Selective embolization of the splenic vein for shunt-preserving disconnection of the portal and systemic circulation: report of two cases

Osamu Ikeda1, Yutaka Nakasone1, Toru Beppu2, Toshiro Masuda2, Hideo Baba2 and Yasuyuki Yamashita1

1Department of Diagnostic Radiology; 2Department of Gastroenterological Surgery, Kumamoto University Graduate School of Medical and Pharmaceutical Sciences, Kumamoto, Japan

Correspondence to: Osamu Ikeda. Email: osamu-3643ik@do9.enjoy.ne.jp

Abstract

In carefully selected patients with portosystemic encephalopathy, it is possible to safely disconnect the portal and systemic circulation while preserving the shunt. We report two patients with chronic hepatitis and recurrent episodes of hepatic encephalopathy due to a portosystemic shunt who underwent successful selective embolization of the splenic vein for a shunt-preserving disconnection of the portal and systemic circulation via the percutaneous transhepatic route.

Keywords: Portosystemic encephalopathy, shunt-preserving disconnection of portal and systemic circulation, percutaneous transhepatic access

Submitted February 11, 2012; accepted for publication August 7, 2012

Case reports

The Human Subjects Research Review Board at our institution approved our interventional protocol and our retrospective study. Patient consent for inclusion in this retrospective study was waived.

Case 1

This 50-year-old man with chronic hepatitis due to hepatitis C virus infection suffered from recurrent episodes of hepatic encephalopathy since undergoing transcatheter arterial chemoembolization for hepatocellular carcinoma 1 year earlier. He had repeated episodes of acute encephalopathy despite protein restriction and administration of lactulose and branched-chain amino acids and was admitted with hepatic encephalopathy (West Haven Criteria stage 4). His liver function was Child-Pugh B (total bilirubin, 1.9 mg/dl; albumin, 2.9 g/dl; prothrombin time, 94%; no ascites). The blood ammonia level was 333 μg/dl (normal range, 15–65 μg/dl). Contrast-enhanced computed tomography (CT) scans were acquired with a 64-row multidetector scanner (Brilliance-64, Philips Medical Systems, Best, The Netherlands) and 3D images revealed portosystemic shunts.

Case 2

This 70-year-old woman with chronic hepatitis due to hepatitis C virus infection had had recurrent episodes of hepatic encephalopathy despite protein restriction and the administration of lactulose and branched-chain amino acids.
She was admitted with hepatic encephalopathy (West Haven Criteria stage 4). The liver function was Child-Pugh A (total bilirubin, 0.7 mg/dl; albumin, 3.2 g/dl; prothrombin time, 96%; no ascites) and the blood ammonia level 214 μg/dl. Contrast-enhanced CT scans were acquired with a 64-row multidetector scanner (Brilliance-64); 3D images revealed portosystemic shunts and a portal-hepatic vein shunt in the left lobe.

Three-phase contrast-enhanced CT scans of the liver were acquired in the arterial, portal venous, and equilibrium phases in both patients. An automatic bolus-tracking program was used to time the start of scanning for each phase after contrast injection. Hepatic arterial, portal venous, and equilibrium phase scanning was started 18, 55, and 180 s after triggering. We used a bolus injection of 100 mL iopromide (Iopamiron 300; Nihon Beyer, Osaka, Japan) delivered at a rate of 3 mL/s. All images were obtained through the abdomen in a craniocaudal direction.

First, under local anesthesia, superior mesenteric and celiac arteriography was performed to develop a treatment plan and to assess collateral circulations in both patients. DSA of the superior mesenteric artery and the celiac trunk was performed after the administration of 2.5 μg prostaglandin E1 (Liple; Mitsubishi Pharma Corp., Osaka, Japan), using a 4-Fr RC2 catheter (Medikit Co. Ltd., Tokyo, Japan) after injection of iomeprol (iopamiron 300; Bayer, Osaka Japan) at a flow rate of 6 mL/s and 5 mL/s, respectively. In both cases, the venous phase of superior mesenteric arteriography demonstrated a spleno-renal and a spleno-retroperitoneal shunt, and an undetected portal vein trunk (Fig. 1). There was enough distance to allow disconnecting the mesenteric-portal blood flow from the systemic circulation while preserving the shunt.

Standard anesthesia consisted of 10 mL of 1% lidocaine injected locally; conscious sedation was with a continuous intravenous infusion of fentanyl (Phentanest, Sankyo, Tokyo, Japan) delivered at 100 μg/h during the procedure. Using a percutaneous transhepatic approach we performed ultrasonography-guided puncture of the portal vein, introduced a 6-Fr C-curved sheath (Medikit), and inserted a 5-Fr cobra catheter (Medikit) into the portal system to measure the pressure in the portal vein sequentially (23 mmHg). Splenic venography revealed a spleno-renal and a spleno-retroperitoneal shunt (Fig. 2). We also inserted a 5-Fr catheter into the inferior mesenteric vein and identified the junction of the inferior mesenteric and the portal vein.

