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ORIGINAL ARTICLE

Hepatic Venous Outflow Obstruction in Pediatric Living Donor Liver Transplantation Using Left-Sided Lobe Grafts: Kyoto University Experience

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The goals of this study were to evaluate the incidence of hepatic venous outflow obstruction (HVOO) in pediatric patients after living donor liver transplantation (LDLT) using left-sided lobe grafts and to assess the therapeutic modalities used for the treatment of this complication at a single center. Four hundred thirteen primary LDLT procedures were performed with left-sided lobe grafts between 1996 and 2006. All transplants identified with HVOO from a cohort of 380 grafts with survival greater than 90 days were evaluated with respect to the patient demographics, therapeutic intervention, recurrence, and outcome. Seventeen cases (4.5%) were identified with HVOO. Eight patients experienced recurrence after the initial balloon venoplasty. Two patients finally required stent placement after they experienced recurrence shortly after the initial balloon venoplasty. A univariate analysis revealed that a smaller recipient-to-donor body weight ratio and the use of reduced grafts were statistically significant risk factors. The cases with grafts with multiple hepatic veins had a higher incidence of HVOO. In conclusion, the necessity of repeated balloon venoplasty and stent placement was related to poor graft survival. Therefore, the prevention of HVOO should be a high priority in LDLT. When grafts with multiple hepatic veins and/or significant donor-recipient size mismatching are encountered, the use of a patch graft is recommended. Stent placement should be carefully considered because of the absence of data on the long-term patency of stents and stent-related complications. New stenting devices, such as drug-eluting and biodegradable stents, may be promising for the management of HVOO. ***Liver Transpl 16:1207-1214, 2010.*** © **2010 AASLD.**

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Techniques for reduced size liver transplantation, split liver transplantation, and living donor liver transplantation (LDLT) have been developed to address the shortage of organs and the size discrepancy between donors and recipients.[1,2] These types of liver transplantation are technically demanding because of the use of short vascular pedicles, which are more likely to cause postoperative vascular complications. Hepatic venous outflow obstruction (HVOO) is a rare vascular complication; however, it may lead to graft dysfunction without appropriate management.[3,4] The causes of HVOO include technical problems, subsequent fibrosis with inflammatory processes, and compression or twisting of the anastomosis caused by

Abbreviations: BA, biliary atresia; GRWR, graft-to-recipient weight ratio; HVOO, hepatic venous outflow obstruction; IVC, inferior vena cava; LDLT, living donor liver transplantation; LL, left lobe; LLS, left lateral segment; r-LLS, reduced left lateral segment; SD, standard deviation; WD, Wilson's disease.

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graft regeneration.[5] Hepatic vein reconstruction is one of the most crucial factors in recipient surgery, and the creation of a wide outflow orifice is a key point for preventing HVOO.[6] The triangulation technique recommended by Emond et al.[7] has led to a decrease in the incidence of this complication. A previous report documented the early experience with HVOO and introduced a new technique for hepatic vein reconstruction involving an incision on the inferior vena cava (IVC).[6] This modified technique for hepatic vein reconstruction has been applied for more than 10 years in our hospital, and it is important to evaluate how this technique has contributed to the prevention of HVOO.

The management of HVOO is an important issue in LDLT. Balloon venoplasty is an effective treatment, but some patients require multiple sessions of balloon venoplasty, and stent placement is eventually required for most patients.[8,9] The indications for and appropriate timing of stent placement are still unclear, and the long-term outcome for patients with HVOO has not been examined sufficiently.

Various innovations have been achieved in LDLT, such as hepatic vein reconstruction, graft-type selection for smaller children, and HVOO management. In this study, therefore, we review pediatric LDLT using left-sided lobe grafts and evaluate the incidence of HVOO and the outcomes of the therapeutic modalities used in the treatment of this complication.

