Impact Of Reconstructing A Single Hepatic Artery On Small-For-Size Grafts In Living Donor Liver Transplantation

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Abstract

Introduction. The recent outcomes of living donor liver transplantation (LDLT) using small for size grafts (SFSG; graft recipient weight ratio; GRWR <0.8) along with portal vein flow modulation has shown encouraging results. However, these liver grafts can at times have two hepatic arterial stumps. This can result in a dilemma whether to reconstruct a single or both arteries. Hepatic artery (HA) thrombosis is the most dreaded complication in LDLT as it can result in graft loss and re-transplantation. We herein report the feasibility of reconstructing a single HA in LDLT having two arterial stumps in the liver grafts with GRWR<0.8.

Methodology. From 2005 to 2010, 50 patients undergoing LDLT with GRWR<0.8 and having multiple HAs, were retrospectively analyzed and divided into Group 1 (n=28): 2 HA stumps with 1 HA reconstruction and Group 2 (n=22): 2 HA stumps with 2 HAs reconstruction. The decision regarding the reconstruction of single or multiple HAs was made depending on the pre-operative radiological and intraoperative assessments. Recipient portal vein flow modulation was done whenever necessary. Outcomes with respect to graft dysfunction, graft loss and patient mortality were compared between the groups.

Results. The incidence of graft dysfunction was similar among the 2 groups (p=0.418). The incidence of biliary strictures and HA thrombosis was similar among the groups. There was no graft loss or patient mortality due to graft dysfunction.

Conclusion. Single HA reconstruction does not increase the risk of graft dysfunction in recipients undergoing LDLT having GRWR<0.8 and dual hepatic arterial stumps in the liver graft.

LDLT: living donor liver transplantation

HA: hepatic artery

SFSG: small-for-size graft

SFSS: small-for-size syndrome

PVFR: portal vein flow rate

MELD: model for end stage liver disease

GRWR: graft-to-recipient weight ratio

DUS: Doppler ultrasound

CTA: CT angiogram

INR: international normalized ratio

INTRODUCTION

Adult-to-adult liver transplantation has gained widespread acceptance as the standard treatment modality for end stage liver disease patients. Over the last few decades, there has been a shift in the paradigm from deceased to living donor liver transplantation (LDLT), due to the ever decreasing pool

of deceased liver donors. Although living donor livers provide a very high quality of livers as compared to those from deceased donors, there is always an ongoing concern regarding sufficient transplanted liver graft volume to avoid any deleterious effects in the recipient. Therefore, the issue of graft-size matching, to obtain the best outcome for the recipient, is a significant concern in LDLT (1-5). Indeed many liver transplantation centers have arbitrarily suggested a graft-to-recipient weight ratio (GRWR) to be at least 0.8, our anecdotal experience suggested that even small-for-size grafts (GRWR<0.8) can have favorable outcomes (6-7).

In general, small-for-size grafts (SFSG) are prone to graft dysfunction hence utmost care is taken to reconstruct all the venous outflows, more close monitoring of liver functions, alterations of immunosuppression regimens and lower threshold for diagnostic biopsies. In the wake of all this, these grafts can at times have presence of multiple hepatic arteries (HA). Early HA thrombosis is a devastating complication and can result in graft loss and re-transplantation. This raises the question if reconstruction of all the HAs is really necessary in the setting of SFSG. Also whether only single HA reconstruction can have any deleterious effect on the regeneration liver graft is not known. The impact of reconstructing only a single HA on the occurrence of graft dysfunction in liver grafts with GRWR <0.8 has never been documented previously. We herein report our results of reconstructing a single HA in presence of dual HAs in liver grafts with low GRWR (<0.8) in LDLT.

METHODOLOGY

We prospectively analyzed the database of all recipients undergoing LDLT at Kaohsiung Chang Gung Memorial Hospital, Taiwan, from 2005 to 2010.

