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ORIGINAL ARTICLE

The outcome of living donor liver transplantation with prior spontaneous large portasystemic shunts

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Summary

We investigated the outcome of living donor liver transplantation (LDLT) with prior spontaneous large portasystemic shunts. Thirty-three patients of 155 patients (21.2%) undergoing LDLT had spontaneous large portasystemic shunts. Portal venous hemodynamics, surgical procedures for shunts, and morbidity and mortality rates were investigated in three types of shunts: splenorenal shunt (SRS group; $n = 11$), shunt derived from coronary vein (CVS group; $n = 6$) and umbilical vein shunt (UVS group; $n = 15$). The two groups of patients (SRS/CVS) received prophylactic surgical repair of shunts during LDLT except for one patient in the SRS group. The flow direction of main portal vein and grade of steal of superior mesenteric vein flow by shunt were significantly different among three groups. No significant differences were observed among three groups in operative parameters, hospitalization and morbidity except for postoperative portal complication. There was no significant difference in the actuarial survival rate among three groups of SRS, CVS and UVS (81.8% vs. 83.3% vs. 86.6% at 1 year respectively). In the SRS group, two patients had postoperative steal of graft portal venous flow by residual SRS that needed further treatment. The outcome of LDLT with prior spontaneous large portasystemic shunts is satisfactory, despite the complexity of the transplant procedures.

Introduction

Several studies have discussed the outcome of patients with spontaneous or surgical portasystemic shunts in deceased donor liver transplantation (DDLT) [1–12]. Central portasystemic shunts, such as surgical portacaval shunt, should be closed immediately after graft implantation to avoid diversion of portal venous flow (PVF) from the graft [2–9]. With regard to splenorenal shunts (SRS), controversy exists as to whether the occlusion of these shunts during the transplant procedure is warranted [1–12]. In fact, some of the spontaneous or surgical SRS virtually disappear following removal of the cirrhotic liver and transplantation of the liver graft. However, some post-transplant conditions, such as acute rejection and severe ischaemic damage, might cause increased intrahepatic vascular resistance that enhances post-transplant development of preserved or residual SRS. In these circumstances, the graft PVF could be easily stolen by the developed SRS, leading to serious graft dysfunction.

Living donor liver transplantation (LDLT) is an established treatment modality for end-stage liver diseases and serves to alleviate the shortage of cadaveric donor organs. There have been noticeable improvements in recipient outcome in LDLT [13–17]. However, there is little information about the outcome of LDLT with prior spontaneous portasystemic shunts. It has been generally accepted that adequate PVF is essential for postoperative hepatic regeneration after hepatectomy and partial liver transplantation [18–20]. Preserved portasystemic shunt is reported to cause excessive decrease in graft PVF, leading

to graft dysfunction in these conditions, such as acute rejection and severe ischaemic damage [3,6,10–12].

We proposed surgical prophylactic management of spontaneous large portasystemic shunts during the LDLT procedure. In this study, we investigated the outcome of LDLT with spontaneous large portasystemic shunts, and also determined the results of surgical prophylactic management of these portasystemic shunts during the transplant procedure.

