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CASE REPORT

Mesorenal shunt using inferior mesenteric vein and left renal vein in a case of LDLT

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Summary

Adult-to-adult living donor liver transplantation (LDLT) has become an established treatment option around the world. However, small-for-size graft syndrome remains one of the most serious complications affecting transplant outcomes. Excessive portal hypertension and overperfusion have been shown to play a causative role in this graft injury. Recently, portal hypertension *per se* has been considered detrimental to graft function, and thus to be avoided for successful outcomes after LDLT. We constructed a mesorenal shunt with anastomosis of the inferior mesenteric vein and left renal vein in the case of an LDLT recipient who showed high portal vein pressure after graft reperfusion. The inferior mesenteric vein is close to the left renal vein, and the anastomosis was obtained with relative ease. The shunt was effective in decreasing portal vein pressure, and postoperative graft function was satisfactory. This new method represents an option for attenuating portal hypertension when elevated portal vein pressure is observed in adult LDLT after graft reperfusion.

Introduction

Living donor liver transplantation (LDLT) was first attempted in pediatric patients to decrease mortality among patients on deceased donor liver transplantation waiting lists. With excellent patient and graft survival, the encouraging results from pediatric LDLT have led to the development of adult-to-adult LDLT programs. However, one of the main problems in adult-to-adult LDLT is small-for-size graft syndrome (SFS), which often occurs for graft-to-recipient body weight ratio (GRWR) < 0.8% [1]. Portal hypertension and portal overperfusion have been reported as determining factors for injury to endothelial cells and parenchyma related to SFS [2,3]. Moreover, such graft dysfunction is also related to venous congestion of the graft and preoperative recipient conditions, and patients with high Model for End-Stage Liver Disease score or portal hypertension may develop SFS even if receiving a graft with GRWR > 0.8%. When portal vein pressure (PVP) after reperfusion is high, recipient has risk of complications similar to those seen in SFS, irrespective of graft-size matching [4].

In recent years, in the case of suspected SFS syndrome, we routinely monitor PVP during the transplant operation. When PVP is >20 mmHg after graft reperfusion, we conduct 'inflow modulation', such as splenectomy and partial portosystemic shunting, to decrease PVP [5–8].

Herein we describe a new method of constructing a mesorenal shunt as an option for decompression of portal hypertension.

Case report

A 64-year-old woman suffered from massive pleural effusion and ascites resulting from end-stage cryptogenic liver cirrhosis. Child-Turcotte-Pugh score was 9 and Model for End-Stage Liver Disease score was 13.

Preoperatively, computed tomography (CT) revealed thrombus in the portal vein trunk and splenic vein (Fig. 1). The inferior mesenteric vein (IMV) was observed to drain into the superior mesenteric vein.

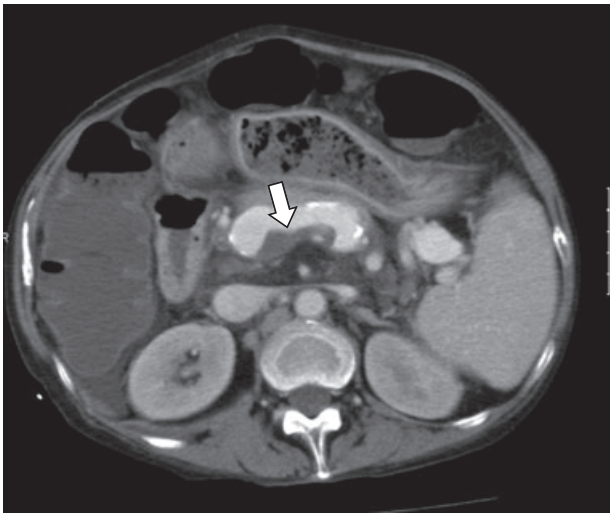

Figure 1 Preoperative computed tomography demonstrated extrahepatic portal vein thrombosis and splenic vein thrombosis (white arrow).

