Effective balloon-occluded retrograde transvenous obliteration of the superior mesenteric vein - inferior vena cava shunt in a patient with hepatic encephalopathy after living donor liver transplantation

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Abstract

Balloon-occluded retrograde transvenous obliteration (BRTO) has become a common and effective procedure for treating hepatic encephalopathy due to a portosystemic shunt related to cirrhosis of the liver. However, this method of treatment has rarely reported in patients after liver transplantation. We herein report the case of a 52-year-old patient who underwent living donor liver transplantation (LDLT) due to a hepatitis C virus-infected hepatocellular carcinoma that was complicated with portal vein thrombosis and a large portosystemic shunt between the superior mesenteric vein (SMV) and inferior vena cava (IVC). The SMV-IVC shunt was not obliterated during LDLT because there was sufficient portal flow into the graft after reperfusion. However, the patient was postoperatively complicated with encephalopathy due to the portosystemic shunt. BRTO was performed and was demonstrated to have effectively managed the encephalopathy due to the SMV-IVC shunt, while preserving the hepatic function after LDLT.

Keywords: BRTO, Living donor liver transplantation, Portosystemic shunt, Hepatic encephalopathy

List of all abbreviations:

ALT: alanine aminotransferase

AST: aspartate aminotransferase

BRTO: balloon-occluded retrograde transvenous obliteration

CECT: contrast-enhanced computed tomography

IVC: inferior vena cava

LDLT: living donor liver transplantation

PV: portal vein

SMV: superior mesenteric vein

T.Bil: total bilirubin

Introduction

Portal hypertension associated with the development of portal vein thrombosis often occurs in patients with cirrhosis of the liver. The subsequent formation of portosystemic shunts due to formed collateral vessels and hepatic encephalopathy as a consequence are also frequently observed [1]. These kinds of severe complications generally are resolved after liver transplantation without the need for occlusion of the prior portosystemic shunt [2]. However, persistent collateral vessels with a portosystemic shunt can remain, especially after LDLT, due to the smaller graft size, portal hyperperfusion and various other factors [3].

We herein present a case report of a patient who underwent effective BRTO treatment for hepatic encephalopathy due to a persistent SMV-IVC shunt after LDLT.

Case report

A 52-year-old female patient underwent LDLT with splenectomy for hepatitis C virus-infected liver cirrhosis complicated with hepatocellular carcinoma. Before LDLT, contrast-enhanced computed tomography (CECT) revealed a thrombosis of the portal vein (PV) that was 4 cm in length starting 2 cm proximal from the superior mesenteric vein and splenic vein confluence and also a direct SMV-IVC shunt with a diameter less than 1 cm (Fig. 1). A left lobe graft was transplanted from her brother, and the graft volume was 365 ml, corresponding to 34.6% of the recipient’s standard liver volume. On the first and second postoperative days, she underwent relaparotomy because of bleeding from the short gastric veins and inferior diaphragmatic veins, respectively, which was

improved after surgical treatment. On postoperative day 7, thrombosis in the PV was detected by CECT examination, and intravenous infusion of heparin (5,000 U/day) was started.

Two months after the transplant, the patient complained of drowsiness and confusion. The laboratory data showed slightly elevated levels of alanine aminotransferase (ALT, 56 U/l), aspartate aminotransferase (ALT, 57 U/l) and high levels of serum ammonia (185 μg/dl, normal range 0-70), alkaline phosphatase **(**821 IU/l) and total bilirubin (T.Bil, 12.8 mg/dl) which had been gradually decreasing after the LDLT (from 15.1mg/dl). CECT revealed no evidence of an increasing size of the collateral vein vessels or SMV-IVC shunt, and stable hepatic blood flow, as confirmed on a Doppler ultrasound examination. In addition, the previously detected PV thrombosis had disappeared. Cholangiography was performed and did not show biliary stenosis.

To treat the encephalopathy, intravenous drip infusion of a branched-chain amino acid solution (Aminoleban) at 500 ml/day and lactulose (30 ml/day) were started. During the treatment, the ammonia level decreased to 92 μg/dl (total bilirubin to 4.7 mg/dl). After the improvement and stabilization of the patient's general condition and laboratory data, she was discharged and transferred to local hospital.