All potential donors with estimated GRWR <0.8 were evaluated for eligibility for the procedure as long as the remnant liver volume in the donor would be >30%. The recipients were well informed about the risk of graft failure due to small graft size. None of the transplants were aborted due to the recipient's concern regarding the graft size. The technique of donor and recipient evaluation has been described elsewhere (8). The liver volume calculations were done according to CT volumetric analysis. The recipient standard liver volume (SLV) was calculated according to the Urata formula (9).The graft weight was taken on the back table after flushing the graft with preservation solution.

Study groups

Fifty patients undergoing LDLT with GRWR<0.8 were identified and divided into Group 1 (n=28): 2 HA stumps with 1 HA reconstruction and Group 2 (n=22): 2 HA stumps with reconstruction of 2 HAs.

Operative techniques

The techniques of donor hepatectomy and recipient hepatectomy have been described in detail elsewhere (10, 11). In the donor, all the arterial structures were carefully dissected and preserved. The proper HA was exposed up to the bifurcation of the left (or middle HA) and the right HA. The plane of division of the HA was determined by the length and size of the artery, its relation with the cutting plane of the liver and the position of the arteries. Before division of the arteries, an intra-operative Doppler ultrasound (DUS) was done to confirm the vascular anatomy.

HA reconstruction

The decision regarding the reconstruction of single or multiple HAs was made depending on the pre-operative radiological and intraoperative assessments. Before transection of the liver parenchyma in the donor, an intra-operative Doppler ultrasound (Acuson, Mountain View, Colorado) was done to confirm the vascular anatomy.

In grafts with multiple arteries, intra-hepatic arterial flow was assessed, after temporary clamping of individual artery and checking for the intra-arterial communications.

In the recipient, during the graft implantation, after reconstruction of the thicker HA, back-bleeding from the thinner arterial branch was assessed and also Doppler arterial flow signals to all the liver segments were confirmed. If any of these findings suggested incomplete liver graft arterial perfusion, both the arterial branches would be reconstructed. There was no selection bias with regards to the surgical expertise or the techniques and all the reconstructions were done by the same experienced micro-vascular surgeon and the details of it have been already published elsewhere (12). Also, intra-operative DUS was used to confirm the patency of the reconstructed HV and portal vein by determining the waveforms, velocity and resistive index of the vessels. The arteries used in the recipients for reconstruction are shown in Table 1.

Portal vein flow

Portal vein flow was assessed by intra-operative DUS and was calculated as flow per minute per 100 gram of liver. If flow was found to be more than 250ml/min/100gm, splenic artery ligation was done to reduce excessive portal flow.

Post-operative care and follow-up

In the recipients, routine DUS was done post liver transplantation to determine adequate blood flow and velocities in the reconstructed arteries and veins. A diagnosis of vascular stenosis or thrombosis was made initially by DUS and then confirmed on 3-dimensional CTA.

Assessment of the outcomes

The outcomes post transplantation were assessed by graft loss, biochemical markers of hepatocyte injury (aspartate amino transferase and alanine amino transferase) and liver function (bilirubin and international normalized ratio; INR). Long-term outcomes were assessed with respect to patient and graft survival.

Small-for-size syndrome (SFSS)

Graft dysfunction was defined according to Dahm et al., as presence of two of the following on 3 consecutive days: bilirubin >100 μmoles/L, prothrombin time INR >2 and encephalopathy grade 3 or 4 in absence of any technical, immunological or infectious cause (13).

Statistical analysis

The cumulative overall survival rates and the graft-dysfunction-free survival rates were calculated using Kaplan Meier methods with the difference evaluated using Log Rank test. Paired t-test was used to analyze the effect of portal flow modulation. To compare frequencies between the 2 groups, we used the Chi-square test.

Univariate analysis was done using the Kaplan Meier method and compared using Log Rank test. Statistical significance was defined as $p<0.05$. All statistics were performed using SPSS version 16.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Donors and grafts

The mean donor age was 54 (50-57) years. There were 5 male and 35 female donors. All the donors recovered uneventfully and none of them required re-operation. The donor average hospital stay was 7.2 days (range, 5-10). Two donors had minimal bile leakage which was managed conservatively. No surgery was aborted due to donor-related factors.

