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CASE REPORT

Severe fatty change of the graft liver caused by a portosystemic shunt of mesenteric varices

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Abstract Portosystemic shunt is a common complication in patients with portal hypertension. Mesenteric varix is one of the collaterals that can cause post-transplant liver dysfunction. In this case report, a 45-year-old woman underwent living relative donor liver transplantation for alcoholic cirrhosis. Although the early postoperative course was uneventful, she was readmitted for treatment of liver hypofunction. Fatty change in the graft liver was confirmed by histopathology of the biopsy specimen. The venous phase of a superior mesenteric angiogram revealed large-caliber mesenteric varices comprising portosystemic venous shunts. Surgery was performed to ligate the shunts. The intraoperative color Doppler ultrasonography showed hepatofugal portal blood flow, which was corrected to hepatopetal blood flow by clamping the shunt vessels. The portal pressure was moderately elevated from 13.6 cm to 21.8 cm H_2O. Two shunt vessels were ligated and divided. Her liver function returned to nearly normal thereafter. We recommend that descending collaterals be divided during liver transplantation.

Keywords Liver transplantation · Fatty liver · Portosystemic shunt · Mesenteric varix · Liver dysfunction · Portal blood flow

Introduction

Graft liver hypofunction is a serious problem in post-transplant patients. Acute rejection, viral infection, stricture of the biliary anastomosis, stricture of the vascular anastomosis, and drug toxicity are common reasons for graft liver dysfunction, while portosystemic shunt of mesenteric varices as a cause of post-transplant liver hypofunction has rarely been reported. We report a case of post-transplant liver hypofunction caused by portosystemic shunt of mesenteric varices.

Case report

A 45-year-old female underwent living relative donor liver transplantation for end-stage liver disease. Primary sclerosing cholangitis was suggested preoperatively. The graft liver was donated from her brother and included the left lobe with the middle hepatic vein (658 g). The graft body weight ratio was 0.93% and comprised 52.8% of her standard liver volume. Histocompatibility was excellent, ABO blood type was identical, Rh was incompatible, HLA was identical, and all lymphocyte crossmatch tests were negative. The intra- and postoperative course was uneventful, and the patient was discharged from the hospital 1 month after the operation. The histopathology of the native liver revealed micronodular cirrhosis with Mallory's body. Her liver disease was alcoholic cirrhosis, although she insisted she had ceased drinking 3 years prior to diagnosis. Three months after transplantation, although she had no physical symptoms, she was readmitted to our facility for treatment of liver dysfunction; we found that her aspartate aminotransferase (AST) was 166 U/L, alanine aminotransferase (ALT) was 40 U/L, gamma glutamyl transpeptidase was 167 U/L, total bilirubin was 1.2 mg/dl, and ammonia was 90 μg/dl. Her liver function tests returned rapidly to normal after rest (i.e., without treatment), and she was discharged 3 weeks later. However, her serum transaminase levels were reelevated to 144 U/L of AST and 31 U/L of ALT in the 6th postoperative month. The plain computed tomography (CT)

scan showed severe fatty change of the grafted liver (Fig. 1), and color Doppler ultrasonography revealed poor portal flow. Fatty change of the grafted liver was confirmed by histopathologic study of the liver biopsy specimen. Angiography was performed to evaluate the portal venous blood flow. The venous phase of superior mesenteric angiography revealed large-caliber mesenteric varices comprising a portosystemic venous shunt (Fig. 2). Surgery was performed to ligate the shunt vessels subsequently to interventional shunt closing failure. Intraoperative color Doppler ultrasonography showed hepatofugal portal blood flow, which was corrected to hepatopetal blood flow by clamping the shunt vessels (Fig. 3). The portal pressure was moderately elevated from 13.6 cm to 21.8 cm H_2O. Two shunt vessels were ligated and divided. Her liver function returned to nearly normal and she was discharged from the hospital. Indocyanine green 15-min retention test was 11% at 6 months after the second operation, which showed marked improvement from the preoperative value of 37%. Similar improvement was also found in the serial CT value, which went from –24.4 Hounsfield units (HU) before the second operation to a postoperative value of +20 HU (Fig. 4). The patient is now well and can do some housework, although she is suspected of alcohol recidivism.