Patients and methods

From August 1986 to September 2006, a total of 155 LDLT procedures were performed at our department on 155 patients with end-stage liver disease. Indications for LDLT were as follows: fulminant hepatic failure in 21, cirrhosis due to hepatitis C in 33, cirrhosis due to hepatitis B in 26, cryptogenic cirrhosis in 11, primary biliary cirrhosis in 19, primary sclerosing cirrhosis in eight, alcoholic cirrhosis in 10, biliary atresia in 14, Wilson's disease in six, and others in seven. None of the patients had prior surgical portasystemic shunt. Doppler ultrasonography (US), computed tomography (CT) and/or transfemoral angiography were arranged before LDLT in 127 patients with chronic end-stage liver disease to evaluate the anatomy and hemodynamics of portal venous and hepatic arterial circulation. We assessed the patency and flow direction of the main portal vein (PV) and the splenic vein (SPV), and detected the type, size and PVF of the preoperative spontaneous portasystemic shunts. A large portasystemic shunt was defined as a shunt with a diameter of more than 10 mm and a PVF of more than 400 ml/min. Based on these evaluations, 33 patients had spontaneous large portasystemic shunt, as assessed by Doppler US and enhanced CT. We categorized these 33 patients into four types of large portasystemic shunts : 11 had SRS, six had shunts derived from coronary veins inflowing directly to systemic venous circulation (CVS), 15 had shunts from umbilical vein (UVS), and one had a shunt from an inferior mesenteric vein. In four patients who had two types of large portasystemic shunts concurrently, we selected the more prominent shunt for categorization. Of these shunts, we retrospectively investigated and compared portal venous hemodynamics, surgical procedures for shunts, and morbidity and mortality after LDLT in three types of spontaneous large portasystemic shunts: SRS (n = 11), CVS (n = 6) and UVS (n = 15).

The three patient groups of SRS, CVS and UVS were comparable in gender, age, child classification and the Model for End-Stage Liver Disease (MELD) score. With regard to grafts, no significant differences were observed among the groups in type of grafts, graft weight and graft-to-recipient body weight ratio (GRWR) (Table 1).

Table 1. Patient and graft characteristics among three groups.

	SRS group	CVS group	UVS group	*P*-value
Patients				
No.	11	6	15	
Male/female	7/4	5/1	6/9	NS
Age (years)	50 ± 2.8	48 ± 5.6	49 ± 3.8	NS
Postnecrotic/cholestatic	10/1	5/1	10/5	NS
Child B/C	3/8	2/4	5/10	NS
MELD score	18.2 ± 1.5	15.3 ± 0.8	16.9 ± 1.1	NS
Grafts				
Right/left lobe	7/4	5/1	7/8	NS
Graft weight (g)	578 ± 35	566 ± 49	565 ± 24	NS
GRWR	0.90 ± 0.07	0.83 ± 0.07	1.02 ± 0.05	NS
GRWR <0.8/>0.8	4/7	2/4	2/13	NS

SRS, splenorenal shunt; CVS, coronary vein shunt; UVS, umbilical vein shunt; MELD, Model for End-Stage Liver Disease; GRWR: graft-to-recipient body weight ratio.

Small-for-size (SFS) grafts with GRWR under 0.8% were present in four patients of the SRS group, in two patients of the CVS group and in two patients of the UVS group. Blood flow direction of the main PV and proximal SPV, patency of main PV and the grade of steal of superior mesenteric vein (SMV) blood flow by portasystemic shunts were assessed preoperatively using Doppler US, CT and/or transfemoral angiography. The results of portal venous hemodynamics were compared among the three groups.

The recipient operation was performed in a piggy-back fashion. The UVS was dissected at the beginning of the hepatectomy in all patients of the UVS group. In eight patients of the UVS group, a passive venovenous bypass between the umbilical vein and the left axillary vein was used. After complete graft revascularization, management of portasystemic shunts (SRS and CVS) was performed as documented in Table 4. In principle, we dissected the SRS at the inflow site to left renal vein with and without splenectomy in the SRS group. In the CVS group, the coronary vein was ligated at the root. In the two patients with completely stolen SMV blood flow by the spontaneous portasystemic shunt, we diverted the SMV and SPV blood flow by ligating SPV at the root. The graft PVF was measured by Doppler US both after complete graft revascularization and after occlusion of spontaneous large portasystemic shunt.

Immunosuppression was achieved by tacrolimus or cyclosporine and low-dose steroids. Acute rejection episodes were treated with steroid boluses and, when unresponsive, with administration of OKT3 monoclonal antibodies for 10–14 days. PV flow was followed by Doppler US and CT after transplantation. Postoperative angiographic studies were not performed routinely but only when vascular complications were suspected.