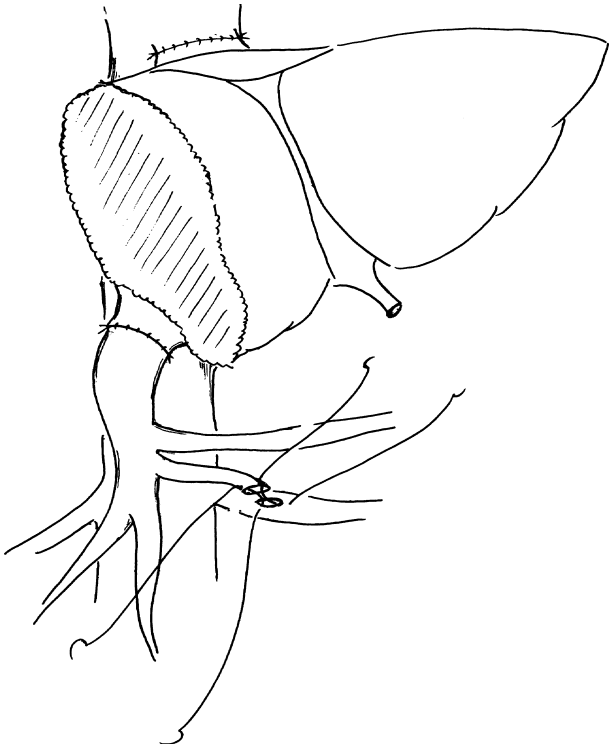

Figure 2 Scheme of the mesorenal shunt with end-to-side anastomosis of the inferior mesenteric vein and left renal vein in this case.

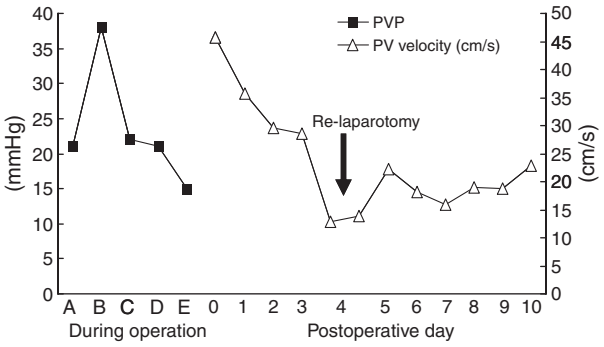

Figure 3 Clinical course of the case. Outlined triangle indicates portal vein velocity and black square indicates portal vein pressure (PVP) intraoperatively. (A) PVP at catheter insertion. (B) PVP after clamping the portal vein. (C) PVP after graft reperfusion. (D) PVP after splenectomy. (E) After construction of the mesorenal shunt.

The patient underwent LDLT using a left lobe graft from her daughter. Graft weight and GRWR were 410 g and 0.95% respectively. Thrombectomy in the portal vein trunk was performed, but the splenic vein thrombus with calcification could not be removed because of technicalities. In the recipient, PVP was 21 mmHg at the time of catheter insertion, rising to 38 mmHg after clamping of the portal vein. After reperfusion of the graft with portal blood, PVP was 22 mmHg. We therefore considered that a decrease in PVP was necessary. First, splenectomy was performed. However, PVP remained high (21 mmHg). Second, we constructed a mesorenal shunt with end-to-side anastomosis of the IMV and left renal vein (Fig. 2). PVP subsequently reduced to 15 mmHg (Fig. 3). Operation time was 12 h 39 min. Blood loss was 7190 ml including massive ascites 3 l at laparotomy. Ten units of packed red cells transfusion and 10 units of fresh frozen plasma were received.

Postoperatively, liver function tests were normalized promptly with no hyperammonemia. However, on postoperative day 4, abdominal Doppler ultrasonography revealed intrahepatic portal vein thrombus and decreased portal vein velocity. CT showed that some of the preexisting splenic vein thrombus had disappeared and portal vein thrombus had appeared in the umbilical portion. Emergent laparotomy was performed and a catheter was inserted into a mesenteric vein branch. Continuous infusion of urokinase and heparin was started through the catheter. The next day, Doppler ultrasonography revealed disappearance of the intrahepatic portal vein thrombus and increased portal vein velocity. After that, postoperative course was uneventful, and the thoracic drainage tube was removed on postoperative day 7. She was discharged on postoperative day 36.