TABLE 1. Patient Characteristics

Characteristic	Value
Gender, male/female	157/223
Age, median (range)	1.5 years (29 days to 16.6 years)
Body weight, median (range)	9.2 kg (2.8-62.4 kg)
Original disease, n (%)	
Cholestatic liver diseases	290 (76.3)
Metabolic liver diseases	28 (7.4)
Fulminant hepatic failure	22 (5.8)
Hepatic malignancy	21 (5.5)
Liver cirrhosis	11 (2.9)
Congenital absence of the portal vein	4 (1.0)
Primary sclerosing cholangitis	3 (0.8)
Autoimmune hepatitis	1 (0.3)
Graft type, n (%)	
r-LLS	37 (9.7)
LLS	298 (78.4)
LL	45 (11.8)
Blood-type combination, n (%)	
Identical	247 (65.0)
Compatible	77 (20.3)
Incompatible	56 (14.7)
GRWR, mean ± SD (range)	2.59% ± 1.18% (0.61%-6.83%)

PATIENTS AND METHODS

Four hundred thirteen primary LDLT procedures with left-sided lobe grafts were performed in children (younger than 18 years old) at Kyoto University Hospital between January 1996 and December 2006. Thirty-three cases were excluded from this study because of death within 90 days after LDLT. The records of 380 cases (157 males and 223 females) were retrospectively reviewed for patient demographics, including age, gender, original disease, and graft type, at the end of March 2009 (Table 1). The median follow-up period was 7.9 years (range = 91 days to 13.2 years). The age at LDLT ranged from 29 days to 16.6 years (median = 1.5 years). The recipient body weight at LDLT ranged from 2.8 to 62.4 kg (median = 9.2 kg). The original diseases included cholestatic liver diseases (n = 290), metabolic liver diseases (n = 28), fulminant hepatic failure (n = 22), hepatic malignancy (n = 21), liver cirrhosis (n = 11), congenital absence of the portal vein (n = 4), primary sclerosing cholangitis (n = 3), and autoimmune hepatitis (n = 1).

The recipient operation was performed in a piggyback fashion without venovenous bypass, as described elsewhere.[1] The anastomotic orifice was prepared according to the number of graft hepatic veins, the shape of the graft, and the anatomy of the recipient IVC.[6,10] The standard type of recipient orifice on the IVC is shown in the first column of Fig. 1. A new wide orifice on the recipient IVC connecting all 3 hepatic veins (type A) was created. One orifice connecting the left and middle hepatic veins by an incision in the IVC on the lower caudal side (type B) was created for larger pediatric recipients. A flat, long graft was difficult to place in the left upper quadrant because of a lack of space. The anastomotic orifice of the recipient IVC was prepared with the recipient right hepatic vein (type C). The diameter of the new orifice was adjusted to that of the graft hepatic vein by an incision in the IVC wall or by sutures from the left corner of the hepatic vein, if necessary. A reduced left lateral segment (r-LLS) allowed the graft to be placed in the left upper quadrant, and a type A hepatic vein reconstruction was used for the recent cases. The separated graft hepatic veins were made into 1 orifice by a back-table procedure if the distance between the separated hepatic veins was short. The graft hepatic veins were anastomosed separately if they were far apart. The anastomosis was made in an end-to-side fashion with running sutures of 5-0 or 6-0 polypropylene monofilament. No conduits or patch grafts were used for hepatic vein reconstruction. Patients received a left lateral segment (LLS; n = 298, 78.4%), left lobe (LL; n = 45, 11.8%), or r-LLS, which consisted of monosegments and reduced monosegments (n = 37, 9.7%). The graft-to-recipient weight ratio (GRWR) ranged from 0.61% to 6.83%, and the mean was 2.59%.

