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Transhepatic splenic vein embolization during temporary balloon occlusion of a spontaneous portosystemic shunt for chronic portosystemic encephalopathy

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Abstract

A 69-year-old woman undergoing medical treatment for liver cirrhosis with chronic hepatic encephalopathy developed consciousness disturbance. Emergency blood analysis revealed hyperammonemia, and abdominal CT demonstrated a large splenorenal shunt as the cause of her symptoms. The splenic vein was embolized at the portal side of the shunt entrance via the percutaneous transhepatic route, while preserving the splenorenal shunt and preventing retrograde flow to the splenorenal shunt from the superior mesenteric vein. The temporary balloon occlusion of the outflow pathway of the shunt allowed safe and accurate embolization with detachable coils under the flow control of the shunt. After the interventional procedure, her blood NH_3 level decreased markedly, and the hepatic encephalopathy immediately disappeared. Postprocedural elevation of portal blood pressure was slight.

Keywords: Interventional radiology; Portosystemic shunt; Portosystemic encephalopathy; Embolization; Balloon occlusion

1. Introduction

Portosystemic shunts are usually formed by portal hypertension caused by liver cirrhosis and can lead to hepatic encephalopathy [1]. Although they actually serve as a mechanism to decrease portal blood pressure, neurotoxic substances such as ammonia from the gut are not efficiently metabolized in the liver and flow directly into the systemic circulation via portosystemic shunts, sometimes causing hepatic encephalopathy [2–4]. Chronic hepatic encephalopathy due to portosystemic shunts may be refractory to medical treatment.

Obliteration of portosystemic shunts by surgical ligation or endovascular procedures, while effective for portosystemic encephalopathy, but is often associated with complications such as retention of ascites, worsening of esophageal varices, and the relapse of hepatic encephalopathy as a result of the formation of new collateral pathways because of a postoperative increase in portal blood pressure [5,6].

We report a patient with chronic hepatic encephalopathy treated with transhepatic splenic vein embolization during temporary balloon occlusion of the portosystemic shunt.

2. Case report

A 69-year-old woman underwent repeat hospitalization for type C hepatitis viral liver cirrhosis and chronic hepatic encephalopathy. Emergency blood analysis for unconsciousness revealed a high level of NH_3 (325 μg/dl) (normal range: 30–80 μg/dl). Abdominal CT showed splenomegaly and a large splenorenal shunt that descended from the splenic vein and flowed into the left renal vein (Fig. 1). No ascites was

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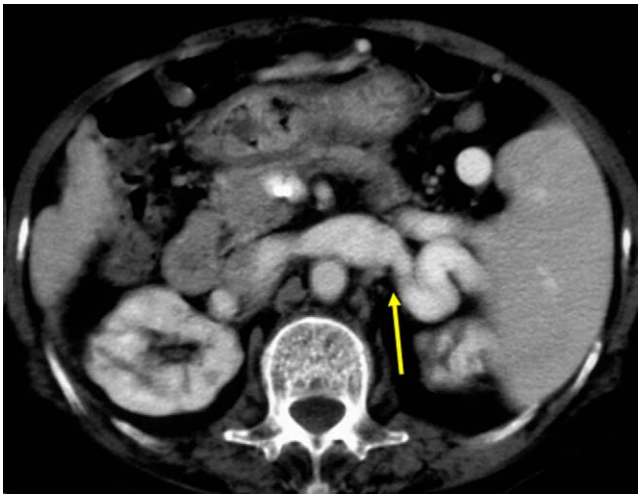

Fig. 1. Abdominal enhanced CT. A large splenorenal shunt flowing into the left renal vein is noted (arrow).

present. Her liver function was comparatively good, with total bilirubin 1.5 mg/ml, albumin 3.81 g/dl, and PT 87.4%. Platelet count was $5.8 \times 10^4\ \mu l^{-1}$. The large portosystemic shunt was considered to be the cause of her severe hepatic encephalopathy. To evaluate her liver blood-flow dynamics, celiac angiography (Fig. 2a) and portography via the superior mesenteric artery (Fig. 2b) were carried out using a 4-Fr. RC2 catheter (Medikit, Tokyo, Japan). Retrograde flow from the superior mesenteric vein to the splenic vein and the large splenorenal shunt were recognized on the arterioportography (Fig. 2b). Although it was thought that the hepatic encephalopathy would disappear upon closing the shunt with endovascular embolization, such a procedure might cause a marked increase in portal blood pressure and cause esophageal varices or new collateral formation, leading to a recurrence of hepatic encephalopathy. We, therefore, selected another interventional procedure, transhepatic splenic vein embolization, to prevent increasing portal blood pressure.