Four months later, six months after LDLT, the patient presented with a new episode of hepatic encephalopathy with mild confusion, and she was readmitted to our department. The CT scans revealed stenosis of the PV anastomosis, which was confirmed by angiography and successfully treated with stent placement and percutaneous transluminal angioplasty ballooning. Subsequently, the patient became asymptomatic and was discharged with recommended anticoagulation therapy. One year later, however, hepatic encephalopathy appeared again. The laboratory data showed an increased ammonia level (169 μg/dl) with normal liver and renal function tests [ALT (30 U/l), AST (35 U/l), T.Bil (0.8 mg/dl), prothrombin time (72%), albumin (3.4 g/dl), platelets

($22.9x10^4$ /μl) and creatinine (0.48 mg/dl)]. CECT revealed satisfactory PV stent placement without stenosis. Furthermore, a Doppler ultrasound examination showed sufficient hepatic blood flow. A biopsy showed mild hepatitis that was not considered severe enough to induce portal hypertension. Nevertheless, the encephalopathy was refractory to conservative pharmacotherapy, and the ammonia level continued to rise up to 219 mg/dl. The hepatic encephalopathy was graded as Western Haven Criteria grade III. Ultimately, the previously detected SMV-IVC shunt (Figs. 2A and 2B) was deemed to be the main cause of hepatic encephalopathy, and BRTO was therefore indicated.

BRTO was performed according to the method reported by Kanagawa et al [4]. In brief, a 6-Fr balloon catheter (Cobra type Selecon MP II, Terumo, Tokyo, Japan) was introduced into the SMV-IVC shunt via the right femoral vein. The SMV-IVC shunt was visualized after retrograde venography using Iopamiron 300 contrast medium (Schering, Osaka, Japan) under inflation of the balloon (Fig. 2C). Interlocking detachable coils were used to embolize the small outflow vessels. Initially, 10% ethanolamine oleate solution was used as a sclerosing agent; however, the degree of blood flow stagnation was insufficient to use ethanolamine oleate. Therefore, the sclerosing agent was a mixture of n-butyl-2-cyanoacrylate and Lipiodol (ratio=1:4) and the total volume inserted through the 1.8 Fr microcatheter was 4 ml. After BRTO, control venography showed the disappearance of the portosystemic shunt with sufficient filling by the sclerosing agent (Fig. 2D). Afterwards, the whole catheter system was withdrawn. No complication occurred during the procedure. Subsequently, the patient's blood ammonia level decreased to within the normal range (51 μg/dl) with a normal liver function [ALT (50 U/l), AST (54 U/l), T.Bil (0.7 mg/dl), prothrombin time (60%), albumin (3.6 g/dl), platelets ($32.9x10^4$ /μl)], and she has not experienced any episodes of hepatic encephalopathy since then. Presently, eleven months after BRTO, the patient is well, and follow-up CT has shown no evidence of the portosystemic shunt (Fig. 3), with sufficient hepatic blood flow.

Discussion

It remains controversial as to whether a portosystemic shunt detected before liver transplantation should be occluded during liver transplantation. A portosystemic shunt could decrease the portal vein flow after liver transplantation, leading to the subsequent formation of portal vein thrombosis, graft atrophy and other serious consequences [5, 6]. On the other hand, the presence of a shunt can have a positive effect on the liver perfusion in cases with relative portal hypertension in the early postoperative period, especially after LDLT [5, 7]. Moreover, even after shunt vessel ligation during LDLT, there is still chance of recurrence after surgery and this procedure might be ineffective [8].

It should be noted that in our case, the portosystemic encephalopathy occurred one year after stent placement and percutaneous transluminal angioplasty ballooning for stenosis of the PV anastomosis. Even the stent placement without further recurrence of the PV stenosis and with sufficient hepatic blood flow did not prevent the occurrence of portosystemic encephalopathy due to the persistent portosystemic shunt.