Data were presented as mean ± standard error of the mean (SEM). Differences in qualitative variables were assessed using the Fisher exact or chi-squared test, while differences in quantitative variables were analysed using the Mann–Whitney test. Cumulative probability curves of survival were calculated using Kaplan–Meier methods, and differences between these curves were compared using the Wilcoxon signed-rank test. *P*-values <0.05 were considered statistically significant. All statistical analyses were performed using the SPSS II statistical software package (SPSS Inc., Tokyo, Japan).

Results

Preoperative blood flow direction and patency of main PV

Preoperative blood flow direction of main PV assessed by Doppler US was hepatopetal in 10 and hepatofugal in one of 11 patients of the SRS group (Table 2). In the UVS group, there was no patient with hepatofugal blood flow of the main PV. In contrast, four of six patients of the CVS group showed hepatofugal blood flow of the main PV. Thus, the blood flow direction was significantly different between the CVS group and the other two groups. Narrowing of the main PV was observed in two of 11 patients of the SRS group and in two of six patients of the CVS group. In contrast, there was no patient with the narrowed main PV in the UVS group.

Grade of preoperative steal of SMV blood flow by shunt

Preoperative blood flow direction of proximal SPV assessed by Doppler US was hepatopetal in one and hepatofugal in five of six patients of the CVS group (Table 3). In contrast, 14 of 15 patients of the UVS group showed hepatopetal blood flow of the proximal SPV. The steal of SMV blood flow by shunt was significantly higher in the CVS group than that in the SRS and CVS groups. The grade of steal of SMV blood flow in the CVS group was partial in two and complete in four of six patients.

Surgical procedures for shunts and PV reconstruction

Table 4 summarizes the surgical procedures for shunts and PV reconstruction. The SRS were treated in 10 of 11 patients (91%) in the SRS group (in the early stages of our liver transplant program, LDLT was performed in one patient without repair of such shunt). SRS were ligated at the inflow site to the left renal vein without splenectomy in seven, and were transected by splenectomy in two. The SMV and SPV blood flow were diverted by ligation at the root of SPV in one patient, in whom SMV blood flow had been completely stolen by SRS preoperatively. In the CVS group, CVS were ligated at the root in five, and diversion of SMV and SPV blood flow by the ligation at the root of SPV was performed in one. In all patients of the UVS group, the UVS was dissected

Table 2. Preoperative blood flow direction and patency of main portal vein (PV).

	Flow direction of main PV			Patency of main PV			
	Hepatopetal	Hepatofugal		Patent	Narrowed		Total
Shunt type							
SRS	10	1	$P < 0.05$	9	2	$P < 0.05$	11
CVS	2	4		4	2		6
UVS	15	0	$P < 0.01$	15	0		15
Total	27	5		28	4		

SRS, splenorenal shunt; CVS, coronary vein shunt; UVS, umbilical vein shunt.

Table 3. Preoperative blood flow direction of proximal SPV and the grade of steal of SMV blood flow by shunt.

	Flow direction of proximal SPV			Steal of SMV flow by shunt				
	Hepatopetal	Hepatofugal		None	Partial	Complete		Total
Shunt type								
SRS	7	4		6	4	1	$P < 0.01$	11
CVS	1	5	$P < 0.01$	0	2	4		6
UVS	14	1		0	14	1	$P < 0.05$	15
Total	22	10		6	20	6		

SPV, splenic vein; SMV, superior mesenteric vein; SRS, splenorenal shunt; CVS, coronary vein shunt; UVS, umbilical vein shunt

Table 4. Surgical procedures for shunts and portal vein (PV) reconstruction.