The left branch of the portal vein was punctured under local anesthesia using an 18-gauge needle under ultrasonic guidance. A 4 Fr. straight catheter was advanced into the splenic vein over the previously inserted 0.035 in. guidewire, and direct portography was performed. A large and tortuous splenorenal shunt was diagnosed on percutaneous transhepatic portography (Fig. 3a). Contrast medium rapidly flowed into the left renal vein and the inferior vena cava through the splenorenal shunt (Fig. 3b). A microcatheter was coaxially advanced to the splenic vein to embolize the proximal side of the vessel, to preserve the splenorenal shunt. We used seven spiral-type detachable microcoils (DCS-18; Cook Inc., Indiana, USA) of 20 cm in length, 10 mm in diameter, and embolized the vessel at a site 10 mm distant from the shunt site (Fig. 4). Concurrently, a 7-Fr. cobra-shaped 13 mm diameter balloon catheter (Selecon MP catheter; Clinical Supply, Gifu, Japan) was inserted from the right femoral vein and advanced into the left renal vein, a main outflow pathway of the splenorenal shunt. The outflow pathway of the shunt was

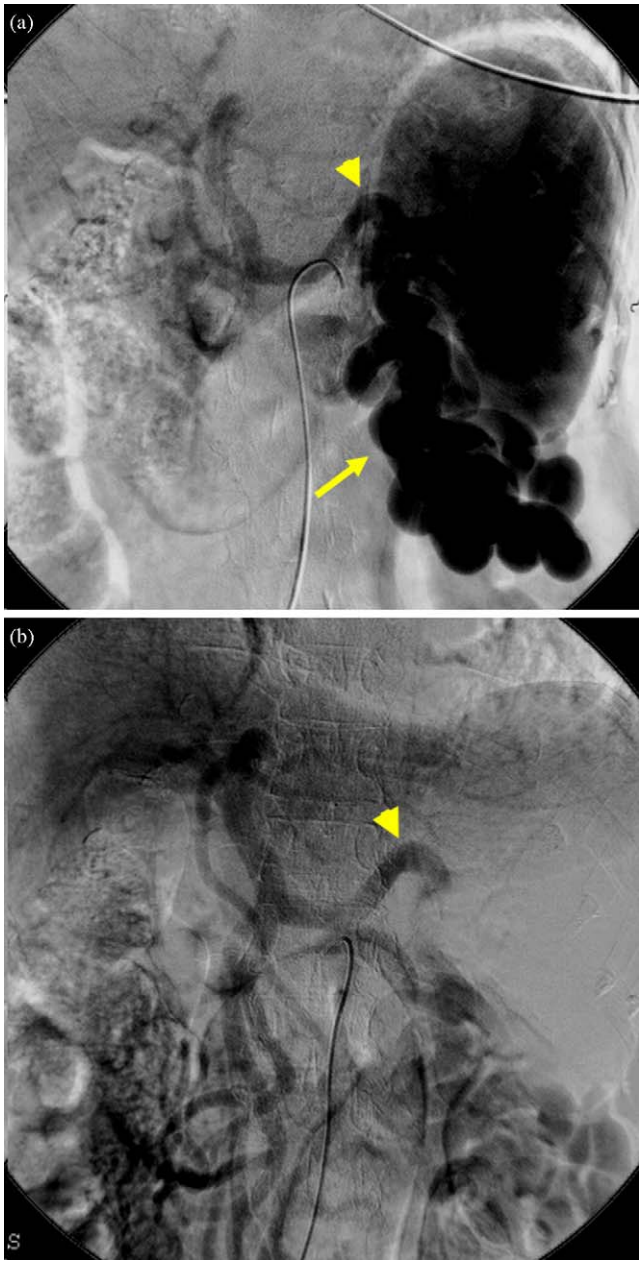

Fig. 2. (a) Venous phase of celiac angiography and (b) portography via the superior mesenteric artery. Splenomegaly and a large, tortuous splenorenal shunt are recognized (arrow). The entrance of the shunt is about 4.0 cm from the portal stem (arrowhead).

temporarily occluded by the inflated balloon. Temporal balloon occlusion of shunt flow prevented migration or deviation of the coils.

After embolization, splenic venous blood flow to the systemic circulation through the shunt was preserved, and flow to the portal vein from the splenic vein was not seen on celiac angiography (Fig. 5a). Moreover, superior mesenteric venous retrograde flow to the splenorenal shunt was not recognized on portography via the superior mesenteric artery (Fig. 5b). Portal blood pressure was measured through the portal vein catheter by polygraph system (RMC-3100;