovascular interventional procedure for the shunt therapy to the patient and obtained written informed consent from her. We planed to occlude the orifice of the splenic vein into the portal vein, which was a shunt pathway from the SMV to the dilated IMV by placing a covered stent in the main trunk of the portal vein and the SMV to change SMV hepatofugal flow to hepatopetal flow and block the hepatopetal flow of the splenic vein. This stenting was performed on February 1, 2006.

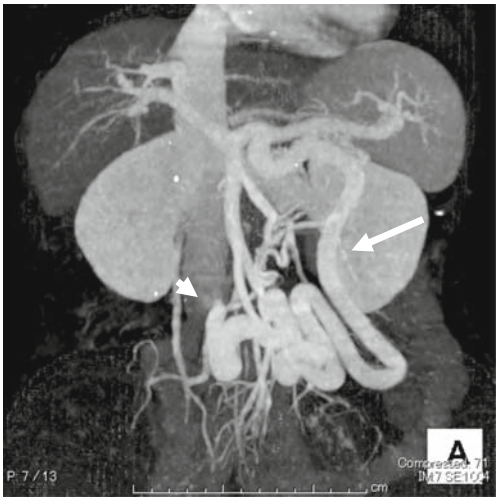

Fig. 1. Maximum intensity projection (MIP) computed tomography (CT) portography. The diameter of the inferior mesenteric vein (IMV) shunt (*arrow*) is larger than those of the intrahepatic portal branches and the superior mesenteric vein (SMV). The efferent orifice of the tortuous IMV to the inferior vena cava (IVC) is clearly visualized (*arrowhead*)

The portal vein was accessed through the ileocolic vein under laparotomy and a 9F introducer sheath was placed. We placed the ϕ 10 $\times$ 40 mm polytetrafluoroethylene (PTFE)-covered stent (Passager TM biliary stent endoprosthesis; Boston Scientific, Natick, MA, USA) to cover the orifice of the splenic vein, thereby blocking hepatofugal flow from the SMV to the IMV shunt. After this procedure, a high serum ammonia level was decreased. Three months later, however, the ammonia level was again increased. She was readmitted to our department to determine the cause of the hyperammonemia on May 22, 2006.

The CT scans revealed that the portal vein was dilated around the covered stent, and venous flow was noted around the stent. Angiography revealed recanalization, with hepatofugal flow from the SMV to the IMV shunt through the splenic vein. We explained the necessity and risk of complete occlusion of the IMV shunt by BRTO to prevent her from having encephalopathy and again obtained written informed consent from her. The BRTO was performed on June 28, 2006.

A 6F balloon catheter (Cobra type; balloon diameter 20 mm; Clinical Supply, Gifu, Japan) was inserted into the cephalad IMV portion near the splenic vein through the 6F sheath via the right femoral vein. A 2.3F microcatheter (Renegade; Boston Scientific, Cork, Ireland) was advanced coaxially through the balloon catheter into the cephalad orifice of the IMV at the splenic vein side. Five microcoils (interlocking detachable coil, or IDC; Boston Scientific, Ireland) were placed in the cephalad portion of the IMV to prevent migration of the sclerosant into the main trunk of the portal vein. After inflating the balloon in the caudal IMV portion near the orifice of the IVC, 10% ethanolamine oleate (Oldamin; Takeda Pharmaceutical, Osaka, Japan) and the contrast medium iopamidol (Iopamiron 300; Schering, Osaka, Japan) were equally mixed (volume/volume) to make the sclerosant. A total of 40 ml of the sclerosant was injected through the catheter to fill the IMV shunt (Fig. 2). The systemic infusion of 4000 units of haptoglobin was started just before BRTO to avoid hemoglobinuria induced by ethanolamine oleate. The patient was returned to her ward with the inflated balloon catheter in place overnight.