The mean measured graft weight on the back table, post infusion of preservation solution, was 572.5 ± 116 grams. The mean actual GRWR across the study population was 0.72 ± 0.06. The median % Standard Liver Volume (SLV) was 42 ± 3.7%. The demographic and the operative data of the 3 groups are shown in Tables 2 and 3. The groups were comparable in all respects except for the duration for surgery which was longer in Group 2 (p=0.038). This could be due to the extra time required for second HA reconstruction in the recipient.

Portal flow hemodynamics

The upper limit of portal vein flow rate (PVFR) regarded as safe cutoff was 250ml/min/100gm. Portal flow modulation was done if the flow was greater than this value. Splenic artery ligation was done in 4 cases and splenectomy was done in 5 cases. The median PVFR before modulation was 272ml/min/100gm and post modulation it was 190ml/min/100gm. The portal vein flow decreased significantly (p=0.000) post intervention. Figure1. All the liver grafts appeared well perfused post implantation and had good arterial, portal and hepatic venous flows as assessed by intra-operative Doppler ultrasound.

Survival outcomes

The median period of follow-up was 13 months (range, 1-48). There was no case of 30-day in-hospital mortality in any of the 2 groups. Also, there was no incidence of graft loss or primary non-function of any graft.

SFSS

After strict application of the definition of SFSS as described by Dahm et al., (13), there were a total of 4 cases in Group 1and 2 cases in Group 2. The incidence of SFSS was comparable among the groups (p=0.418). Figure 2. However, all these patients had an uneventful recovery. The graft regeneration was adequate in all the other recipients since the synthetic function of the liver had recovered by 1 month post transplantation and the clinical conditions of the patients were good. The changes in the liver enzymes, bilirubin and international normalized ratio are shown in Figures 3, 4, 5 and 6. It is to be noted that all the enzyme levels returned to normal by 4 weeks post transplantation.

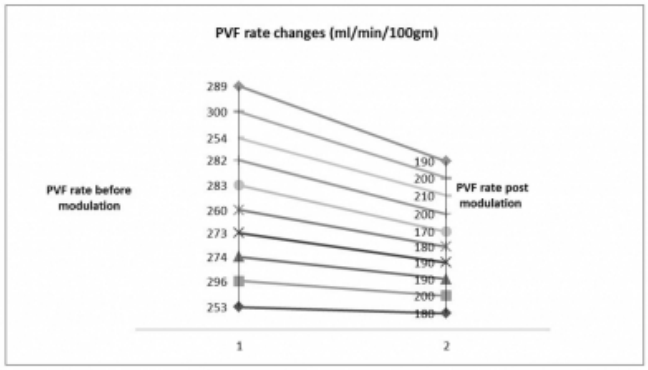
On univariate analysis, only a portal vein flow rate of >200ml/min/100gm of liver graft was found to be a risk factor for graft dysfunction; p=0.000. Further multivariate analysis was not done as only a single factor was found to be significant on univariate analysis. Table 4.

Other complications. The incidence of biliary strictures was comparable between Group 1(n=2) and Group 2 (n=0), p=0.803, within 6 months post transplantation.

Also there were 4 cases of bile leakage from the cut surface of liver (Group 1: 2 cases and Group 2: 2 cases). All these cases were managed non-surgically. There were 3 cases of acute rejections seen 2 in Group 1 and 1 in Group 2. The incidence of hepatic artery thrombosis in Group 1 (n=1) and Group 2 (n=0) was comparable (p=0.498). These cases were managed by early re-exploration and re-do arterial anastomosis with the same artery after trimming it.

Figure 1

Portal flow rate changes post modulation (paired t test; p=0.000 and 95%CI: 57.64-95.87)



PVF: Portal vein flow

Figure 2

Graft dysfunction free survival was comparable among the three 2 groups (p=0.418).

