Objectives: The most serious, life-threatening complication after living-donor liver transplant is a hepatic arterial thrombosis. Although possible therapies for acute hepatic arterial thrombosis include revascularization to salvage the graft, or retransplant, these may be difficult to perform owing to technical aspects and donor shortages. Previously, we reported the usefulness of partial portal arterialization in such cases.

Materials and Methods: Four cases of partial portal arterialization for hepatic arterial occlusion after living-donor liver transplant were reviewed. The surgical procedure of partial portal arterialization involves making an arteriovenous shunt via a side-to-side anastomosis, using mesenteric vessels approximately 2 mm in diameter.

Results: After partial portal arterialization, hepatic arterial flow was not detected, but graft injury owing to hypoxia gradually improved in all cases. In 1 case, occlusion of the arteriovenous shunt itself and the collateral artery to the graft were identified by angiography 45 days after partial portal arterialization. In another case, massive ascites, pleural effusion, and variceal changes of the mesenteric veins owing to portal hypertension were identified, and surgical closure of the shunt was performed 152 days after partial portal arterialization. In the other 2 cases, there were no definite problems related to partial portal arterialization, but the patients died owing to other complications.

Conclusions: When hepatic arterial thrombosis occurs after living-donor liver transplant, revascularization should be performed first. However, this sometimes may be difficult, as when the arterial dissection reaches into the graft. Partial portal arterialization is an easy and effective surgical procedure. Therefore, partial portal arterialization appears to be a useful option to gain time until collateral arterial vessels develop or retransplant, even if revascularization cannot be performed.

Key words: Surgical arteriovenous shunt, Portal hypertension, Arterial reconstruction, Small intestinal mesentery, Doppler ultrasound

Introduction

The most serious, life-threatening complication after living-donor liver transplant is a hepatic arterial thrombosis. Because of the small vascular diameter, arterial reconstruction is a major technical problem in living-donor liver transplant, and some technical refinements have been reported. However, in some cases, a hepatic arterial thrombosis still occurs after living-donor liver transplant. Although possible therapies for an acute hepatic arterial thrombosis include revascularization to salvage the graft, or retransplant, these may be difficult to do owing to technical aspects in certain cases and donor shortages. Previously, we reported the usefulness of partial portal arterialization in such cases.

At our institution, there were 4 cases of partial portal arterialization for a hepatic arterial thrombosis.
after a living-donor liver transplant. This study sought to review our experience with partial portal arterialization for a hepatic arterial thrombosis, focusing on technical aspects and clinical benefits.

**Materials and Methods**

From July 1999 to April 2010, sixty-two living-donor liver transplants were done in 61 recipients, including 1 retransplant. Informed consent was obtained from each patient according to institutional guidelines. The procedures were in accordance with the Helsinki Declaration of 1975. And all protocols, experimental studies, and clinical trials involving human subjects were approved by the ethics committee of the institution before the study began.

The liver grafts were right lobe without the middle hepatic vein (n=37), right lobe with the middle hepatic vein (n=2), the left lobe (n=20), the right posterior segments (n=1), the left lateral segments (n=1), and reduced-size left lateral segments (n=1). There were 34 male and 27 female recipients (56 adult, 5 pediatric). The median age at transplant was 54 years (range, 1 to 66 y).

In the recipients, the implanted graft was reperfused after reconstructing the hepatic and portal veins. After reperfusion, hepatic artery reconstruction was done using microsurgical techniques. Whenever possible, an end-to-end vascular anastomosis was done between the recipient and graft hepatic artery using interrupted Prolene 8-0 monofilament polypropylene sutures (Ethicon, Tokyo, Japan) and an operative microscope. In the recipients, color flow Doppler ultrasound was done at least 3 times daily to determine adequate blood flow and velocities during the first 10 days after transplant. A diagnosis of hepatic arterial thrombosis was initially made by Doppler ultrasound and confirmed by dynamic computed tomography or angiography.