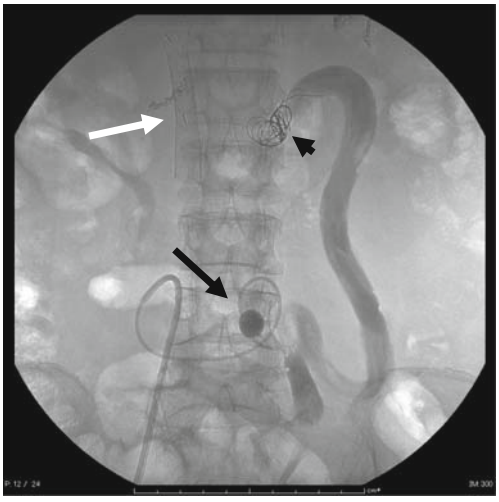

Fig. 2. Radiograph shows the opacified sclerosant in the dilated IMV shunt between the inflated 6F balloon catheter (*black arrow*) at the efferent portion of the shunt and the microcoils (*arrowhead*) at the afferent orifice of the tortuous IMV to prevent sclerosant migration. The covered stent is clearly visualized (*white arrow*) in the portal vein and the SMV to cover the orifice of the splenic vein

The next morning we retrieved as much of the floating sclerosant and blood clot as possible from the IMV shunt with the balloon inflated. The entire catheter system was then withdrawn after the balloon was deflated. The blood ammonia level decreased to the normal range on the day after BRTO and continued in the normal range for 2 years. Furthermore, the Child-Pugh score decreased from 9 to 7 points. The subsequent contrast-enhanced CT scans a week after the BRTO revealed that the shunt was completely thrombosed, and the diameter of the portal vein was enlarged from 13 mm to 16 mm (Fig. 3). The patient had no evidence of complications after the procedure.

Contrast-enhanced CT performed 2 years after obliterating the portal-systemic shunt revealed that the shunt had completely disappeared, and there was no evidence of other portal-systemic shunts. The blood ammonia level has remained in the normal range without hepatic encephalopathy after the BRTO.

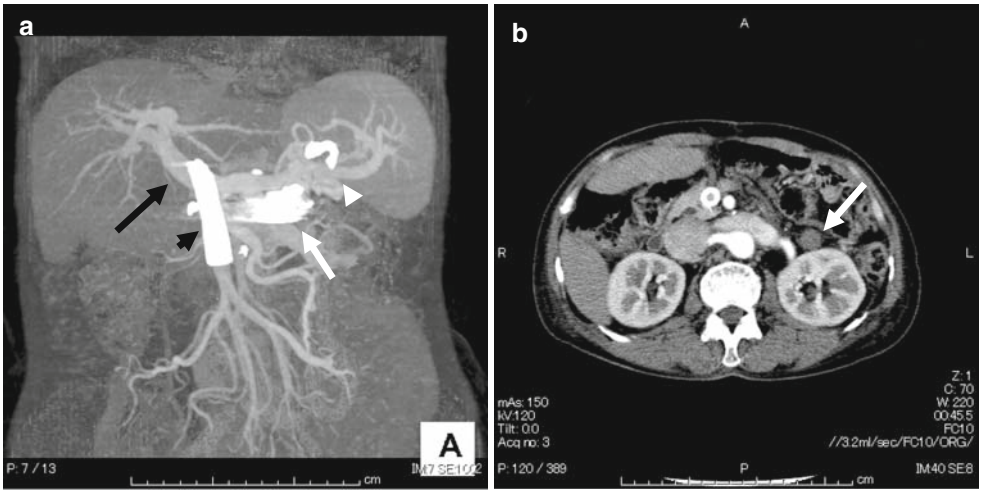

Fig. 3. a MIP CT portography a week after balloon-occluded retrograde transvenous obliteration (BRTO). The tortuous IMV has disappeared, and the diameter of the portal vein is larger than before BRTO (*black arrow*). The covered stent is clearly visualized (*black arrowhead*) in the portal vein and the SMV to cover the orifice of the splenic vein. Microcoils at the efferent orifice of the IMV made metallic artifact (*white arrowhead*). *White arrow* indicates staples of pylorogastrectomy. **b** Contrast-enhanced CT a week after the BRTO revealed that the tortuous IMV in front of the left kidney was not enhanced owing to thrombus formation (*arrow*)

Discussion

Extrahepatic portal-systemic shunts frequently develop in patients with portal hypertension. Multidetector CT provided the precise location and morphology of the IMV varicose shunt in our particular patient. Surgical ligation and an endovascular interventional procedure such as transhepatic or transvenous embolization or obliteration of the shunt have been the primary treatment choices for extrahepatic portal-systemic shunts. Nowadays, less invasive endovascular procedures have been advocated as alternatives to surgical ligation.[5–9,12–16]

