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ORIGINAL ARTICLE

The outcomes of pediatric living donor liver transplantation using small-for-size grafts: experience of a single institute

Naoya Yamada[1] · Yukihiro Sanada[1] · Yuta Hirata[1] · Noriki Okada[1] · Yoshiyuki Ihara[1] · Hideki Sasanuma[2] · Taizen Urahashi[1] · Yasunaru Sakuma[2] · Yoshikazu Yasuda[2] · Koichi Mizuta[1]

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Abstract
Purpose We aimed to evaluate patients who had undergone pediatric LDLT with small-for-size graft (SFSG) and identify risk factors of graft failure to establish a preoperative graft selection strategy.
Methods The data was collected retrospectively. SFSG was used in 14LDLTs (5.7 %) of 245 LDLTs performed between May 2001 and March 2014. The mean patient age and body weight at LDLT were 12.6 ± 2.0 years and 40.5 ± 9.9 kg, respectively. The graft type was left lobe in six patients, left + caudate lobe in seven patients, and posterior segment in one patient.
Results The graft survival rates in SFSG and non-SFSG groups were 78.9 and 93.1 %, respectively ($p = 0.045$). In the univariate analysis, bleeding volume during LDLT were an independent risk factors for graft failure ($p = 0.011$). Graft failure was caused by sepsis in all three patients and occurred at a median of 70 postoperative days 70 (range 14–88 days). Among them, two cases showed high preoperative PELD/MELD score (PELD; 19.4 and MELD; 22, respectively).
Conclusions Pediatric LDLT using SFSG had poor outcome and prognosis, especially when it accompanies the surgical infectious complications with preoperative high PELD/MELD scores.

Keywords Pediatric living-donor liver transplantation · Small-for-size graft · Model for end-stage liver disease score · Predictive graft CT-volumetry · Portal flow modulation

List of Abbreviations

LDLT	Living-donor liver transplantation
SFSS	Small-for-size syndrome
SFSG	Small-for-size graft
GV	Graft volume
SLV	Standard liver volume
MELD	Model for end-stage liver disease
PELD	Pediatric end-stage liver disease
PV	Portal vein

Introduction

Small-for-size syndrome (SFSS) was first described in 1999 and known mainly in the field of adult living-donor liver transplantation (LDLT) [1]. It occurs when the graft

✉ Naoya Yamada
yn708@jichi.ac.jp

Yukihiro Sanada
yuki371@jichi.ac.jp

Yuta Hirata
yutaktyy1234@jichi.ac.jp

Noriki Okada
r0906no@jichi.ac.jp

Yoshiyuki Ihara
ihara@jichi.ac.jp

Hideki Sasanuma
h-ssnm@jichi.ac.jp

Taizen Urahashi
turahashi@jichi.ac.jp

Yasunaru Sakuma
naruchan@jichi.ac.jp

Yoshikazu Yasuda
yyasuda@jichi.ac.jp

Koichi Mizuta
koimizu@jichi.ac.jp

[1] Department of Transplant Surgery, Jichi Medical University, 3311-1 Yakushiji, Shimotsuke, Tochigi 329-0498, Japan

[2] Department of Surgery, Jichi Medical University, Shimotsuke, Japan

volume (GV) is too small to satisfy the recipients' metabolic demand. The symptoms of SFSS are caused by functional impairment of the graft due to the small-for-size graft (SFSG) such as prolonged cholestasis, ascites, coagulopathy, and encephalopathy [2, 3]. In addition, a number of studies have shown that adult LDLT with SFSG is associated with poor prognosis [1, 4–8].

In the field of pediatric LDLT, the patients' characteristics are different from those of adult LDLT patients [9]. However, SFSS has not been thoroughly discussed until now; some pediatric recipients in a later childhood are at risk of SFSS.

The purpose of this study was to analyze our experience with pediatric LDLT using SFSG and identify the risk factors of graft failure in order to establish a preoperative strategy for graft selection.

Patients and methods

Patients

Between May 2001 and December 2013, 245 LDLTs were performed at Jichi Medical University Hospital, Tochigi, Japan. In 14 of the 245 transplantations performed, SFSG were used.