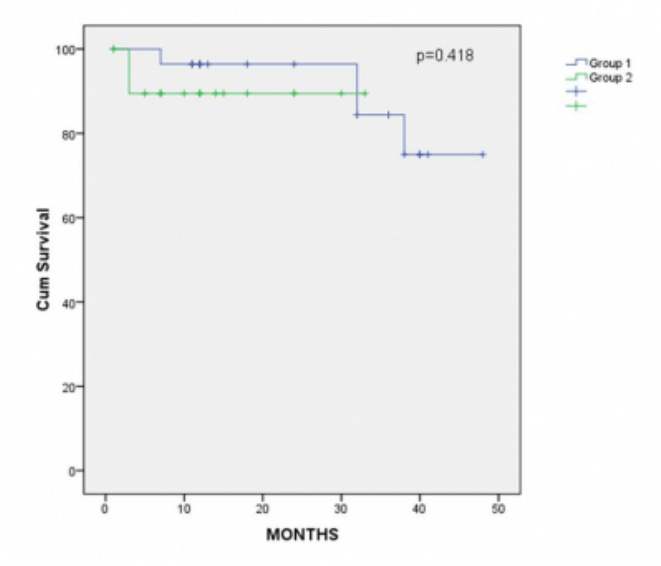


Figure 3

Post-transplantation serum bilirubin levels.

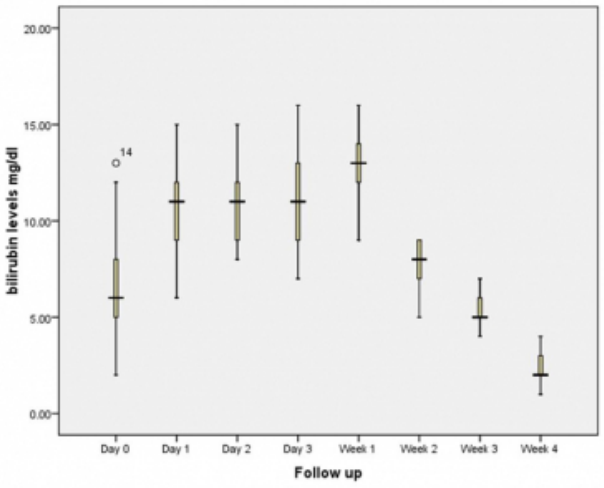


Figure 4

Post-transplantation Prothrombin INR levels.

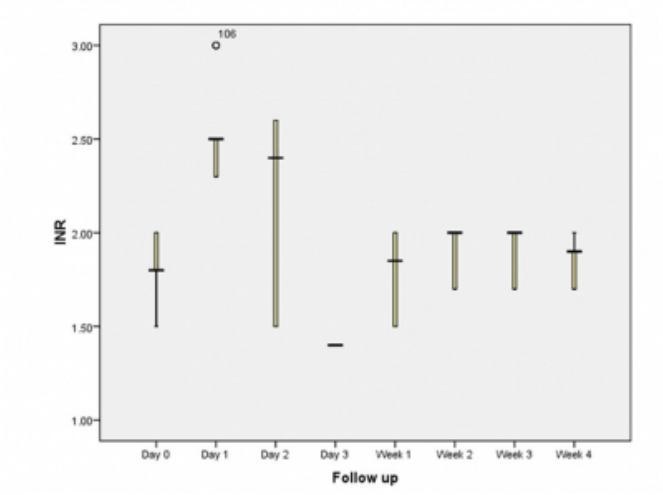


Figure 5

Post-transplantation serum amino alanine transferase levels.

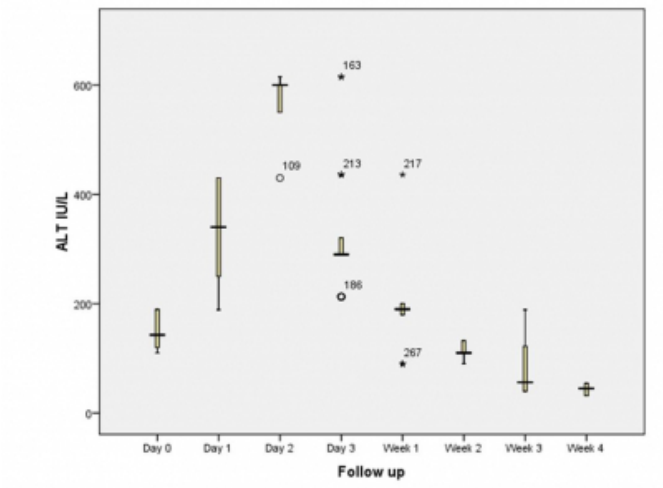


Figure 6

Post-transplantation amino aspartate transferase levels.

