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

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 SMV hepatofugal flow and splenic vein hepatopetal flow, but 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...
(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 × 10^4/μl, normal 380–480 × 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 × 10^3/μl, normal 13–32 × 10^3/μl. 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−). 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 planned 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.

The portal vein was accessed through the ileocolic vein under laparotomy and a 9F introducer sheath was placed. We placed the φ 10 × 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.