Because we use a balloon catheter as a flow control to prevent coil deviation, we used two 6-F balloon catheters with a 25-mm diameter balloon (Moiyan, Miyano, Osaka, Japan). One was inserted from the percutaneous transhepatic approach and wedged into the splenic vein during embolization, and the other from the right femoral vein wedged into the left adrenal vein.

In case 1, two coil anchors (Medikit) were placed in the proximal part of the splenic vein to prevent coil deviation. Embolization was performed with nine metallic coils (Vortex; Boston Scientific, Watertown, MA, USA) and 10 microcoils (interlocking detachable coils (IDC), Boston Scientific) using a 2.5-Fr microcatheter (Target, Boston Scientific) to prevent obstruction of the inferior mesenteric vein by coil migration. In case 2, one coil anchor (Medikit) was placed on the proximal part of the splenic vein to prevent coil deviation. Embolization was with one 3-dimensional Guglielmi detachable coil (GDC; Boston Scientific) and 11 IDCs (Boston Scientific) using a 2.5-Fr microcatheter (Target, Boston Scientific) to prevent inferior mesenteric vein obstruction by coil migration. In sequence, we then embolized the portal-hepatic vein shunt in the left lobe by introducing five IDCs (Boston Scientific) using a 2.5-Fr microcatheter (Target, Boston Scientific). This disconnected the mesenteric-portal blood flow from the systemic circulation while preserving the shunt.

After successful embolization, the venous phase of superior mesenteric arteriography demonstrated a portal vein trunk toward the liver (Fig. 3). There was enough distance to allow disconnection of the mesenteric-portal blood
flow from the systemic circulation while preserving the shunt (Fig. 1a). The patients’ encephalopathy resolved immediately and permanently and in the course of 30-month follow-up there was no evidence of ascites or esophageal varices. The pre- and postprocedure difference in portal pressure was 5 mmHg (from 18 to 23 mmHg, case 1) and 5 mmHg (from 11 to 16 mmHg, case 2). The blood ammonia level fell by 49 μg/dl from 333 μg/dl (case 1) and by 25 μg/dl from 214 μg/dl (case 2). Neither patient experienced episodes of hepatic coma during the follow-up period.

Discussion

Esophageal and isolated gastric varices with a gastrorenal shunt can be controlled by endoscopic embolization and transvenous retrograde obliteration (BRTO), respectively (8, 9). Gastric varices with a gastropericardiac shunt are much less common than a gastrorenal shunt and more difficult to control with the BRTO technique. At present there are no standard therapies (10).

Spontaneous portal-systemic shunts can be classified as intra- or extrahepatic. Congenital or acquired spontaneous intrahepatic portosystemic shunts rarely result in chronic encephalopathy (11, 12). The portal-systemic shunts in patients with chronic encephalopathy tend to be extrahepatic. A large spontaneous portal-systemic shunt is the most frequent cause of recurrent or persistent hepatic encephalopathy in patients with cirrhosis and no history of surgical or transhepatic portosystemic shunts; this was true in 71% of patients in a recent case-control study (13). After excluding or treating precipitating factors, and in patients who fail to respond to optimal medical treatment, shunt occlusion should be considered immediately as the next therapeutic option (14).

Obliteration of portal-systemic shunts by surgery or percutaneous procedures is effective for intractable portal-systemic encephalopathy. However, due to a postoperative increase in the portal venous pressure, it is often associated with complications such as retention of ascites and worsening of esophageal varices (3, 4). Uflacker et al. (4) reported the occurrence of intraperitoneal bleeding and bleeding from esophageal varices 7 days and 7 months after the embolization of splenoportal shunts, respectively. They concluded that shunt embolization is contraindicated in patients with a marked rise in portal pressure.

BRTO, a different interventional radiologic shunt occlusion technique described by Kanagawa et al. (15) has been performed for gastric varices with a splenoportal shunt. It is less invasive than percutaneous transhepatic or
transileocolic vein obliteration of the shunt. However, introduction of a balloon catheter into the shunt via the left renal vein is difficult in some cases and a second and sometimes a third procedure is needed to obliterate the shunt completely (12, 15, 16). The postoperative increase in the portal venous pressure may lead to the retention of ascites and worsening of the esophageal varices (16).

Selective embolization of the splenic vein has been attempted in some cases with splenorenal shunts because the portal venous pressure does not increase after this procedure. Metallic coils are placed in the splenic vein at the junction of the superior mesenteric vein and the first dividing branch of collateral veins including the splenorenal shunt. A hepatofugal flow can be changed to a hepatopetal splenic venous flow via the splenorenal shunt and the hepatopetal portal-mesenteric venous flow is retained after this procedure. In these patients SPDPS achieved the immediate and permanent clearing of encephalopathy and in the course of 10–30-month follow-up there was no evidence of ascites or esophageal varices. The pre- and postprocedure difference in the portal pressure was 18 mmHg in a patient with a closed shunt and 3 mmHg in another with a preserved shunt. In both of our patients there was enough distance to allow disconnecting the mesenteric-portal blood flow from the systemic circulation while preserving the shunt, therefore we decided to perform SPDPS.