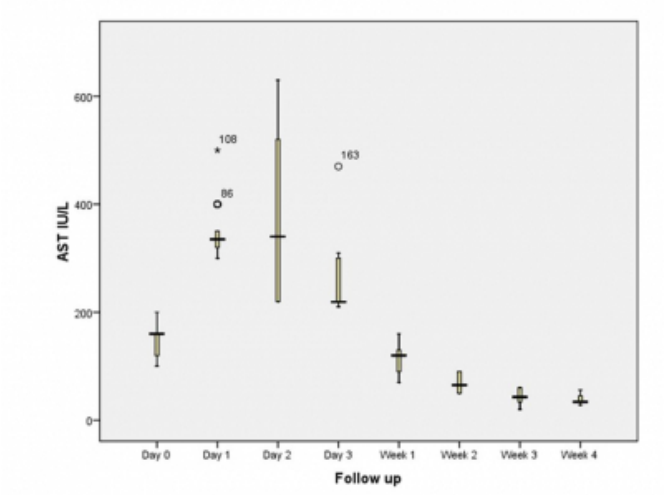


Table 1

Recipient hepatic arteries (HA) used for reconstruction in LDLT.

Groups	Stump of recipient HA	Number of arteries (n)
1	LHA	20
	RHA	6
	Branch of RHA	2
2	RHA	13
	LHA	30
	PHA	1

HA: hepatic artery, LDLT: living donor liver transplantation, LHA: left hepatic artery, RHA: right hepatic artery, PHA: proper hepatic artery.

Table 2

Demographic and clinical data

Category	Group 1 (n=28)	Group 2 (n=22)	p-value
Donor age (yrs)	35 (4.3)	37 (5.1)	0.517
Recipient age (yrs)	53 (5.3)	52 (6.0)	0.600
Sex (M:F)	27:1	18:4	0.087
CTP	7 (2.2)	8 (2.6)	0.778
MELD	10 (2.5)	12 (5.6)	0.777
SLV (%)	42.8 (3.7)	40.2 (3.2)	0.222
Spleen size (ml)	676 (407)	763 (559)	0.474
Graft (right : left)	11:17	13:9	0.164
Diagnosis			
HCV	2	3	-
HBV	5	3	-
HCV and HCC	14	9	-
HBV and HCC	4	2	-
Others	3	5	-

Data is expressed as mean ± SD.

CTP: Child Turcot Pugh score, MELD: model for end stage liver disease, SLV: standard liver volume, HCV: hepatitis C virus, HCC: hepatocellular carcinoma, HBV: hepatitis B virus.

Table 3

Operative details of the recipients

Category	Group 1	Group 2	p-value
Graft HA size (mm)	2.5 (0.6)	2.4 (0.6)	0.239
Cold ischemic time (min)	40 (9.4)	37 (11)	0.441
Warm ischemic time (min)	37 (3.5)	36 (3.4)	0.383
Duration of surgery (min)	534 (70)	857 (156)	0.038
Blood loss (ml)	5520 (1200)	5670 (1600)	0.320
GRWR	0.7 (0.05)	0.7 (0.07)	0.613
Graft weight (gm)	611 (120)	523 (90)	0.355
PVF (ml/min/100gm)	186 (60.4)	204 (62.7)	0.394
Portal flow modulation SAL : Splenectomy	2:3	3:1	0.576

Data is expressed as mean ± SD.