Procedures for shunts	PV reconstruction
SRS (n = 11)	
Ligation of shunts at the inflow site to LRV (7)	Native PV (9)
Diversion of SMV and SPV flow (1)	Interposed vein graft (2)
Splenectomy (2)	
No procedure (1)	
CVS (n = 6)	
Ligation of coronary vein at the root (5)	Native PV (3)
	Native PV after thrombectomy (1)
Diversion of SMV and SPV flow (1)	Interposed vein graft (2)
UVS (n = 15)	
Ligation of umbilical vein shunt (15)	Native PV (15)

SRS, splenorenal shunt; CVS, coronary vein shunt; UVS, umbilical vein shunt; LRV, left renal vein; SPV, splenic vein; SMV, superior mesenteric vein.

at the beginning of hepatectomy. In eight patients of the UVS group, cannula (Anthron bypass tube; Toray Medial Inc, Tokyo, Japan) were inserted from UVS and the passive bypass between umbilical vein and left axillary vein was performed during hepatectomy for the decompression of the portal circulation. In the SRS group, the portal inflow was reconstructed by direct anastomosis to the native PV in nine, and with an interposed vein graft to the spleno-portal junction in two. In the CVS group, PV reconstruction with native PV was performed in three, native PV after thrombectomy in one, and an interposed vein graft in two. In all patients of the UVS group, PV reconstruction with native PV was performed.

Morbidity and mortality after LDLT

There were no significant differences among the three groups in operative time, blood loss, intensive care unit (ICU) stay, hospital stay and postoperative mortality (Table 5). The mean PVF of grafts before the occlusion of portasystemic shunts was 621 ± 47 ml/min in the SRS group, and 739 ± 116 ml/min in the CVS group. The mean PVF of grafts after occlusion of portasystemic shunts increased significantly to 1278 ± 134 ml/min in the SRS group and 1661 ± 159 ml/min in the CVS group, compared with those before shunt occlusion. With regard to postoperative complications, the number of portal complications in the CVS group was significantly higher than those in the SRS and UVS groups. In the CVS group, PV anastomotic stricture was detected in one patient and PV thrombosis derived from the dissected

Table 5. Morbidity and mortality after living donor liver transplantation among three groups.

	SRS group (n = 11)	CVS group (n = 6)	UVS group (n = 15)	*P value*
Operation				
Operative time (min)	595 ± 23	712 ± 65	596 ± 36	NS
Blood loss (g)	6920 ± 1440	13820 ± 8460	5867 ± 1610	NS
Graft PVF (ml/min)				
Shunt open	621 ± 47	739 ± 116		NS
Shunt occluded	1278 ± 134 ($P < 0.01$)	1661 ± 159 ($P < 0.01$)		NS
Hospitalization (day)				
ICU stay	13 ± 2.9	16 ± 8.8	9.9 ± 1.0	NS
Hospital stay	61 ± 8.9	77 ± 17	65 ± 6.9	NS
Postoperative complication				
Intra-abdominal haemorrhage	2/11	2/6	1/15	NS
Arterial complications	0/11	0/6	0/15	NS
Portal complications	0/11	2/6	0/15	<0.05*
Biliary complications	4/11	2/6	2/15	NS
Intractable ascites	0/11	0/6	2/15	NS
Prolonged hyperbilirubinemia	0/11	1/6	2/15	NS
Steal of graft PVF by shunt	2/11	0/6	0/15	NS
Postoperative mortality	1/11	1/6	2/15	NS
1-year patient survival rate (%)	81.8	83.3	86.6	NS

SRS, splenorenal shunt; CVS, coronary vein shunt; UVS, umbilical vein shunt; PVF, portal venous flow.
*0.048 (CVS versus SRS); 0.022 (CVS versus UVS).

stump of coronary vein in one patient, while there was no portal complication in the SRS and CVS groups. Graft PVF steal was detected postoperatively in the preserved or remaining SRS in two of 11 patients in the SRS group. In contrast, none of the patients of the CVS group had postoperative steal of graft PVF by the residual portasystemic shunt, although the difference between the SRS and CVS groups was not significant. Furthermore, none of the patients in the SRS and CVS groups developed postoperative intractable ascites. Prolonged hyperbilirubinemia was observed in one patient with postoperative PV thrombosis in the CVS group and in two patients in the UVS group.