The immunosuppression consisted of tacrolimus and low-dose steroids. The ABO blood-type combination for the donor and recipient included 56 incompatible cases (14.7%), and the protocol to prevent antibody-mediated rejection related to anti-ABO blood

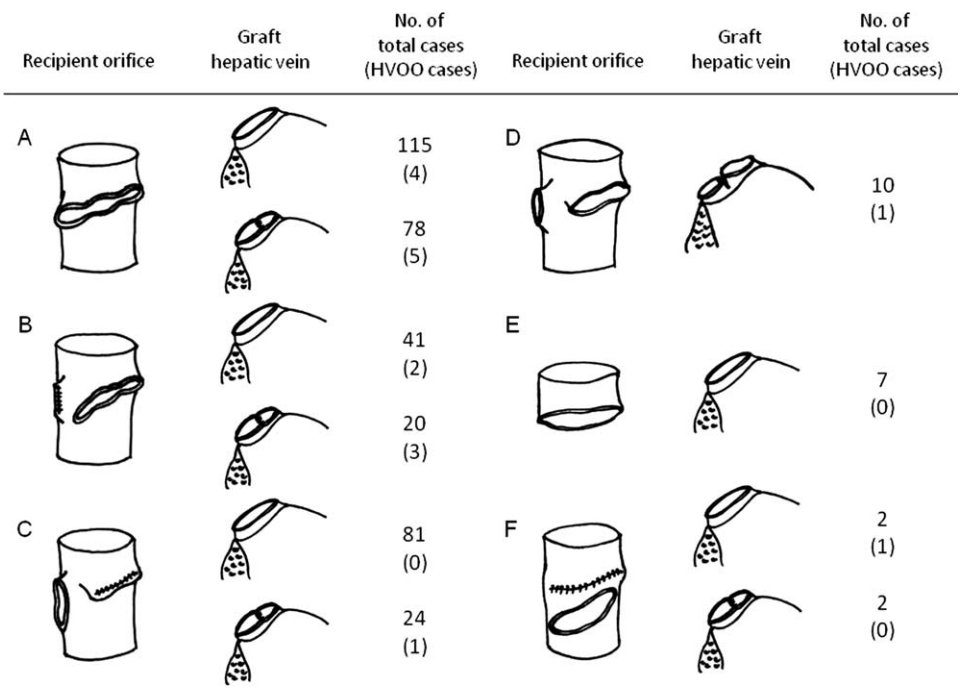

Figure 1. Combinations of the recipient orifice on the IVC and the graft hepatic veins. The number of HVOOs is also indicated. The standard type of recipient orifice on the IVC is shown in the first column. (A) A new wide orifice on the recipient IVC connecting all 3 hepatic veins was created. (B) One orifice connecting the left and middle hepatic veins was created by an incision in the IVC on the lower caudal side. (C) The anastomotic orifice of the recipient IVC was prepared with the recipient right hepatic vein. The special type of hepatic vein reconstruction is shown in the second column. (D) Two anastomoses were made individually between the graft hepatic veins and the recipient hepatic vein stumps. (E) The direct anastomosis to the IVC or the right atrium was performed. (F) A new orifice on the IVC was created.

antigens depended on factors such as the age of the recipient and the LDLT era, as described elsewhere in detail.[11]

The hepatic venous flow was followed by Doppler ultrasound after LDLT every day for the first week, once to twice a week during the rest of the hospitalization, and at least once every 3 months after discharge. HVOO was indicated by intractable ascites, abnormal hepatic venous flow patterns, histological findings suggesting HVOO, or liver dysfunction. Doppler ultrasound findings suggesting HVOO were the disappearance of the pulsatile hepatic venous flow and the flattening of the hepatic venous wave.[6] Liver biopsy findings suggesting HVOO included congestion, hemorrhaging, and necrosis around the central veins.[12] Common abnormal laboratory findings included hypoalbuminemia and hyperbilirubinemia. A hepatic vein venogram was obtained if HVOO was suspected. The details of these procedures are described elsewhere.[8]