HA: hepatic artery, GRWR: graft-to-recipient weight ratio, PVF: portal vein flow, SAL: splenic artery ligation.

Table 4

Risk factors for graft dysfunction

Category	Univariate analysis p-value
Splenomegaly	0.361
Type of graft	0.702
PVF >200ml/min/100gm	**0.000**
MELD	0.308
Cold ischemic time	0.410
Warm ischemic time	0.782
Duration of surgery	0.278
Blood loss	0.270
Non-reconstructed artery	0.929
GRWR	0.725
CTP	0.762
Graft HA size (mm)	0.763
Hepatic artery thrombosis	0.759

HA: hepatic artery, PVF: portal vein flow, MELD: model for end stage liver disease, GRWR: graft-to- recipient weight ratio, CTP: Child Turcot Pugh score.

DISCUSSION

This study illustrates that reconstructing a single HA in a liver graft having dual HA stumps and low GRWR has no impact on post-transplantation incidence of SFSS in LDLT with SFS liver graft.

One of the landmark studies demonstrating the impact of partial liver graft weight on post-transplantation outcomes was done by Kuichi et al. They published the results in a large study population showing that grafts with GRWR <1 had statistically inferior outcomes as compared to those with GRWR >1 (1). Recently, grafts with GRWR <0.8 have been called small for size grafts (SFSG) and are cautioned to be at an increased risk of developing SFSS. This is based on the results extrapolated from animal studies demonstrating progressive damage linked to portal hyperperfusion leading to sinusoidal congestion, disruption of sinusoidal lining cells and eventually collapse of the space of Disse and cholestasis (14-18). However, some studies have reported excellent survival in recipients undergoing LDLT with liver grafts having a GRWR of <0.8 with excellent outcomes by creating hemiporto-caval shunts (19, 20), splenic artery ligation or splenectomy (19, 21-24). In our study population, we encountered SFSS in 7/54 (13%) of the cases. However, none of them suffered from graft loss or in-hospital 30-day mortality.

We followed the protocol of portal flow modulation in case the PVFR was >250ml/min/100gm. Splenic artery ligation or splenectomy effectively reduced the PVFR to <250ml/min/100gm.

The reciprocal hemodynamic relation between the portal vein flow and recipient hepatic artery flow post transplantation has been described previously (25). It was shown that the mean portal contribution to graft blood flow was 94% and the mean portal-to-arterial ratio was 29. They also suggested that performing splenic artery ligation at the root of the artery allowed an increase in recipient arterial flow and a decrease in portal vein flow to the graft. Thus it was interesting to see what impact would reconstructing only single HA would have on the incidence of SFSS. All the HAs were reconstructed using microsurgical techniques resulting in better outcomes as already described elsewhere by our group (12).

Studies have been done in the past demonstrating the outcomes of reconstructing a single HA with respect to incidence of biliary strictures in LDLT (26, 27). However, none of these studies included grafts with a GRWR of <0.8 and they did not study the incidence of graft dysfunction. One of the most common causes of SFSS is portal hyperperfusion (28). In most of the series, a PVFR >250 ml/min/100gm is regarded as risk factor for development of SFSS (29). In our study, a PVFR >200 ml/min/100gm was identified as a risk factor for the development of SFSS.

It is generally accepted that the recipient's general condition also influences the occurrence of SFSS (30-330. In our study, most of the recipients were having a better clinical condition being mostly Child A/B with lower median MELD score.

It is a well-known fact that graft weight may be overestimated in about 20% with respect to volumetric

measurements (34). This has to be taken in to consideration when selecting donors for LDLT.

In conclusion, the problem of presence of multiple HAs in the SFSG graft can be dealt with by reconstructing a single HA, after confirming the presence of intra-hepatic arterial communications. This offers as a safe and feasible option in LDLT with SFSG and does not have an impact on the incidence of SFSS post transplantation.