Survival rates of patients with and without spontaneous large portasystemic shunts

There was no significant difference in the actuarial survival rate among patients with SRS/CVS/UVS ($n = 32$) and those without these portasystemic shunts ($n = 122$) (84.3% vs. 88.2% at 1 year respectively; $P = 0.083$). Because re-transplantation has not been performed in our series, the graft survival rates were the same as the actuarial survival rates. Furthermore, there was no significant difference in the actuarial survival rate among the three groups of SRS, CVS and UVS (81.8% vs. 83.3% vs. 86.6% at 1 year respectively) (Table 5).

Postoperative steal of graft PVF by spontaneous portasystemic shunt

Two patients developed postoperative steal of graft PVF by spontaneous SRS (Table 6). In one patient in whom the SRS was not occluded during the transplant procedure, the graft PVF was completely stolen by the preserved SRS on postoperative day (POD) 9 because of steroid-resistant acute rejection. Although SRS was occluded during an emergency laparotomy on POD9 together with commencement of infusion of OKT3, liver graft function deteriorated rapidly and the patient died on POD39. In the other patient who underwent occlusion of the main SRS during the transplant procedure, graft PVF was completely stolen by the residual SRS on POD2 probably because of severe ischaemic graft injury. We diverted SMV and SPV blood flow by ligation at the root of SPV on POD2 to prevent the steal of graft PVF, leading to recovery of liver graft function.

Discussion

There is a general agreement that central portasystemic shunts, such as surgical portacaval shunt and spontaneous shunt derived from the main PV inflowing directly to systemic venous circulation, should be closed to avoid diversion of PVF from the graft [2–9]. However, the management of prior spontaneous or surgical SRS is a controversial issue [1–12]. In DDLT, postoperative analysis have identified potential graft PVF steal by the preserved portasystemic shunt, leading to graft dysfunction and loss [6,10,11]. The presence of spontaneous portasystemic shunts after DDLT was reported to have a detrimental effect on graft perfusion when portal hypertension reappears in the early postoperative period, as during rejection episodes [10]. Thus, several authors have reported that both surgical and spontaneous large portasystemic shunts should be occluded during the transplant procedure to achieve a similar patient and graft survival as in patients without such shunts [2,3,5,6,10]. Because a sufficient restoration of the liver vascular bed could not be achieved in the early postoperative period in adult LDLT, post-transplant portal hypertension caused by acute rejection or severe ischaemic damage might appear more strongly in LDLT than in DDLT. The possibility that the preserved portasystemic shunt steals the post-transplant graft PVF might be more prone to occur in LDLT than in DDLT. Therefore, we proposed surgical prophylactic management of spontaneous large portasystemic shunts, especially in the SRS group, during the LDLT procedure.

In this study, the actuarial survival rate of patients with prior spontaneous large portasystemic shunt was similar to that with those without these portasystemic shunts, despite the complexity of the operative procedure for repair of these shunts. In addition, there were no significant differences among the three types of spontaneous large portasystemic shunt in operative parameter, hospitalization and postoperative morbidity except for

Table 6. Postoperative steal of graft portal venous flow by spontaneous portasystemic shunt.

Patients	Shunt type	Procedure for shunt	Time after LDLT (POD)	Cause	Management	Prognosis
1	SRS	No procedure	9	Rejection	Ligation at inflow site to LRV OKT3	Died (POD39)
2	SRS	Ligation at inflow site to LRV	2	Ischaemic graft injury	Diversion of SMV and SPV flow	Alive

LDLT, living donor liver transplantation; POD, postoperative day; SRS, splenorenal shunt; LRV, left renal vein; SMV, superior mesenteric vein; SPV, splenic vein.