Hepatic function is another important factor for evaluating the eligibility of patients to undergo SPDPS. If the procedure is performed in patients with very small liver vascular beds, the slightly increase in the portal pressure and portal blood volume overload can lead to the retention of ascites and worsening of gastroesophageal varices. Even if the portal flow is increased in patients with poor hepatic function, hepatic encephalopathy may not improve because ammonia is not metabolized. Therefore, this procedure is appropriate only in patients with slightly compromised hepatic function. Mezawa et al. (16) reported a patient with impaired liver function and Child-Pugh class C disease in whom SPDPS was successful and elicited no postoperative liver damage. It is currently unknown whether SPDPS is safe and effective in patients with severe liver dysfunction.

Shunt occlusion with metallic coils (15) and by selective embolization of the splenic vein has been attempted (16). Several metallic coils are required to effectively occlude a shunt and, in addition, a balloon catheter is needed to prevent the coils from migrating into a systemic vein or the heart (12). We avoided coil migration by placing two coil anchors and by wedging a balloon catheter into the left adrenal vein. According to Park et al. (17), introducing an Amplatzer vascular plug (AVP; AGA Medical Corp., Golden Valley, MN, USA) is a useful option for occluding portal-systemic shunts responsible for encephalopathy. We think that this technique is appropriate in patients without sufficient distance to allow disconnecting the mesenteric-portal blood flow from the systemic circulation.

In conclusion, both of our patients experienced improvement in their portosystemic encephalopathy without the manifestation of ascites or esophageal varices and their blood ammonia level decreased without hepatic coma during the follow-up period. SPDPS was an effective and safe method to treat portosystemic encephalopathy in our patients.

REFERENCES

1. Kashida H, Kondo M, Fukunaga T, et al. Reversal of portal-systemic encephalopathy by shunt-preserving disconnection of portal and systemic circulation. Jpn J Gastroenterol 1996;93:96–103
2. Hanna SS, Smith RS III, Henderson JM, et al. Reversal of hepatic encephalopathy after occlusion of total portosystemic shunts. Ann J Surg 1981;142:285–9
3. Potts JR, Henderson JM, Millikan WJ, et al. Restoration of portal venous perfusion and reversal of encephalopathy by balloon occlusion of portal systemic shunt. Gastroenterol 1984;87:208–12
4. Uflacker R, deSilva A, Camerio d’Albuquerque LA, et al. Chronic portosystemic encephalopathy; embolization of portosystemic shunts. Radiology 1987;165:721–5
5. Ito J, Ikeda N, Watanabe A, et al. Obliteration of portal systemic shunts as therapy for hepatic encephalopathy in patients with non-cirrhotic portal hypertension. Jpn J Gastroenterol 1992;77:59–64
6. Kawanaka H, Ohta M, Hashizume M, et al. Portosystemic encephalopathy treated with balloon-occluded retrograde transvenous obliteration. Am J Gastroenterol 1995;90:508–10
7. Akahane T, Iwasaki T, Kobayashi N, et al. Changes in liver function parameters after occlusion of gastrorenal shunts with balloon-occluded retrograde transvenous obliteration. Am J Gastroenterol 1997;92:1026–30
8. Chikamori F, Nishio S, Kuniyoshi N, et al. Blood supply routes of recurrent esophageal varices following endoscopic embolization. Dig Surg 2000;17:17–22
9. Chikamori F, Kuniyoshi N, Shibuya S, et al. Eight years of experience with transjugular retrograde obliteration for gastric varices with gastrorenal shunts. Surgery 2001;129:414–20
10. Chikamori F, Kuniyoshi N, Shibuya S, et al. Correlation between endoscopic and angiographic findings in patients with esophageal and isolated gastric varices. Dig Surg 2001;18:176–81
11. Tanoue S, Kiyosue H, Komatsu E, et al. Symptomatic intrahepatic portosystemic venous shunt: Embolization with an alternative approach. Am J Roentgenol 2003;181:71–8
12. Pocha C, Maliakkal B. Spontaneous intrahepatic portal-systemic venous shunt in the adult: Case report and review of the literature. Dig Dis Sci 2004;49:1201–6
13. Riggi O, Efrati C, Catalano C, et al. High prevalence of spontaneous portal-systemic shunts in persistent hepatic encephalopathy: A case control study. Hepatology 2005;42:1158–65
14 Semia H, Zidi D, Zanditenas, et al. Treatment of chronic portosystemic encephalopathy in cirrhotic patients by embolization of portosystemic shunts. Liver Internatl 2007;27:1389–93
15 Srivastava DN, Yadav S, Sahni P, et al. Emergency percutaneous occlusion of surgical portosystemic shunt using steel coils and balloon catheter. Am J Roentgenol 1999;172:1453–4
16 Mezawa S, Homma H, Akiyama T, et al. Selective embolization of the splenic vein in patients with hepatic encephalopathy and splenorenal shunt. J Vasc Interv Radiol 2004;15:1475–81
17 Park SW, Kang HS, Kim M, et al. Successful occlusion of spontaneous portosystemic shunts leading to encephalopathy in a non-cirrhotic patient by using the Amplatzer vascular plug. Acta Radiol 2007;10:1077–81