References

1. Kiuchi T, Kasahara M, Uryuhara K, Inomata Y, Uemoto S, Asonuma K, et al.: Impact of graft size mismatching on graft prognosis in liver transplantation from living donors. Transplantation; 1999; 67: 321-327.
2. Kiuchi T, Tanaka K, Ito T, Oike F, Ogura Y, Fujimoto Y, et al.: Small-for-size graft in living donor liver transplantation: how far should we go? Liver Transpl; 2003; 9: S29-S35.
3. Tanaka K, Ogura Y: ''Small-for-size graft'' and ''small-for-size syndrome'' in living donor liver transplantation. Yonsei Med J; 2004; 45: 1089-1094.
4. Tucker ON, Heaton N: The ''small for size'' liver syndrome. Curr Opin Cirt Care; 2005; 11: 150-155.
5. Dahm F, Georgieve P, Clavien PA: Small-for-size syndrome after partial liver transplantation: definition, mechanisms of disease and clinical implications. Am J Transpl; 2005; 5: 2605-2610
6. Kaiso T, Mori A, Ogura Y, Hata K, Yoshizawa A, Lida T, et al.: Lower limit of the graft-to-recipient weight ratio can be safely reduced to 0.6% in adult-to-adult living donor liver transplantation in combination with portal pressure control. Transplant Proc; 2011; 43: 2391-2393.
7. Markus S, Arash K, Mark S, Selzner N, Greig PD, Lilly L, et al: A graft-to-body-weight ratio less than 0.8 does not exclude adult-to-adult right-lobe living donor liver transplantation. Liver Transpl; 2009; 15: 1776-1782.
8. Chen CL, Concejero A, Wang CC, Wang SH, Lin CC, Liu YW, et al.: Living donor liver transplantation for biliary atresia: a single-center experience with first 100 cases. Am J Transplant; 2006; 6: 2672-9.
9. Urata K, Kawasaki S, Matsunami H, Hashikura Y, Ikegami T, Ishizone S, et al.: Calculation of child and adult standard liver volume for liver transplantation. Hepatology; 1995; 21: 1317.
10. De Villa VH, Chen CL, Chen YS, Wang CC, Wang SH, Chiang YC, et al.: Outflow tract reconstruction in living donor liver transplantation. Transplantation; 2000; 70: 1604-8.
11. Chen CL, Chen YS, De Villa VH, Wang CC, Lin CL, Goto S, et al.: Minimal blood loss living donor hepatectomy. Transplantation; 2000; 69: 2580-86.
12. Takatsuki M, Chiang YC, Lin TS: Anatomical and technical aspects of hepatic artery reconstruction in living donor liver transplantation. Surgery; 2006; 140: 824-8.
13. Dahm F, Georgieve P, Clavien PA: Small-for-size syndrome after partial liver transplantation: definition, mechanisms of disease and clinical implications. Am J Transpl; 2005; 5: 2605-2610.
14. Ito T, Kiuchi T, Yamamoto H, Oike F, Ogura Y, Fujimoto Y, et al.: Changes in portal venous pressure in the early phase after living donor liver transplantation: pathogenesis and clinical implications. Transplantation; 2003; 75: 1313-1317.
15. Man K, Lo CM, Ng IO, Wong YC, Qin LF, Fan ST, Wong J: Liver transplantation in rats using small-for-size grafts: a study of hemodynamic and morphological changes. Arch Surg; 2001; 136: 280-285.
16. Kelly DM, Demetris AJ, Fung JJ, Marcos A, Zhu Y, Subbotin V, et al.: Porcine partial liver transplantation: a novel model of the ''small-for-size'' liver graft. Liver Transpl; 2004; 10: 253-263.
17. Boillot O, Delafosse B, Mechet I, Boucaud C, Pouyet M: Small-for-size partial liver graft in an adult recipient: a new transplant technique. Lancet; 2002; 359: 406-407.
18. Wang HS, Ohkohchi N, Enomoto Y, Usuda M, Miyagi S, Asakura T, et al.: Excessive portal flow causes graft failure in extremely small-for-size liver transplantation in pigs. World J Gastroenterol; 2005; 11: 6954-6959.