In Japan, BRTO is widely accepted for obliterating gastric varices with the transvenous approach.[4,17] This method is also used for obliterating portal-systemic shunts.[7,8] There have been many reports about obstructing the portal-systemic shunt wherein the hyperammonemia and clinical symptoms were alleviated in patients with hepatic encephalopathy.[7–9,13,15,16]

In our patient, liver function was preserved before the procedure. However, we were apprehensive about the possible development of ascites and worsening of esophageal varices by a sudden increase in portal pressure after complete obliteration of the dilated IMV shunt because the IMV shunt vessel was large and received most of the SMV and splenic flow.[4–6] We initially considered performing selective coil embolization of the splenic vein near the portal vein, as reported by Mezawa et al.,[12] which would block hepatofugal SMV flow and lead the splenic flow alone into the IMV shunt. However, we were apprehensive about coil migration to the systemic circulation via the IMV or to the portal vein because the splenic vein was also large; moreover, the embolization site of the splenic vein between the portal vein and the cephalic orifice of the IMV shunt was too short (8 mm) to place coils between them. Therefore, we decided to block splenic hepatopetal flow by placing the covered stent in the portal vein. We thought that this procedure would allow most of the SMV blood to drain into the portal vein to decrease the serum ammonia level, and elevation of the portal venous pressure might be more gradual with this procedure than with total occlusion of the dilated IMV shunt. In fact, it did reduce the hyperammonemia. However, the portal vein around the covered stent dilated gradually, and the blood of the SMV was again draining into the IMV shunt 3 months later. We next chose total shunt occlusion with BRTO to block the recanalized SMV hepatofugal flow.

We used coils and a sclerosant (ethanolamine oleate) to occlude the shunt. The use of coils alone carried a risk of coil migration because of the very large shunt, which would require many coils. The cost of the occlusion coils was much higher than that of sclerosant. Therefore, we infused the sclerosant under occlusion of the caudal orifice of the IMV shunt near the IVC with the balloon inflated after occlusion of the cephalad orifice of the shunt near the splenic vein by five coils. Ethanolamine oleate takes a few hours to make a thrombus in the vein.[4] Thus, we kept the balloon catheter inflated overnight to prevent the sclerosant from migrating to the IVC and producing a pulmonary embolism. Although we infused 40 ml of the sclerosant into the dilated IVM shunt to occlude it completely, a lesser amount of the sclerosant might have been sufficient for this purpose as 20 ml and 15 ml of the same sclerosant were infused into the same types of IMV-to-IVC shunt by others.[7,8]

Chronic or recurrent portal-systemic encephalopathy refractory to conventional treatment is generally considered an indication for shunt occlusion, although complications such as the development of ascites and worsening of esophageal varices occurs in some patients.[4–6] The threshold for increased portal pressure after BRTO in regard to increasing the risk of complications is unknown.[17] Some authors recommended separation therapy instead of complete occlusion of the shunt to avoid abrupt increases in portal venous pressure.[12,14]

In the present case, we could not directly measure the portal venous pressure because we did not choose the percutaneous portal vein approach owing to the narrow intrahepatic portal vein. Therefore, we could not anticipate the change in portal venous pressure before and after occlusion of the shunt. Thus, we first tried separation therapy of the splenic vein by placing the covered stent into the main trunk of the portal vein and the SMV to block SMV hepatofugal flow and splenic vein hepatopetal flow. This was considered much safer than the direct shunt occlusion.[14] Although this therapy brought a temporally good result, the portal vein dilated around the covered stent and the SMV flow drained again into the IMV shunt through the gap around the stent. We subsequently achieved a better result with BRTO for the portal-systemic shunt. We have not observed development of ascites and worsening of esophageal varices in our patient after the therapy. This might be due to her good liver function.[4] However, definitive criteria remain to be established regarding BRTO for such a portal-systemic shunt as was observed in our patient, although portal venous pressure measurement before and after temporal balloon occlusion of the shunt is recommended to test the tolerance to subsequent shunt occlusion.[17]

There has been no evidence of recanalization of the shunt vessel 2 years after this treatment. However, long-term observation is necessary to evaluate the outcome of this shunt occlusion procedure.

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