The characteristics of the 14 patients are shown in Table 1. The original disease was biliary atresia in nine patients (64.3 %), Wilson disease in one, graft failure in one, ornithine transcarbamylase deficiency in one, congenital extrahepatic portosystemic shunt in one, and hepatoblastoma in one. The mean age and body weight of the patients at LDLT were 12.6 ± 2.0 years and 40.5 ± 9.9 kg, respectively. The graft type was left lobe in six patients (42.9 %), the left + caudate lobe in seven patients (50.0 %), and the posterior segment in one patient (7.1 %). SFSG was defined as a GV <40 % of the standard liver volume (SLV), and SLV was calculated using Urata's formula [10]. The mean GV and GV/SLV were 331.1 ± 58.4 kg and 36.5 ± 2.5 %, respectively.

Graft selection and surgical procedures for LDLT

The type of donor hepatectomy was determined according to the recipient's SLV and preoperative CT volumetry of the graft liver. In principle, we selected left lobe graft. If the GV/SLV of the left lobe graft was <40 %, we consider the selection of left and caudate lobe, right lobe or posterior segment graft. Routine graft hepatectomy was performed using intraoperative ultrasonic guidance. The donor biliary anatomy was evaluated using either intraoperative repeated real-time or preoperative magnetic resonance cholangiography. The allografts were preserved with University of Wisconsin solution (Viaspan). If necessary, graft hepatic vein venoplasty was performed on the back table.

For the recipient procedure, a Mercedes-Benz or transverse incision was created, and total hepatectomy was performed. In many recipients who had undergone total hepatectomy, the right, middle, and left hepatic veins were formed into a single orifice, which was then anastomosed end-to-end to the graft left hepatic vein, and the portal vein was reconstructed between the recipient right or left portal vein branch patch and the graft left portal vein. Hepatic artery reconstruction was performed using a microsurgical technique. Biliary reconstruction was performed using Roux-en-Y hepaticojejunostomy or duct-to-duct choledochocho-hepaticostomy. Intraoperative color Doppler ultrasonography was performed to assess the blood flow velocity and pattern after vascular reconstruction, and during abdominal wall closure.

Immunosuppressive therapy

Tacrolimus and methylprednisolone were used as the standard postoperative immunosuppression therapy regimen. The target trough levels of tacrolimus and methylprednisolone decreased gradually. Mycophenolate mofetil was used when more potent immunosuppression was required, for example, in ABO-incompatible recipients older than 5 years, in patients with steroid-resistant acute rejection episodes, and in those who developed liver dysfunction after the cessation of methylprednisolone therapy.

Statistical analysis

Data are expressed as median (range) and mean ± standard deviation values. Graft survival was calculated according to the Kaplan–Meier product-limited method. We compared the survival ($n = 11$) and non-survival groups ($n = 3$) using the Student t and Fisher exact tests. All the statistical analyses were performed using the StatView software package (SAS Institute, Cary, NC), and differences with $p < 0.05$ were considered as statistically significant.

Results

The median recipient operative time and amount of bleeding were median 20 h 3 min ± 6 h 1 min and 5321.8 ± 3888.6 mL, respectively.

The graft survival rates in the patients without SFSG ($n = 231$) and in those with SFSG ($n = 14$) were 93.1 and 78.6 %, respectively ($p = 0.045$; Fig. 1). In the SFSG group, we compared the characteristics between the survival ($n = 11$) and graft failure groups ($n = 3$; Table 2).