Hepatic encephalopathy that occurs due to a portosystemic shunt after LDLT requires immediate and adequate treatment. Nevertheless, conservative therapeutic treatment for this complication is often applies, and may be ineffective, like it was in our case [9]. Therefore, BRTO has recently been reported as a less invasive treatment method for portosystemic encephalopathy [6, 7, 9-12]. However, the effectiveness of BRTO treatment for portosystemic encephalopathy after LDLT has rarely been reported, and BRTO for a SMV-IVC shunt after liver transplantation has not been reported previously [13]. Yokoyama et al. reported successful BRTO treatment for a patient with hyperammonemic encephalopathy, which occurred 10 years after LDLT [13]. However, that patient did not have a cirrhotic liver, and the cause of the portosystemic shunt was unknown. Therefore, this condition may occur at any time, despite the presence of a

normal liver function without signs of portal hypertension, in patients with a persistent portosystemic shunt after LDLT.

The effectiveness of BRTO treatment for patients after LDLT with gastric varices and liver dysfunction, including hyperbilirubinemia and hyperammonemia, without hepatic encephalopathy caused by prolonged portosystemic shunts has also been reported [14]. BRTO seems to be an effective treatment, regardless of the interval between the development of symptoms due to the portosystemic shunt and LDLT.

In conclusion, the lower invasiveness and lack of impact on the transplanted graft function are the most important points of BRTO treatment for portosystemic encephalopathy after LDLT, especially for patients like our present case, with normal liver function and who have previously undergone several abdominal surgeries that have likely led to the formation of severe intra-abdominal adhesions. Moreover, the other advantages that we experienced were complete disappearance of the encephalopathy symptoms and rapid recovery after BRTO treatment. BRTO is therefore considered to be an effective management strategy for portosystemic encephalopathy that preserves the hepatic function after LDLT.

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Figure legends:

Fig. 1. An axial section of abdominal CECT showed collateral vessels with a direct SMV-IVC shunt (white arrow).

Fig. 2. Portography showed collateral vessel from the SMV (white arrows) in the early phase (**A**), and the shunt (white arrowheads) was visualized directly going to the IVC (black arrows) in the late phase (**B**). **C**, Retrograde venography before BRTO after inserting a 1.8 Fr microcatheter directly into the SMV-IVC shunt (white arrows). **D**, The control view after BRTO showed that the shunt was sufficiently filled with sclerosing agent (white arrow).

Fig. 3. A follow-up CT scan taken nine months after BRTO showed the sclerosant (white arrow) with the complete disappearance of the SMV-IVC shunt.