In brief, patients with a pressure gradient across the stenosis of more than 3 mm Hg were considered to have significant HVOO requiring treatment, and balloon venoplasty was initiated. A balloon with a diameter of 6 to 10 mm and a length of 40 mm was used for venoplasty. The diameter of the balloon was matched to that of the vein on the hepatic side of the stenosis. The balloon was placed across the stenosis and was inflated for 60 seconds with an atmospheric pressure of 10. Dilatation with the balloon was performed 3 times, and venography and manometry were repeated to evaluate the effectiveness of venoplasty. The disappearance of the stenosis and a pressure gradient across the stenosis of less than 3 mm Hg were considered signs of success. Immediately after the procedure, heparin was used for several days as a transition to warfarin. Warfarin was administered so that the international normalized ratio was maintained within a range of 1.5 to 2.5 for at least 1 year. Patients with HVOO were followed as outpatients, and clinical manifestations, laboratory data, and Doppler ultrasound were closely observed every 1 to 2 months. Hepatic vein venography was repeated if recurrence of HVOO was suspected. When recurrence was confirmed, balloon venoplasty was performed again. If the patient had experienced recurrence less than 3 months after the initial intervention, had already undergone 2 to 3 sessions of balloon venoplasty, or had done both, expandable metallic stent placement was considered. The SMART stent (Cordis Endovascular, Warren, NJ) was used. The stent size for each case was selected on the basis of the measured hepatic vein diameter on the hepatic side of the stenosis. Stents were intentionally oversized by approximately 1 to 2 mm to minimize the risk of migration.

All data are presented as medians or as means and standard deviations (SDs). Statistical analyses were performed with the Student t test, chi-square test, or Kaplan-Meier analysis. Statistical significance was defined as a P value < 0.05.

RESULTS

Overall Incidence of HVOO and Risk Factors

HVOO occurred in 17 identified cases [4.5% (17/380); 8 males and 9 females]. The age (median = 1.7 years) and recipient body weight (median = 9.5 kg) at LDLT were not significantly different from those for the cases without HVOO (median age = 1.5 years, median weight = 9.1 kg). The incidence of HVOO was highest for the patients with metabolic liver diseases [10.7%

TABLE 2. Characteristics of the Patients with HVOO and the Patients without HVOO

Patient Characteristic	HVOO (n = 17)	No HVOO (n = 363)
Gender, male/female	8/9	149/214
Age, median	1.7 years	1.5 years
Body weight, median	9.5 kg	9.1 kg
GRWR, mean ± SD	2.51% ± 1.42%	2.60% ± 1.17%
Acute cellular rejection, n (%)	7 (41.2)	135 (37.2)
Cytomegalovirus infection, n (%)	4 (23.5)	72 (19.8)
HVOO Case Characteristic	**n**	**% of Total**
Original disease		
Cholestatic liver diseases	14	4.8
Metabolic liver diseases	3	10.7
Graft type		
r-LLS	4	10.8
LLS	11	3.7
LL	2	4.4
Blood-type combination		
Identical	7	2.8
Compatible	8	10.4
Incompatible	2	3.6

(3/28)]. The incidence of HVOO in the LLS group [3.7% (11/298)] was lowest, and it was followed by the LL group [4.4% (2/45)] and the r-LLS group [10.8% (4/37)]. The GRWR (mean = 2.51%) was not significantly different from that of the cases without HVOO (mean = 2.60%). Biopsy-proven acute cellular rejection occurred in 7 cases with HVOO [41.2% (7/17)] versus 135 cases without HVOO [37.2% (135/363); Table 2].

To identify the risk factors for HVOO, a univariate analysis was performed with the following variables: gender, recipient age at LDLT (<1 versus ≥1 year), recipient body weight at LDLT (<10 versus ≥10 kg), original disease (metabolic liver diseases versus other diseases), recipient-to-donor body weight ratio (<0.1% versus ≥0.1%), blood compatibility (incompatible versus identical and compatible), graft type (r-LLS versus LLS and LL), GRWR (≥4% versus <4%), presence of biopsy-proven acute cellular rejection (yes versus no), and presence of cytomegalovirus infection (yes versus no). Table 3 shows that the recipient-to-donor body weight ratio (<0.1%, $P = 0.029$) and graft type (r-LLS; $P = 0.049$) were statistically significant risk factors.