Table 1 Characteristics of 14patients with SFSG

No.	Original disease	Age (years)	Gender	Body weight (kg)	PELD	MELD	Graft type	GRWR (%)	GV/SLV (%)	Portal flow modulation	Prognosis
1	BA	10	F	28.2	0.1	–	LL	1.04	39.9	–	Alive
2	WD	9	M	29.0	5.2	–	LL	0.95	35.7	–	Alive
3	BA	15	M	50.1	–	9	LL + caudate	0.74	34.6	–	Dead (14 POD, sepsis)
4	BA	12	F	39.5	–	13	LL + caudate	0.89	38.4	–	Alive
5	BA	13	M	53.7	–	12	LL + caudate	0.81	39.1	splenectomy	Alive
6	BA	14	F	32.8	–	13	LL	0.85	33.8	splenectomy	Alive
7	BA	14	F	45.6	–	10	LL + caudate	0.72	33.0	splenectomy	Alive
8	BA	10	F	36.8	4.7	–	LL	0.90	36.8	splenectomy	Alive
9	OTCD	15	M	50.4	–	11	LL + caudate	0.81	37.4	splenectomy	Alive
10	BA	16	F	44.3	–	7	LL	0.72	33.7	–	Alive
11	Graft failure	11	F	30.2	19.4	–	LL + caudate	0.80	33.2	splenectomy	Dead (71 POD, sepsis)
12	BA	13	F	54.5	–	22	PS	0.57	35.6	splenectomy	Dead (88 POD, sepsis)
13	CEPS	13	M	47.2	–	8	LL	0.90	33.9	–	Alive
14	Hepatoblastoma	11	F	24.8	4.2	–	LL + caudate	1.07	39.6	–	Alive

M male, *F* female, *BA* biliary atresia, *WD* Wilson's disease, *OTCD* ornithine transcarbamylase deficiency, *CEPS* congenital extrahepatic portosystemic shunts, *PELD* pediatric end stage liver disease, *MELD* the model for end-stage liver disease, *LL* left lobe, *PS* posterior segment, *GRWR* graft-recipient weight ratio, *GV/SLV* graft volume/standard liver volume, *POD* postoperative days

Bleeding volume was larger in the graft failure group than in the survival group [9132 mL (7438–13,338) vs 2112 ml (700–9618), $p = 0.011$]. No significant differences were observed in the other factors. We plotted the relationship of GV/SLV and PELD/MELD score (Fig. 2), and it revealed that the extremely high PELD/MELD score with SFSG are higher risk of patient survival. In addition, to clarify how the relationship between preoperative PELD/MELD score and GV/SLV affects graft survival, we categorized the recipients older than 8 years into four groups based on the presence or absence of a SFSG and a PELD/MELD score >15 or not, and compared the graft survival rates (Table 3). A preoperative PELD/MELD score >15 is considered an indicator of an extremely high risk of graft failure in patients with a predictive SFSG.

The causes of graft failure were sepsis in all three recipients [bowel perforation (Case 3), acute peritonitis (Case 11), and cholangitis (Case 12)]. We performed liver biopsy in Case 11 and Case 12, and they did not show any findings of acute cellular rejection. Graft failure occurred after a median of 70 postoperative days (range 14–88 days).

To examine the impact of splenectomy and septic surgical complications, we examined the age matched 26 recipients who were older than 8 years at LDLT and absence of SFSG. The splenectomy was performed in eight

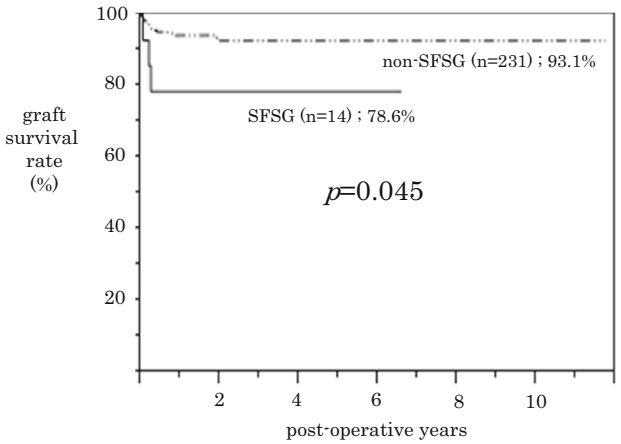

Fig. 1 Graft survival in the SFSG ($n = 14$) and non-SFSG groups ($n = 231$). The patients with SFSG showed poor outcome compared with the patients without SFSG ($p = 0.045$)

patients (31.8 %) and septic surgical complications occurred in ten patients (38.5 %). These are not statistically different compared with the presence or absence of SFSG ($p = 0.31$ and $p = 0.521$, respectively). In five patients with absence of SFSG, splenectomy was performed and septic surgical complications occurred. Among them, one patient could not be rescued, but the four patients (80.0 %) were cured and alive now.