Fig. 1

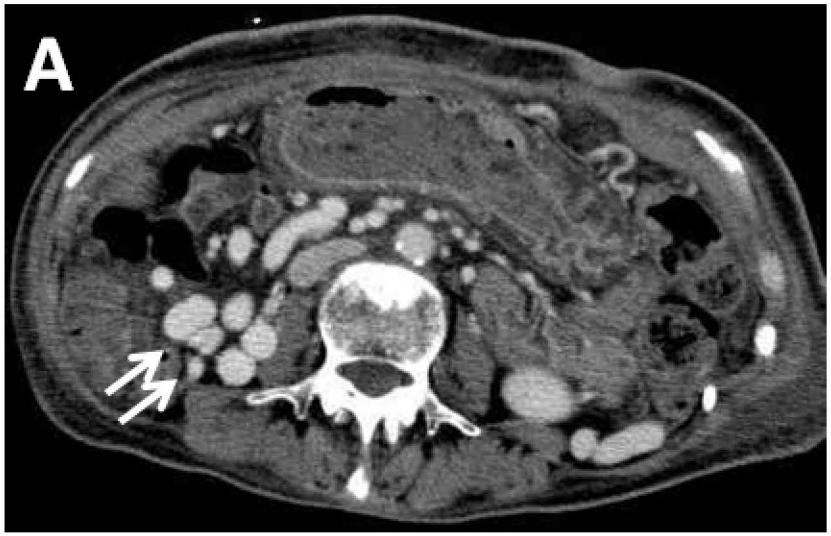

Fig. 2

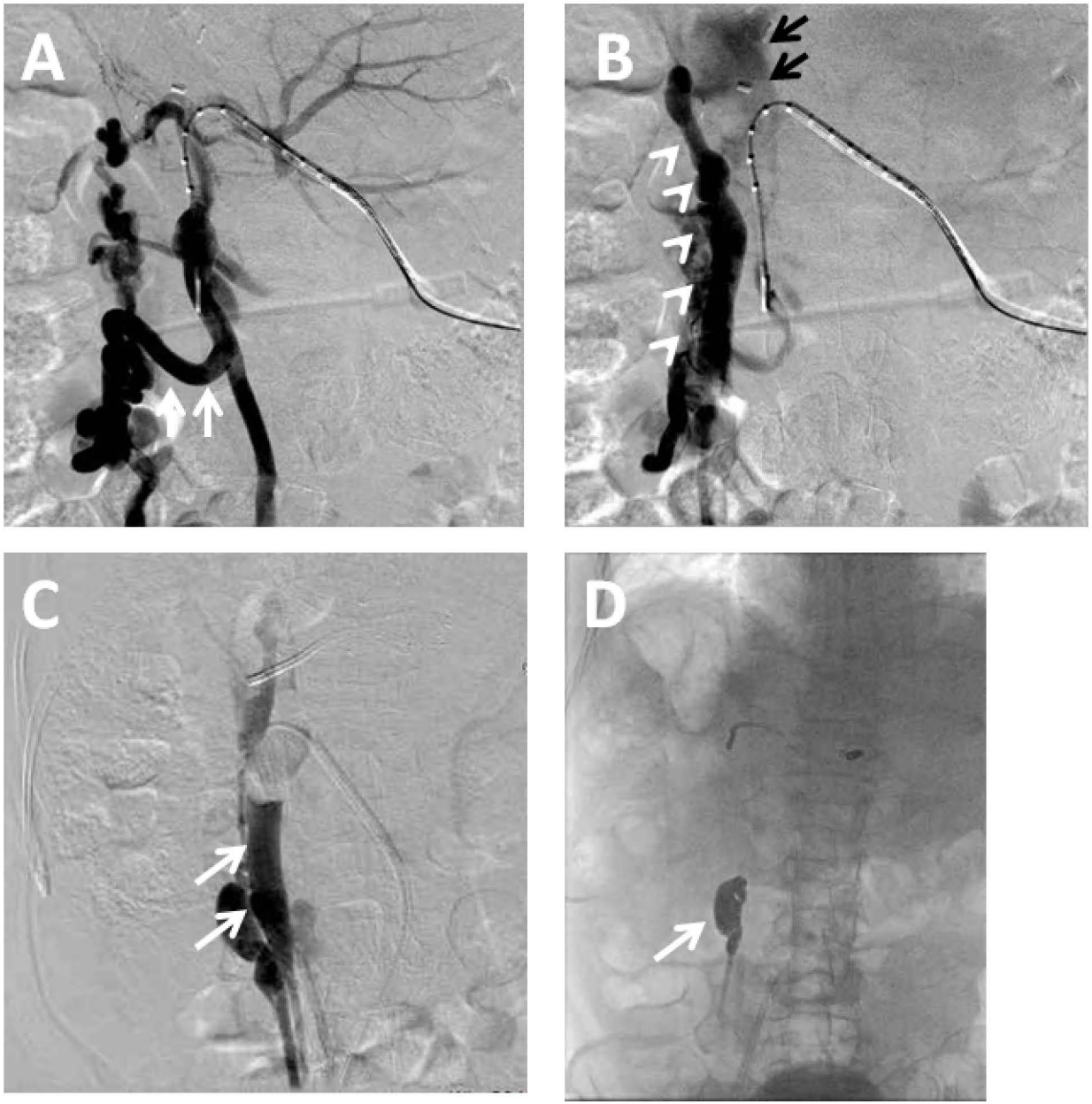

Fig. 3