TABLE 3. Potential Risk Factors for HVOO

Variable	Odds Ratio	*P* Value
Male versus female	1.277	0.623
Age: <1 versus ≥1 year	0.879	0.804
Weight: <10 versus ≥10 kg	0.927	0.879
Metabolic liver diseases versus others	2.897	0.097
Recipient-to-donor body weight ratio: <0.1% versus ≥0.1%	3.185	0.029
r-LLS versus others	3.077	0.049
GRWR: ≥4% versus <4%	2.231	0.166
Incompatible versus others	0.763	0.724
Acute cellular rejection: yes versus no	1.182	0.740
Cytomegalovirus infection: yes versus no	1.244	0.710

Analysis of HVOO According to the Type of Hepatic Vein Reconstruction

As shown in Fig. 1, various types of hepatic vein reconstructions were applied during the long-term study period. In almost half of the cases, type A was used in the recipient orifice (n = 193, 48.3%), and this was followed by type C (n = 105, 26.3%) and type B (n = 61, 15.3%). Two anastomoses were made individually between the graft hepatic veins and the recipient hepatic vein stumps in 10 cases. Seven cases were treated with direct end-to-end anastomosis to the IVC or to the right atrium in patients without an IVC (n = 3), an atrophic IVC (n = 3), or en bloc resection of the IVC due to the direct invasion of hepatoblastoma (n = 1). A new orifice on the IVC was created in 4 cases, regardless of the recipient's hepatic veins.

The analysis of the type of hepatic vein reconstruction is summarized in Table 4. The cases treated by venoplasty showed a significantly high incidence of

TABLE 4. Analysis of HVOO According to the Type of Hepatic Vein Reconstruction (n = 359)

	Total (n)	HVOO [n (%)]	*P* Value
Type of recipient hepatic venous orifice			0.071
A	193	9 (4.7)	
B	61	5 (8.2)	
C	105	1 (1.0)	
Type of graft hepatic vein			0.030
Single vein	237	6 (2.5)	
Multiple veins with venoplasty	122	9 (7.4)	
Combination of the recipient orifice and graft veins			0.061
A and single vein	115	4 (3.5)	
A and multiple veins with venoplasty	78	5 (6.4)	
B and single vein	41	2 (4.9)	
B and multiple veins with venoplasty	20	3 (15.0)	
C and single vein	81	0 (0.0)	
C and multiple veins with venoplasty	24	1 (4.2)	

NOTE: Cases treated with a special type of hepatic vein reconstruction (see the right half of Fig. 1) were excluded.

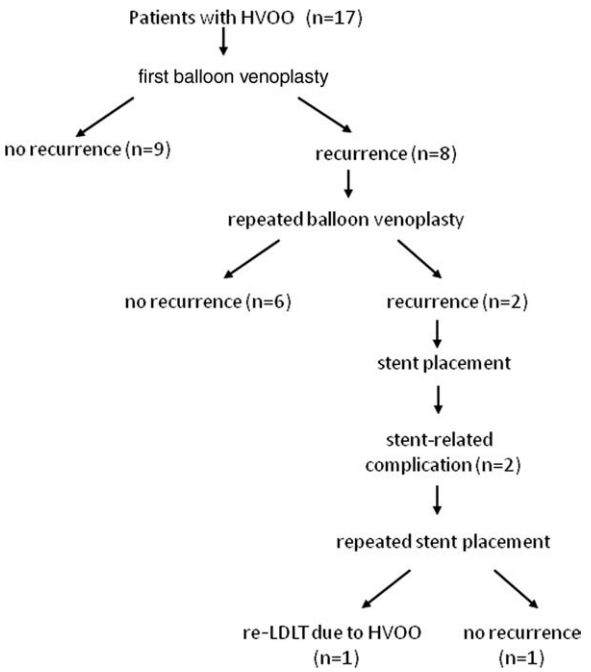

Figure 2. Clinical outcomes of patients with HVOO.