Postoperative graft liver regeneration was confirmed. At 1, 4, 12 and 20 months postoperatively, according to CT volumetry, graft volume was estimated at 696.5 ml, 798 ml, 698.6 ml, and 765 ml respectively. Plasma ammo-

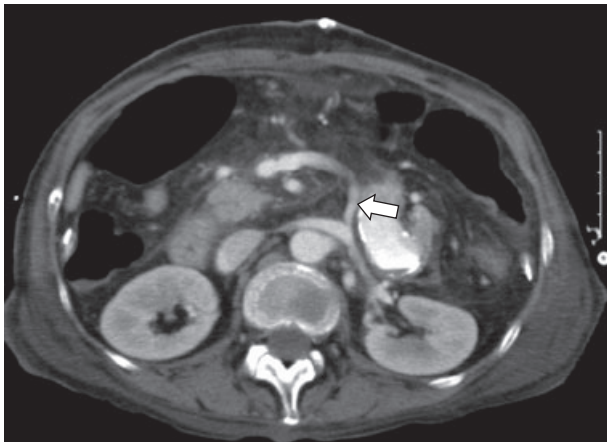

Figure 4 The mesorenal shunt remained patent without expansion (white arrow).

nia concentration remained low. As of 20 months postoperatively, the mesorenal shunt remained patent without expansion (Fig. 4).

Discussion

Adult-to-adult LDLT has become an established treatment option around the world. However, one of the most important factors for successful liver transplantation is avoiding excessive portal hypertension and perfusion. Portal hypertension, along with excessive portal inflow after reperfusion, plays an important role in causing SFS by aggravating sinusoidal microcirculatory injury to the graft [3]. Our previous study found that patient survival rate was significantly worse when portal vein pressure was >20 mmHg in the early period after LDLT [4]. Moreover, elevated PVP was also associated with higher incidences of bacteremia, cholestasis, prolonged prothrombin time, and refractory ascites.

It is important to consider outflow and inflow modulations in cases of adult LDLT. Outflow modulation is one of the strategies for avoidance of SFS. For example, in case of a right lobe graft, we reconstruct middle hepatic vein tributaries draining segment V and VIII to avoid venous congestion. Also, the impact of elevated PVP has led to the development of methods of inflow modulation, including splenic artery ligation [4], splenectomy, [5] mesocaval shunt with downstream ligation of the superior mesenteric vein [9], and hemi-portocaval shunt [6,7]. In the present case, we constructed a mesorenal shunt with an anastomosis of the IMV and left renal vein, because PVP was still high after splenectomy.

In our institute, splenectomy is the treatment of first choice to reduce PVP. If it is not sufficient, we construct a portosystemic shunt [8]. Splenectomy is efficient method to decrease PVP. Although it is associated with an increased risk of thrombosis, most of the thrombosis just stay in splenic vein and rarely cause any clinical problems. On the other hand, it has been reported that there are some complications associated with portosystemic shunt. Steroid-resistant acute rejection or ischemic graft dysfunction could strengthen the steal of portal flow by portosystemic shunt [10,11]. For this reason, we think that splenectomy should be the treatment of first choice to reduce PVP. And if splenectomy cannot play a satisfactory role of inflow modulation, portosystemic shunt is additionally constructed as second method.

This method of mesorenal shunt offers various advantages. First, IMV as a shunt vessel with sufficient length can be obtained easily. IMV is close to the left renal vein in the retroperitoneal tissue. These allow easy anastomosis. Second, because the IMV is relatively small, shunt flow is modest, so excessive portosystemic shunt causing portal flow steal or hyperammonemia can be avoided. This method may be suitable for cases with mild portal hypertension.

This patient displayed postoperative complications of intrahepatic portal vein thrombosis. We suspected some pre-existing or newly formed thrombus in the splenic vein that came free and migrated to the peripheral branch distal to the umbilical portion. Thrombosis in the splenic vein is sometimes encountered after splenectomy. Although the cause of postoperative thrombus is not exactly known, the possible formation of portal vein thrombosis pre- and postoperatively needs to be kept in mind while performing splenectomy and when such portosystemic shunts are applied.

In conclusion, this method represents one option for attenuating portal hypertension when elevated PVP is observed following adult LDLT.

Authorship

HK: performed the study, collected and analyzed data and wrote the paper. YT: performed the study, analyzed the data and wrote the paper. YO: analyzed data and wrote the paper. FO: analyzed data. HE: analyzed data. SU: performed the study and analyzed data.

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