Partial portal arterialization was performed by constructing an arteriovenous shunt using mesenteric vascular branches with diameters of about 2 mm. A side-to-side anastomosis was performed with Prolene 8-0 continuous monofilament sutures (Ethicon), using magnification loupes or a surgical microscope as described previously (Figure 1). The size of anastomosis was about 5 mm along the longitudinal axis. After constructing partial portal arterialization, turbulent portal venous flow was identified using Doppler ultrasonography.

**Results**

Hepatic arterial thrombosis occurred in 4 recipients. The patients’ characteristics are given in Table 1. The grafts were right lobe without the middle hepatic vein (n=2), left lobe (n=1), and reduced-size left lateral segments (n=1). In all cases, partial portal arterialization was done. The reasons for selecting partial portal arterialization were dissection of the graft artery that extended into the graft parenchyma (n=3), and technical difficulty of revascularization owing to adhesions from a prior surgery (a Kasai procedure for congenital biliary atresia) and choledochojejunostomy (n=1). The serial changes of alanine aminotransferase after partial portal arterialization are shown in Table 1.

**Table 1. Characteristics of patients developing hepatic arterial thrombosis after living-donor liver transplant.**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Recipient’s age/sex</th>
<th>Recipient’s disease</th>
<th>MELD/PELD score</th>
<th>GRWR</th>
<th>Graft</th>
<th>Occurrence of HAT (POD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1/M</td>
<td>Liver GVHD</td>
<td>31</td>
<td>4.75</td>
<td>Reduced-sized lateral segment</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>19/M</td>
<td>BA post Kasai</td>
<td>13</td>
<td>0.55</td>
<td>Left Lobe</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>27/F</td>
<td>BA post Kasai</td>
<td>7</td>
<td>1.31</td>
<td>Right lobe</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>57/F</td>
<td>LC(C)</td>
<td>25</td>
<td>1.24</td>
<td>Right lobe</td>
<td>9</td>
</tr>
</tbody>
</table>

Abbreviations: BA, biliary atresia; BMT, bone marrow transplant; GRWR, graft recipient weight ratio; GVHD, graft-versus-host disease; HAT, hepatic arterial thrombosis; LC(C), liver cirrhosis due to hepatitis C; MELD, model for end-stage liver disease; PELD, pediatric end-stage liver disease; POD, postoperative day.
arterialization are shown in Figure 2. Except in case 2, which was complicated by a bowel perforation 6 days after this procedure, liver enzymes gradually decreased after partial portal arterialization.

Of the 4 partial portal arterialization recipients, 2 died. One recipient (case 1), who was transplanted with reduced-size left lateral segments, died from lung graft-versus-host disease complicated by a hematologic disorder 12 days after partial portal arterialization. The other recipient (case 2) was transplanted with a left lobe, and died owing to peritonitis from a small intestinal perforation and severe acute cellular rejection 43 days after partial portal arterialization. In both cases, graft function was maintained sufficiently, and no adverse effects or complications related to partial portal arterialization (such as portal hypertensive symptoms or graft abscess) were observed. Furthermore, turbulent portal venous flow was confirmed using Doppler ultrasonography as evidence of arteriovenous shunt patency (Figure 3).

The other 2 recipients survived after partial portal arterialization without retransplant. Both cases had a right lobe graft. In 1 case (case 3), the arteriovenous shunt occluded spontaneously, and hepatopetal collateral arterial flow from the inferior pancreaticoduodenal artery developed. These findings were confirmed by angiography 45 days after partial portal arterialization. In the other case (case 4), variceal changes of the mesenteric veins, massive ascites, and pleural effusion owing to portal hypertension because of partial portal arterialization appeared about 3 months after surgery (Figure 4). In this case, hepatopetal collateral arterial flow from the right subphrenic artery was confirmed by angiography 124 days after partial portal arterialization. Then, 152 days after partial portal arterialization, the arteriovenous shunt was surgically occluded because the patient’s portal hypertensive symptoms resisted medication.