HVOO ($P = 0.030$). Although no significant differences were observed in HVOO between types A, B, and C, the cases treated with type C in the recipient orifice tended to show the lowest incidence of HVOO. The combination of type B in the recipient orifice and multiple graft veins with venoplasty led to a higher incidence of HVOO than the other combinations. One of the cases treated with 2 separated hepatic vein anastomoses developed HVOO in 1 of the 2 anastomoses. One of the cases treated by the creation of a new orifice on the IVC developed HVOO.

Clinical Outcomes of Patients with HVOO (Fig. 2)

All 17 HVOO patients successfully underwent balloon venoplasty after hepatic vein venography. The time to balloon venoplasty varied from 41 days to 6.2 years (median = 1.5 years). Eight patients (47.1%) developed a recurrence after the initial balloon venoplasty. The onset of recurrence after the initial intervention ranged from 59 days to 6.3 years (median = 12.8 months). Although 6 of the 8 patients with recurring HVOO were successfully managed by 2 to 3 sessions of balloon venoplasty, the remaining 2 patients required stent placement. However, both of these patients had stent-related complications of restenosis inside the stent and required further stent placement. One of these patients was alive without recurrence after 3 sessions of stent placement,[13] and the other underwent retransplantation because of liver cirrhosis related to restenosis inside the stent.

The overall graft survival in the cases with HVOO was 72.7% at 10 years; this was lower than that in the patients without HVOO. However, the difference was not statistically significant ($P = 0.983$; Fig. 3).

Analysis of Patients with Recurrent HVOO

The details for the cases with HVOO are summarized in Table 5. The age (median = 1.8 years) and recipient body weight (median = 10.5 kg) at LDLT of the cases with recurrence were not significantly different from those of the cases without recurrence (median age = 1.1 years, median weight = 8.3 kg). The other variables were also not significantly different between the groups. The onset of HVOO after LDLT for the cases with recurrence (mean ± SD = 1.7 ± 1.8 years) was sooner than that for the cases without recurrence (mean ± SD = 2.5 ± 2.3 years), but this difference was not statistically significant. The onset of recurrence after the initial intervention in the cases requiring stent placement was significantly sooner for all cases with recurring HVOO.

DISCUSSION

LDLT is recognized as an established curative therapy for children with liver diseases, and this has resulted in its frequent application in smaller children. This procedure is technically challenging because of the smaller vascular structures of small children, which increase the incidence of vascular complications. A graft for smaller children, even if it is an LLS, becomes relatively large for size, and this compromises the hepatic venous outflow because of compression.[14,15] Buell et al.[16] noted that HVOO predominantly presented in patients of younger ages and lower weights. The present study did not reveal any significant increase in the risk for younger patients or patients of lower weights. In contrast, the factors related to donor-recipient size mismatching, the recipient-to-donor body weight ratio (<0.1%), and the graft type (r-LLS) were identified as significant risk factors. The use of reduced grafts has been introduced with the aim of preventing vascular complications.[17,18]

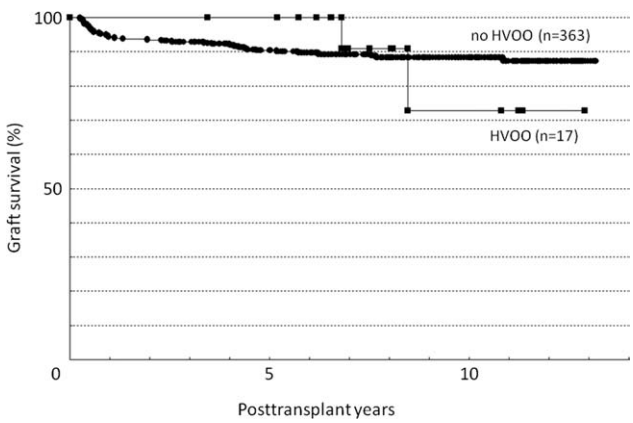

Figure 3. Graft survival in cases with HVOO and in cases without HVOO.