Table 2 Comparison of the characteristics between survival group and graft failure group

	Survival group ($n = 11$)	Graft failure group ($n = 3$)	p value
Age (years)	13 (9–16)	13 (11–15)	0.712
body weight (kg)	39.5 (24.8–53.7)	50.1 (30.2–54.5)	0.418
Graft type	Left lobe 6/11 left + caudate lobe 5/11	Left lobe 1	–
		Left + caudate 1	
		Posterior segment 1	
GV/SLV (%)	36.8 (33.0-39.9)	34.6 (33.2-35.6)	0.225
Operation time	18 h 21 min (12 h 37 min–25 h 56 min)	21 h 08 min (21 h 06 min–37 h 10 min)	0.535
Bleeding volume (ml)	2112 (700–9618)	9132 (7438–13,338)	0.011
Splenectomy	5 (45.5 %)	2 (66.7 %)	1.000
Acute cellular rejection	1 (9.1 %)	0 (0.0 %)	1.000
Vascular complication	1 (9.1 %)	1 (33.3 %)	0.396
Biliary complication	4 (36.3 %)	1 (33.3 %)	1.000

MELD model for end-stage liver disease, *GV/SLV* graft volume/standard liver volume

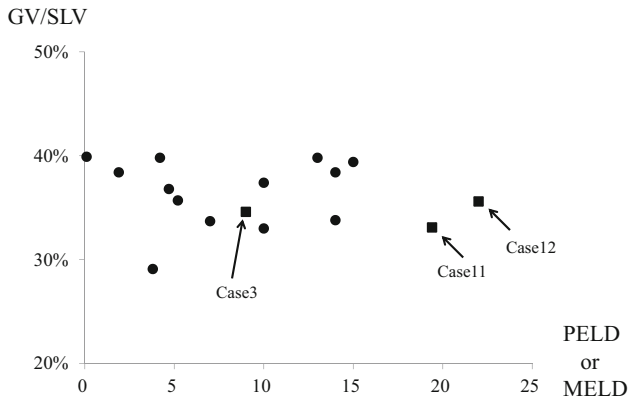

Fig. 2 Relationship of GV/SLV and PELD/MELD score in 14 patients. This revealed that the patients with extremely high PELD/MELD score are high risk and poor outcomes

Table 3 graft survival rates in four groups; the presence or absence of a SFSG and a PELD/MELD score >15 or not

	PELD or MELD <15	PELD or MELD ≥15
GV/SLV >40 %	89.4 % ($n = 19$)	100 % ($n = 7$)
GV/SLV ≤40 %	91.7 % ($n = 12$)	0 % ($n = 2$)

PELD pediatric end-stage liver disease, *MELD* model for end-stage liver disease, *GV/SLV* graft volume/standard liver volume

Discussion

In LDLT, the biggest problem due to SFSG is primary non-function derived from SFSS. Once SFSS occurs, the patient are suffer from much amount ascites due to portal hypertension, coagulopathy, and cholestasis and it may sometime result in graft failure. The SFSG was first defined Graft recipient weight ratio <0.8 % [1] and the definition is slightly differentiated based on the each institute now. In our institute, we use GV/SLV because the body surface area is more reliable especially in cases of obesity or skinny, and considered that the GV/SLV <40 % as a SFSG. In pediatric LDLT, SFSG is a relatively rare condition and it had not been discussed enough, and the strategy is not established until now. However, there actually exist many cases which could not be avoided using SFSG, especially when the recipient is teenagers and the mother is selected as the living donor. To avoid SFSG, a larger graft such as the right lobe graft, has been used as the standard strategy for adult-to-adult LDLT. Although LDLT using a right lobe graft can provide an adequate graft size to meet the metabolic demands of patients, it poses greater risks to the living donors [11, 12]. Therefore, the focus of the SFSG issue is now shifting from how to obtain a larger graft from the living donor to how to manage the use of a smaller graft to save the recipient, prioritizing donor safety [13].