Discussion

The success of liver transplant is usually dependent on arterial and portal venous perfusion. Hepatic arterial reconstruction is one of the major technical problems because of the small vascular diameter, especially in living-donor liver transplant. Hepatic arterial thrombosis is still the most serious, life-threatening complication. Total interruption of the graft arterial flow may cause serious complications,
such as disruption of biliary reconstruction or a liver abscess. Although the most desirable therapy for acute hepatic arterial thrombosis may be revascularization to salvage the graft or retransplant, these are difficult to perform; the reasons for technical difficulty include graft arterial dissection into the graft parenchyma in our series. Further (particularly in Japan), retransplant is difficult owing to donor shortages. In such situations, partial or total portal venous arterialization is done to obtain an arterial blood supply to rescue the graft. In 2001, Cavallari and associates reported portal venous arterialization for hepatic arterial thrombosis after a deceased-donor liver transplant.9

Our surgical method of creating a partial portal arterialization uses mesenteric vessels. This procedure is simple and safe because the operative field is separated from the hepatic hilum. In surgical procedures around the hepatic hilum after a living-donor liver transplant, there is difficulty in maintaining the operative field because of vascular or biliary reconstructions. The advantage of our procedure is important not only at the time of creating partial portal arterialization, but also, closing the shunt in cases with portal hypertensive symptoms after confirmation of collateral arterial blood flow. Furthermore, arteriovenous anastomosis is easy because the orifice of the anastomosis is large enough when done in a side-to-side fashion. In addition, the operative field of the mesentery in our method is flatter and shallower than the hepatic hilum. Finally, because the operative field is separated from the graft and hepatic hilum, it is not affected by the respiratory movement of the diaphragm.

From our perspective, an important role of partial portal arterialization is a bridge therapy to gain time until collateral arterial flow into the graft develops in cases where arterial revascularization is impossible or until retransplant. Actually, in the surviving cases, collateral arterial flow into the grafts was confirmed by angiography. Thus, it is important to recognize when collateral arterial vessels have developed sufficiently to maintain graft oxygenation. In a prior study, hepatopetal arterial collaterals developed within 1 month, as confirmed by angiography in the cases of interrupted hepatic arterial flow.10 Thus, it appears that arteriovenous shunt patency is needed for at least 1 month after partial portal arterialization.

It has been shown that portal arterialization carries a risk of delayed portal hypertension.11, 12 In our previous experimental study, the increase in portal pressure after construction of partial portal arterialization was slight, because the arterial flow into the portal circulation was not excessive. In addition, the mesenteric arteriovenous shunt was effective in preventing changes in hepatic function and morphology.13, 14 In our series, partial portal arterialization was performed with the mesenteric vascular branches. This was not followed by any clinically apparent portal hypertension during the short postoperative time of approximately 2 months. However, in 1 case, apparent portal hypertensive symptoms developed approximately 3 months after surgery. Because the purpose of partial portal arterialization is bridging therapy to maintain graft oxygenation until collateral arteries develop to supply sufficient oxygen or until retransplant, patency of shunting is not essential after confirmation of collateral artery development or retransplant. Furthermore, this case suggests the possibility that partial portal arterialization may have detrimental long-term effects. Thus, shunt occlusion, whether spontaneous or artificial, is important.

When hepatic arterial thrombosis occurs after a living-donor liver transplant, revascularization should be performed first. However, this may be difficult, such as when the arterial dissection reaches into the graft. Partial portal arterialization is an easy and effective surgical procedure even in such cases. Therefore, we suggest that partial portal arterialization can be a useful option to gain time until collateral arterial vessels development or retransplant, even if revascularization cannot be performed. Further clinical studies are necessary to evaluate portal hypertension adverse effects and the timing of shunt occlusion.

References