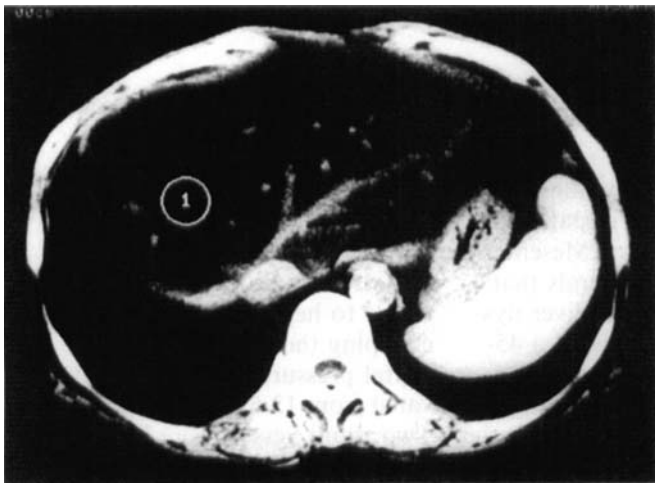

Fig. 1. Plain CT scan of the grafted liver in the 6th postoperative month shows marked low density of the liver parenchyma, with a CT value of –24.4 HU. Note that intrahepatic vessels are effectively contrast-enhanced, given their relatively high density

Discussion

There are several reasons for post-transplant graft liver hypofunction, commonly including acute rejection, stricture of the vascular anastomosis, stricture of the biliary anastomosis, viral infection, and drug toxicity. These postoperative complications should be treated rapidly; delay can result in graft loss or patient death. We report a rare cause of portosystemic shunt of mesenteric varices as a cause of post-transplant liver hypofunction.

Extrahepatic portosystemic collaterals are common clinical consequences in patients with portal hypertension. Dilation of preexisting embryonic channels is thought to be the main mechanism of evolution of these collaterals [3]. Gastroesophageal and rectal collaterals along with umbilical vein remnant are common. On the other hand, collaterals developing between the portal system and the posterior abdominal wall are relatively rare. Mesenteric varix is one of the latter type of col-

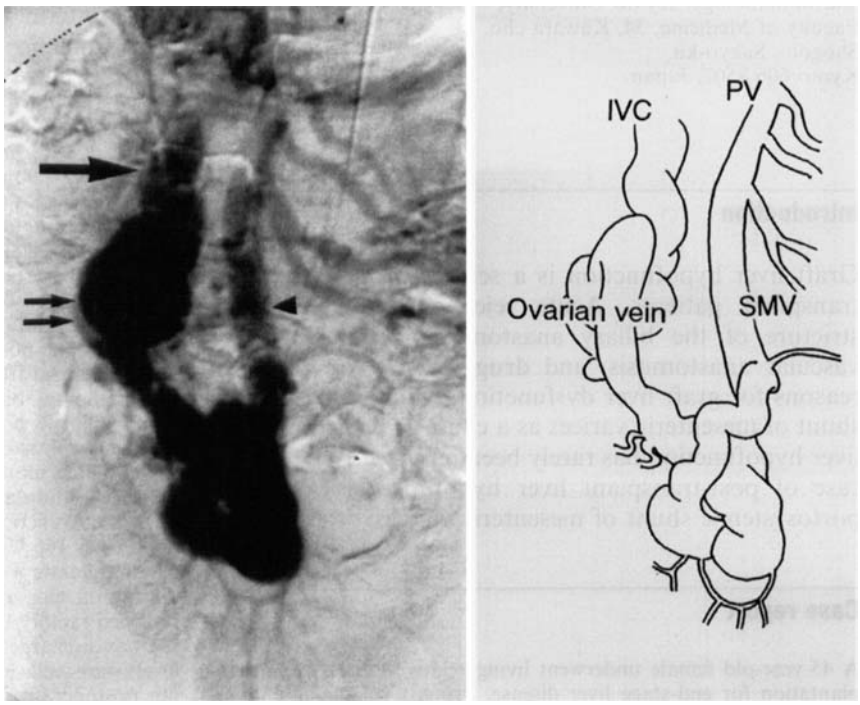

Fig. 2. Portogram shows descending portosystemic venous shunt. The portal flow is sealed from the superior mesenteric vein (*arrowhead*) to the IVC (*arrow*) by shunting of the dilated right ovarian vein (*double arrows*). *PV* Portal vein, *SMV* superior mesenteric vein, *IVC* inferior vena cava

