Case report

Varicose bleeding after liver transplantation in a patient with severe portosystemic shunts

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Recipients for liver transplantation often have portosystemic shunts due to portal hypertension. It is an important problem whether such shunts should be ligated during operations. Ligating the shunts seems of benefit for increasing portal blood flow to the liver, but it is sometimes difficult technically, and it is invasive to the patient. We experienced a recipient with huge portosystemic shunts and no esophageal varices before living-related liver transplantation. Some shunts were ligated during operation to increase portal blood flow to the graft. Unfortunately, the patient suffered severe bleeding from esophagogastric varices after he underwent retransplantation owing to accidental liver failure. Based on our experience, extreme care should be exercised to avoid varicose bleeding after ligating the portosystemic shunts of liver transplantation patients.

Key words: liver transplantation, portosystemic shunt, varix, portal blood flow

Introduction

The development of portosystemic collateral pathways is one of the characteristics of portal hypertension, and these vessels are often seen in candidates for orthotopic liver transplantation (OLT). The collaterals are thought to close after operation because portal hypertension can be relieved by implantation of a normal graft, but they could decrease the portal blood flow to the graft and cause liver failure. We report a patient who suffered recurrent varicose hemorrhage after some portosystemic shunts were ligated during the OLT operation.

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Case report

A 30-year-old man was admitted to our hospital with severe liver dysfunction in February 1999. He had been noted to have splenomegaly 8 months after birth. He had suffered gastroenteral bleeding at 14 years of age and was diagnosed as having idiopathic extrahepatic portal vein stenosis and portal hypertension, as the liver biopsy demonstrated no cirrhotic change. Liver cirrhosis was proved by liver biopsy at age 22 years.

He was 181 cm in height and 58 kg in weight. His skin and conjunctiva were icteric, and his spleen was palpable. He was negative for hepatitis B surface antigen (HBsAg), antibody (HBsAb) and hepatitis C virus (HCV) antibody. He showed severe liver dysfunction with total bilirubin of 5.0 mg/dl, NH₃ 69 mg/dl, indocyanine green retention at 15 min 52.4%, and a Child-Pugh score 11. His liver was cirrhotic, and the portal vein had severe stenosis. There were huge retroperitoneal pathways from the splenic vein in addition to the collateral vessels around the portal vein (Fig. 1).

He underwent orthotopic living-related liver transplantation with the right lobe (480g) from his mother on September 2. The recipient’s portal vein was narrow and had poor blood flow. A white, fibrous embolus was removed from the lumen. Reconstruction was performed between the trunks of the donor’s and recipient’s portal veins with running sutures of 5–0 polypropylene. There were so many large retroperitoneal vessels that the resection was expected to be extremely difficult technically. Some pathways were ligated at the end of the operation, as the portal flow was expected to increase.

The patient’s clinical condition was stable after the operation, though his mental deterioration continued and laboratory findings showed liver dysfunction. Doppler ultrasonographic studies, done daily postoperatively, revealed good arterial blood flow and hepatopetal portal flow. Liver biopsy revealed mild re-
jection on September 15 and acute rejection on September 21. Accidental pleural bleeding during the second biopsy caused hypotension followed by hepatic and renal failure. Doppler ultrasonography demonstrated that the portal venous flow was hepatofugal (Fig. 2a).

Retransplantation was performed with the right lobe (640 g) from his brother’s liver on September 28. The resected liver revealed total hepatocellular necrosis, indicating severe ischemic damage. The portal venous flow was hepatopetal or pendulating (“back and forth”) after the retransplantation (Fig. 2b).

The patient suffered repeated varicose bleeding beginning 2 weeks after the operation that was difficult to control. Several sclerotherapy sessions and radiological coils placed in the splenic artery completed the hemostasis in somewhat over 1 month. Endoscopic examination after that showed some large varices developing in the rectum, which fortunately did not bleed. He died from pneumonia and sepsis on January 11, 2000. The autopsy examination demonstrated that there were no surgical problems with the reconstructed portal vein, hepatic vein, or hepatic artery (e.g., stretching, kinking, obstruction). There were numerous collateral vessels occupying the upper retroperitoneal space and in part spreading along the esophagus and the rectum.

**Discussion**

Recipients before OLT often have portal hypertension and portosystemic collateral vessels. The collateral pathways are usually not ligated because the portal pressure can be reduced by implanting a normal graft, after which the collaterals tend to diminish.1 Some collateral vessels may remain open, however, and hepatofugal blood flow through such collateral pathways may persist after implantation of a normal graft, but the presence of such vessels matters little. Hadengue et al.2 reported that hepatic blood flow was increased after liver transplantation, although blood flow in the azygos vein, which provides the superior portosystemic collateral blood flow, remained elevated several months after liver transplantation.

It is feared that portal blood flow would be shunted away from the new liver through spontaneous or surgi-