TABLE 5. Analysis of Patients with Recurring HVOO

Case	Age/Gender	Body Weight (kg)	Recipient-to-Donor Body Weight Ratio (%)	Original Disease	Graft Type/ GRWR (%)	Onset After LDLT	Balloon Venoplasty Procedures (n)	Recurrence After First Intervention	Stent Placement	Outcome	Follow-Up
Cases with Recurrence (n = 8)											
1	7 months/male	6.4	0.08	BA	LLS/4.22	1 year 3 months	1	5 months	Yes	Re-LDLT, died	8 years 5 months
2	8 months/female	6.0	0.08	BA	r-LLS/3.36	8 months	3	2 months	Yes	Alive	5 years 2 months
3	1 year 7 months/ male	11.5	0.25	BA	LLS/1.74	1 months	3	6 years 3 months	No	Alive	11 years 3 months
4	1 year 8 months/ male	9.5	0.21	BA	LLS/2.00	3 months	2	3 years	No	Alive	5 years 8 months
5	1 year 10 months/ male	7.8	0.12	BA	LLS/2.94	5 years 1 months	2	1 year 2 months	No	Alive	6 years 10 months
6	6 years/female	21.5	0.39	BA	LLS/0.88	1 year 6 months	2	3 years 10 months	No	Alive	10 years 8 months
7	10 years/male	27.8	0.59	WD	LL/1.08	3 years 10 months	2	7 months	No	Alive	8 years
8	11 years/female	29.0	0.52	BA	LL/1.28	9 months	3	11 months	No	Alive	11 years 4 months
Cases without Recurrence (n = 9)											
1	7 months/male	8.3	0.14	BA	LLS/2.60	5 years 8 months				Alive	6 years 2 months
2	7 months/female	6.7	0.09	BA	r-LLS/4.81	1 year 9 months				Alive	6 years 6 months
3	7 months/female	7.0	0.08	BA	r-LLS/4.95	9 months				Alive	6 years 6 months
4	8 months/female	5.3	0.07	BA	r-LLS/2.83	3 years 4 months				Alive	3 years 5 months
5	1 year 1 month/ female	7.0	0.13	BA	LLS/4.58	1 months				Alive	7 years
6	3 years/female	18.0	0.35	BA	LLS/1.36	2 years 9 months				Alive	12 years 11 months
7	7 years/female	18.2	0.33	BA	LLS/1.46	3 months				Alive	7 years 6 months
8	8 years/male	22.0	0.43	WD	LLS/1.34	2 years				Alive	8 years 1 months
9	10 years/male	29.0	0.44	WD	LLS/1.17	6 years 2 months				Re-LDLT, alive	6 years 10 months

This takes into account the fact that the present techniques used for reducing grafts are not sufficient for preventing these complications. However, for all the cases with reduced grafts, the recipient-to-donor body weight ratio was less than 0.1% (Table 5). We speculated that the present technique used for reducing the grafts did not change the graft thickness, although it did decrease the overall size. Thus, compression might have compromised the hepatic venous outflow. A new technique for reducing both the overall graft size and the graft thickness is needed.