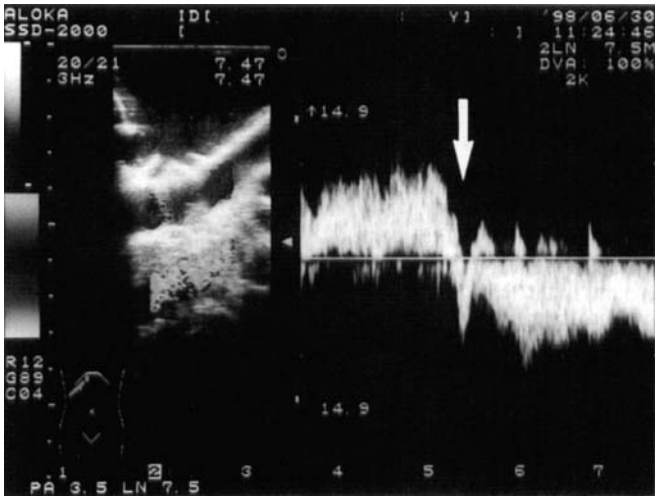

Fig. 3. Intraoperative color Doppler ultrasonography shows hepatofugal portal blood flow, which is corrected to hepatopetal blood flow by clamping the shunt vessels (*arrow*)

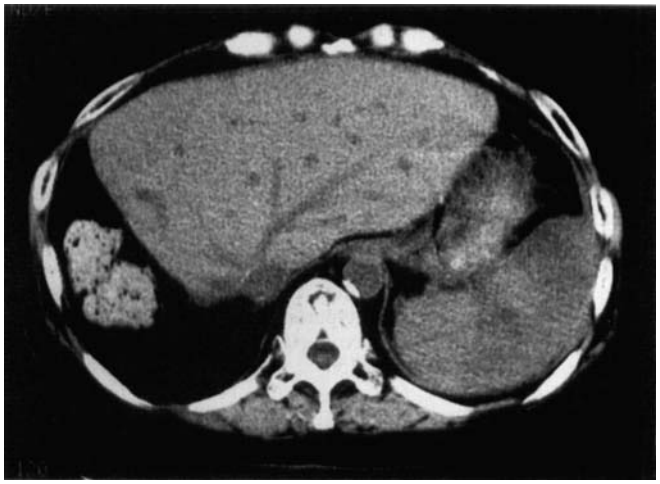

Fig. 4. Plan CT scan of the grafted liver in the 6th month after the shunt ligation. Scan reveals recovery of the density of liver parenchyma, with a CT value of +20 HU

laterals, and only a few case reports involving it have been published to date [6, 7]. The collaterals can be divided into two major categories: ascending and descending collaterals [2]. Ascending collaterals involve mainly the gastric coronary vein, and rupture of esophageal varices is frequent. In descending collaterals, splenorenal shunt is typical, and refractory hepatic encephalopathy often occurs. Descending collaterals such as mesenteric varices form large-caliber shunt vessels, and blood flow increases considerably according to the siphon phenomenon, that is, not in proportion to graft vascular resistance. The division of these shunts is expected to increase the hepatopetal blood flow without obvious elevation of the portal blood pressure [4]. In the present case, portal blood flow was corrected from hepatofugal to hepatopetal by shunt occlusion with minimum elevation of the portal pressure. The occlusion of these collaterals during liver transplantation is controversial. Some transplant surgeons insist that these embryonic preexisting shunts are spontaneously occluded because portal pressure drops to normal after the introduction of a normal liver. However, descending collaterals seem to have difficulty closing spontaneously because of the siphon phenomenon.

Reasons for fatty liver include alcohol excess, metabolic diseases, medications, nutritional disorders, and so on. The mechanisms for the development of fatty liver are varied, though a frequent mechanism is reduction in the hepatic oxidation of fatty acids, often due to mitochondrial dysfunction [5].

It is apparent that the cause of the present patient's early post-transplant liver hypofunction is alcohol ingestion. Her post-transplant course was typical for alcohol recidivism. The Mallory's body found in the native liver shows her alcohol recidivism just before the liver transplantation [1]. Her alcohol ingestion continued without any cessation pre- and post-liver-transplantation. Our frequent investigation failed to prove her alcohol recidivism, because all her family members denied her alcohol ingestion. Such recidivism was confirmed 2 years after the transplantation. Considering her alcohol history, improvement of the fatty change of the grafted liver was established by the shunt occlusion surgery because her CT value 6 months after the second surgery significantly improved, despite continuous exposure to alcohol toxicity. Her post-transplant course seemed a vicious cycle: alcohol ingestion led to liver damage and an increase in vascular resistance of the grafted liver. The increase of this resistance may lead to portosystemic shunt flow, while descending collaterals of mesenteric varices increase its blood flow considerably according to the siphon phenomenon. Such a shunt decreases portal blood flow to the liver, which leads to fatty change of the grafted liver, which in turn leads to vascular resistance of the grafted liver. The second surgery regarding the shunt occlusion disrupted this vicious cycle. We therefore recommend that descending collaterals be divided during liver transplantation, despite the likelihood that the trigger of the hepatofugal blood flow in our case was alcohol recidivism.

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