The physiopathology of SFSS is complex, but portal hyperperfusion has been reported to be an important etiological factors of SFSS [14–20]. Nowadays, a portal vein (PV) pressure <15 mmHg is considered adequate to avoid SFSS [21]. To adequately control PV pressure and flow, portal inflow modulation techniques such as splenectomy [21–23], splenic artery ligation [22, 24, 25], or portosystemic shunting [26–31] has been considered. They are effective for temporary control of portal flow, however, they are accompanied by other problems such as overwhelming postsplenectomy infection [32], splenic abscess [33] or the potential risk of the portal flow steal phenomenon [21], respectively. Meanwhile, Ishizaki et al. [34] reported that portal flow modulation is not required in adult LDLT using SFSG. In our institute, as shown in this study, we considered SFSG as the cause of graft failure, and portal flow modulation is needed for patients with

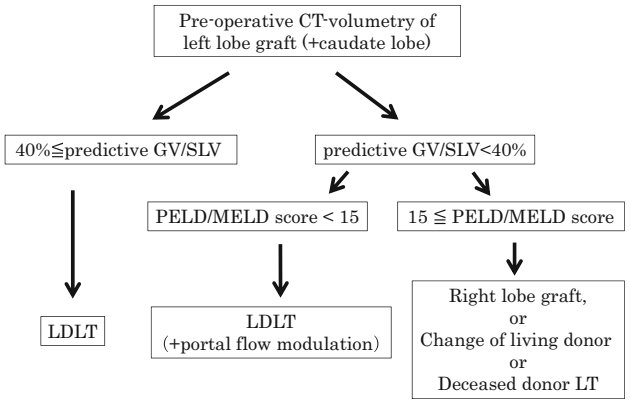

Fig. 3 Our preoperative strategy of graft selection for the predictive SFSG. When the predictive left lobe graft volume allows for the use of a SFSG and the preoperative PELD/MELD score is >15, we have to consider using right lobe graft, posterior segment graft, change of donor or a whole-size matched liver transplantation from deceased donor

excessively high portal flow on LDLT findings and additional splenectomy. The adaptation of portal flow modulation was unclear, but no statistically significant differences were observed in the efficacy and risk of infection with splenectomy in this study.

In the field of pediatric LDLT, the most common indications are cholestatic liver disease represented by biliary atresia. These patients, especially in later childhood, require extremely difficult surgical procedures because of severe adhesion or portal hypertension, which are accompanied by much more bleeding and longer operative time in LDLT. In addition, they are at a higher risk of surgical infections such as cholangitis, abdominal abscess, gastrointestinal perforation, or pancreatic fistula after LDLT. SFSG causes spontaneous cholestasis and portal hypertension; therefore, SFSG with infectious complications in the early period after LDLT may lead to graft failure. In our experience, all the three graft failure patients with SFSG had biliary atresia and were derived from surgical abdominal infections, which resulted in progressive graft failure in the early period after LDLT. We have to recognize that surgical infections in the early phase after LDLT using SFSG are the life-threatening conditions. Therefore, biliary atresia patients with SFSG are at an extremely high risk of graft failure.

Sugawara et al. [35] reported that adult recipients with MELD scores >15 were at higher risk in LDLT using SFSG. They divided the criteria according to the presence or absence of SFSG, with a MELD score >15. In fact, adult recipients of LDLT using SFSG with high MELD scores are considered at high risk of graft failure [11]. Although the statistical analysis is difficult because of small number of cases, our clinical data support the strategy of graft selection according to a PELD/MELD score >15 in pediatric recipients. Based on this study, we show our strategy of preoperative graft selection in Fig. 3. We performed predictive CT-volumetry of the left lobe graft. If the PELD/MELD score of patients is <15, we considered that as sufficient basis to use SFSG. However, if the PELD/MELD score is >15, we have to change the graft to maintain the GV/SLV at >40 %. Then, we have to consider right lobe graft, living donor change, or whole-sized matched liver transplantation from deceased liver transplantation using whole-liver graft.

This study had limitation. The number of cases was small, and we could not find a significant difference in the multivariate analysis. In addition, we did not measure PV pressure in all the cases, and the impact of splenectomy performed as portal flow modulation was not evaluated. However, we believe that this study will help in the selection of the appropriate graft for pediatric recipients who need LDLT.

In conclusion, pediatric LDLT using SFSG is associated with poor outcome and prognosis, when it accompanies the surgical infectious complications in early period after LDLT. In high risk patients with higher PELD/MELD score and with surgical difficulties, using SFSG should be avoided and the graft should be changed to maintain the GV/SLV at >40 %.

Compliance with ethical standards

Conflict of interest No financial support and commercial sponsorship.

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