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postoperative portal complication. The actuarial survival rate was similar in the SRS, CVS and UVS groups, and there was no postoperative mortality associated with spontaneous portasystemic shunt except for one patient, whose SRS was not surgically treated during LDLT in the early period of our liver transplant programme.

Inter-individual differences were observed with regard to portal venous hemodynamics, including blood flow direction of main PV and the degree of steal of SMV blood flow by portasystemic shunts. In this regard, portasystemic shunts enhance the likelihood of PV phlebosclerosis, which makes subsequent vascular anastomosis difficult. Thus, pretransplant Doppler US, CT and/or angiography are important to assess portal venous hemodynamics. In our study, both hepatofugal blood flow of main PV and the grade of steal of SMV blood flow by shunt were significantly higher in the CVS group than in the SRS and UVS groups. In four patients, it was necessary to perform PV reconstruction with an interposed vein graft for the narrowed main PV, which had been assessed preoperatively. Such pretransplant assessment of portal venous hemodynamics by Doppler US, CT and/or angiography was useful for the selection of PV reconstruction, including the interposition of vein graft.

Previous studies indicated that the size of the graft liver correlates with clinical outcome [21–24]. Kiuchi *et al.* [22] reported that the graft survival rate in patients with GRWR of <0.8% was significantly worse than with larger GRWR. The clinical manifestations, referred to as SFS syndrome, consist of delayed synthetic function, prolonged hyperbilirubinemia and intractable ascites, leading to higher mortality. Although various recipient and donor factors are involved in the development of SFS syndrome, severe portal hypertension and excessive PVF have been suggested to be important mechanisms of SFS graft injury [25–27]. Accordingly, several surgeons reported that SFS grafts could be successfully treated by reduction of portal venous pressure with the construction of surgical portasystemic shunt or temporary transjugular intrahepatic portasystemic shunt [28–31]. On the other hand, Yagi *et al.* [20] investigated the optimal portal venous circulation for liver graft function after LDLT in adult recipients. They tried to maintain portal venous pressure below 20 mmHg and keep graft PVF above 800 ml/min by selecting occlusion or preservation of prior portasystemic shunt in their 28 patients, resulting in a better liver graft function. In our study, the mean graft PVF before the occlusion of portasystemic shunt was below 800 ml/min in both the SRS and CVS groups, and increased significantly after surgical repair of these shunts. Although portal venous pressure was not measured intraoperatively in our study, neither intractable ascites nor prolonged hyperbilirubinemia were seen in the SRS and CVS groups except in one patient with PV thrombosis while six patients, with GRWR of less than 0.8%, were included in the SRS and CVS groups.

We experienced graft dysfunction because of postoperative steal of graft PVF by the residual SRS in one patient of the SRS group. In this patient, a complete steal of graft PVF by the residual SRS was noted on POD2 in spite of occlusion of the main SRS during the transplant procedure. Although ischaemic graft injury in this patient could strengthen the steal of graft PVF by the residual SRS, ligation of the SRS at the inflow site to left renal vein without splenectomy might have been insufficient in this patient. For patients with several routes of SRS, transection of the shunt by splenectomy is recommended, instead of ligation of the shunt at the inflow site to the left renal vein without splenectomy.

In conclusion, the outcome of LDLT with prior spontaneous large portasystemic shunts is satisfactory, despite the complexity of the transplant procedures for repair of these shunts. However, postoperative steal of graft PVF by residual SRS is still a potential cause of graft dysfunction. With regard to patients with SFS grafts, further investigations are necessary to assess the optimal portal venous circulation for SFS grafts and to determine the precise management strategy of portasystemic shunt.

Authorship

HS performed the main role in designing the study, collecting and analyzing the data and writing the article. HM, TI and DS contributed to collect the data and analyze the results. HM, SS and YU assessed the hemodynamics of portal venous circulation by Doppler US and contributed in analyzing the data. TY and NT had a significant role in the design of the study, analyzing the data and in supervising the first author.

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