19. Troisi R, Ricciardi S, Smeets P, Petrovic M, Van Maele G, Colle I, et al.: Effects of hemi-portocaval shunts for inflow modulation on the outcome of small-for-size grafts in living donor liver transplantation. Am J Transplant; 2005; 5: 1397-1404.
20. Yamada T, Tanaka K, Uryuhara K, Ito K, Takada Y, Uemoto S: Selective hemi-portocaval shunt based on portal vein pressure for small-for-size graft in adult living donor liver transplantation. Am J Transplant; 2008; 8: 847-853.
21. Troisi R, de Hemptinne B: Clinical relevance of adapting portal vein flow in living donor liver transplantation in adult patients. Liver Transpl; 2003; 9: S36-S4.
22. Umeda Y, Yagi T, Sadamori H, Matsukawa H, Matsuda H, Shinoura S, et al.: Effects of prophylactic splenic artery modulation on portal overperfusion and liver regeneration in small-for-size graft. Transplantation; 2008; 86: 673-680.
23. Cheng YF, Huang TL, Chen TY, Concejero A, Tsang LL, Wang CC, et al.: Liver graft-to-recipient spleen size ratio as a novel predictor of portal hyperperfusion syndrome in living donor liver transplantation. Am J Transplant; 2006; 6: 2994-2999.
24. Ogura Y, Hori T, El Moghazy WM, Yoshizawa A, Oike F, Mori A, et al.: Portal pressure<15 mm Hg is a key for successful adult living donor liver transplantation utilizing smaller grafts than before. Liver Transpl; 2010; 16: 718-728.
25. Troisi R, Cammu G, Militerno G, De Baerdemaeker L, Decruyenaere J, Hoste E, et al.: Modulation of portal graft inflow: a necessity in adult living-donor liver transplantation? Ann Surg; 2003; 237: 429-436.
26. Uchiyama H, Harada N, Sanefuji, Kayashima H, Taketomi A K, Soejima Y, et al.: Dual hepatic artery reconstruction in living donor liver transplantation using a left hepatic graft with 2 hepatic arterial stumps. Surgery; 2010; 147: 878-86.
27. Sugawara Y, Tamura S, Kaneko J, Iida T, Mihara M, Makuuchi M, et al.: Single artery reconstruction in left liver transplantation. Surgery; 2011; 149: 841-5.
28. Lo CM, Liu CL, Fan ST: Portal hyperperfusion injury as the cause of primary nonfunction in a small-for-size liver graft-successful treatment with splenic artery ligation. Liver Transpl; 2003; 9: 626-628.
29. Shimamura T, Taniguchi M, Jin MB, Suzuki T, Matsushita M, Furukawa H, Todo S: Excessive portal venous inflow as a cause of allograft dysfunction in small-for-size living donor liver transplantation. Transplant Proc; 2001; 33: 1331.
30. Xia VW, Du B, Braunfeld M, Neelakanta G, Hu KQ, Nourmand H, et al.: Preoperative characteristics and intraoperative transfusion and vasopressor requirements in patients with low vs. high MELD scores. Liver Transpl; 2006; 12: 614-620.

31. Habib S, Berk B, Chang CC, Demetris AJ, Fontes P, Dvorchik I, et al.: MELD and prediction of post-liver transplantation survival. Liver Transpl; 2006; 12: 440-447.
32. Yi NJ, Suh KS, Lee HW, Shin WY, Kim J, Kim W, et al.: Improved outcome of adult recipients with a high Model for End-Stage Liver Disease score and a small-for-size graft. Liver Transpl; 2009; 15: 496-503.
33. Ikegami T, Masuda Y, Ohno Y, Mita A, Kobayashi A, Urata K, et al.: Prognosis of adult patients transplanted with liver grafts<35% of their standard liver volume. Liver Transpl; 2009; 15: 1622-1630.
34. Sakamoto S, Uemoto S, Uryuhara K, Kim Id, Kiuchi T, Egawa H, et al.: Graft Size assessment and analysis of donors for living donor liver transplantation. Transplantation; 2001; 71: 1407-1413.

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