The patency of reconstructed hepatic veins depends largely on the size of the anastomotic orifice, the orientation of the vessels, and the position of the graft.[14] In the current study, venoplasty of the graft hepatic veins was a significant risk factor associated with HVOO. This result emphasizes the importance of retrieving a graft with a single hepatic vein whenever possible. This can be achieved by the placement of a vascular clamp near the IVC to make the hepatic vein longer if the tributary of the graft's hepatic veins is close to the cutting line.[19] When the graft has multiple hepatic veins, venoplasty to combine these hepatic veins is probably insufficient for avoiding HVOO. Several methods of hepatic vein reconstruction have been proposed by different institutes. Members of the Tokyo group prefer an end-to-end anastomosis of the hepatic veins because they have experienced acute outflow occlusion in pediatric LDLT patients with a direct anastomosis of the hepatic veins to the IVC.[20-22] They perform venoplasty by combining hepatic veins with patch grafts in both the recipient and the graft. One of the causes of HVOO, especially in late-onset pediatric cases, is the dislocation of the graft due to the regeneration of the liver parenchyma or the accommodation of the graft in the abdominal cavity.[6,8,23] An end-to-end anastomosis might be affected to a greater extent than an end-to-side anastomosis by the dislocation of the regenerated graft. Thus, the technique proposed by the Tokyo group may be more appropriate in larger children with left-sided lobe grafts. In LDLT using a right lobe graft, the use of a venous graft patch has been effective in maximizing venous outflow.[24] This technique involves an end-to-side anastomosis and the use of a venous graft patch attached only to the anterior side of the anastomosis; this might be a feasible procedure for left-sided lobe grafts.

The long-term efficacy of balloon angioplasty in the treatment of HVOO has been previously reported.[8] However, some patients have experienced recurrence requiring multiple interventions and should have been considered for implantation with an inside metallic stent. In the current study, earlier recurrence was also related to a poorer outcome, and this means that stenosis might be due to the fibrotic nature and/or kinking of the anastomosis.[25] This type of HVOO results in elastic recoil after balloon venoplasty, and such patients might be candidates for stent placement earlier in the course of treatment. There have been several studies focused on stent placement for HVOO.[9,13,15,16,25,26] Buell et al.[16] reported a 72% success rate for stent placement. However, most of these reports have noted hesitation in performing stent placement in pediatric cases for several reasons.[8,26] First, stents are susceptible to intimal hyperplasia, which may lead to recurrent stenosis. Second, a placed stent has a fixed diameter, and this can cause stenosis when a child grows. Third, the presence of an internal stent is technically problematic if retransplantation is necessary. Finally, the long-term patency of stents is still unknown. In our present study, 2 of the cases with stent placement experienced stent-related complications. One of them finally required retransplantation because of liver cirrhosis related to HVOO. The macroscopic findings of the first transplanted graft showed the presence of stenosis inside the stent due to intimal hyperplasia, which might not have been prevented even if permanent anticoagulants had been used. The other case required 3 sessions of stent placement.[13] The selection of the stent size for each case and careful stent deployment are important to prevent these complications.[26] However, the current results clearly show that the outcomes of cases with stent placement are not satisfactory, and stent placement is often technically demanding.

Recently, drug-eluting and biodegradable stents have been clinically used for coronary artery disease.[27] Although the efficacy and safety of new stenting devices should be monitored for long-term use, they may be promising for the management of HVOO. Retransplantation with total replacement of the IVC could be ideal for a patient with HVOO. However, it is difficult to obtain another graft for a recipient because there is a serious shortage of grafts from deceased donors. Moreover, retransplantation from living-related donors presents too much of a burden to the family. Akamatsu et al.[28] reported their meticulous technique using patch plasty or venoatrial anastomosis.[28] Surgical repair of HVOO is more invasive and risky; however, it is a more curative method of treatment. Surgical repair might be considered another therapeutic option for cases refractory to repeated balloon angioplasty despite stent placement.

In summary, although various innovations have been introduced to pediatric LDLT, the incidence of HVOO has not been sufficiently reduced. When grafts with multiple hepatic veins and/or significant donor-recipient size mismatching are encountered, surgical techniques using a patch graft on the anterior side of the anastomosis are recommended. Patients with earlier recurrence of HVOO had a poorer outcome. Stent placement has been considered the next treatment after balloon venoplasty, but the outcomes of these cases were not satisfactory. Stent placement should be carefully considered because of the absence of data on the long-term patency of stents and stent-related complications. New stenting devices, such as drug-eluting and biodegradable stents, may be promising for the management of HVOO.

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