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Liver Graft-to-Recipient Spleen Size Ratio as a Novel Predictor of Portal Hyperperfusion Syndrome in Living Donor Liver Transplantation

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Portal hyperperfusion in a small-size liver graft is one cause of posttransplant graft dysfunction. We retrospectively analyzed the potential risk factors predicting the development of portal hyperperfusion in 43 adult living donor liver transplantation recipients. The following were evaluated: age, body weight, native liver disease, spleen size, graft size, graft-to-recipient weight ratio (GRWR), total portal flow, recipient portal venous flow per 100 g graft weight (RPVF), graft-to-recipient spleen size ratio (GRSSR) and portosystemic shunting. Spleen size was directly proportional to the total portal flow ($p = 0.001$) and RPVF ($p = 0.014$). Graft hyperperfusion (RPVF flow >250 mL/min/ 100 g graft) was seen in eight recipients. If the GRSSR was <0.6, 5 of 11 cases were found to have graft hyperperfusion ($p = 0.017$). The presence of portosystemic shunting was significant in decreasing excessive RPVF ($p = 0.059$). A decrease in portal flow in the hyperperfused grafts was achieved by intraoperative splenic artery ligation or splenectomy. Spleen size is a major factor contributing to portal flow after transplant. The GRSSR is associated with posttransplant graft hyperperfusion at a ratio of <0.6.

Key words: Living donor liver transplantation, partial liver graft, portal hyperfusion, portosystemic shunting, small-for-size shunting, spleen size

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Introduction

Liver transplantation is an accepted therapy for end-stage liver disease (1). The shortage of deceased donors and the advancement in techniques of hepatectomy have made living donor liver transplantation routine. However, the use of a small-for-size graft has been found to cause a myriad of clinical problems. Dysfunction in a small-for-size graft may cause postoperative hyperbilirubinemia, liver parenchymal injury and may result in liver regeneration failure (2). Accordingly, the use of a small-for-size graft (graft-to-recipient weight ratio (GRWR) <0.8) is found to be associated with a lower recipient survival (3). Although a moderate increase in portal pressure can cause an elevation in shear stress over the sinusoidal endothelium and be a trigger for liver regeneration, an acute elevation in portal pressure in a small-for-size graft after transplantation may induce portal hypertension and liver failure (4). A recipient portal venous flow (RPVF) flow >250 mL/min/ 100 g graft liver weight has been defined as portal hyperperfusion (4).

Due to shortage of deceased liver grafts and the low possibility of retransplantation using deceased donor grafts in Taiwan, adult living donor liver transplantation (ALDLT) has gained acceptance among liver transplant centers. One of the donor criteria prior to actual donation used at the Chang Gung Memorial Hospital-Kaohsiung Medical Center is a GRWR >0.8. In the authors' clinical experience, however, portal hyperperfusion with initial poor function of the graft can happen in recipients of adequate liver graft size based on the GRWR. A concomitant clinical observation in these patients is the presence of splenomegaly with engorgement of the splenic artery and vein. These observations reflect a hyperdynamic splenic circulation and portal flow. The relationship between the size of the recipient's spleen and liver graft is thought to be another factor for posttransplant portal hyperperfusion leading to a small-for-size syndrome.

The aim of this study is to investigate potential pretransplant predicting factors that may result in portal hyperperfusion in ALDLT.

Patients and Methods

Patients

Between September 2003 and June 2005, 43 right lobe ALDLT were performed at the Chang Gung Memorial Hospital-Kaohsiung Medical Center. The recipients received a right liver graft either with or without the middle hepatic vein (HV).

Computed tomography volumetry of the recipient spleen and donor liver graft

Computed tomography (CT) studies were done using a Multislice CT, Somatom Volume Zone scanner (Siemens AG, Germany). The liver and spleen volumes were measured by hand tracing the organ outline on the axial portal venous phase images in the CT examination. Major vessels, including the inferior vena cava and extrahepatic portal vein and major fissures such as the fissure for the ligamentum teres were excluded. The area of the recipient spleen in each section was multiplied by the slice thickness to calculate the volume. The total volume of the spleen was then determined by adding the individual volumes through the organ. The slice thickness of the CT was 1 cm in the donor liver volumetry calculation. The right and left lobes of the liver were determined based on the location of the middle HV as the middle HV bisects the liver. These methods of calculating the hepatic and splenic volumes were validated in previous studies in adult patients (5, 6). The absolute graft weight was assumed to be the actual graft volume because the liver and the spleen have nearly the same density as water (6).

The time point at which CT assessed the spleen volume of the recipients was 3 months (median; range, 1–12) prior to the transplant operation. The actual graft weight in grams during transplantation was used as the graft volume in determining the liver graft-to-recipient spleen size ratio (GRSSR)

Operative management

The authors' techniques of donor graft right lobe hepatectomy and recipient total hepatectomy in ALDLT were described in detail previously (7,8). The graft consisted of the right lobe with or without the middle HV (9). The recipient right HV opening was widened by trimming the vessel edges. The size of this opening was adjusted to measure wider than the graft HV. Assuring the correct orientation of the graft and recipient vessels, the graft HV was anastomosed to the recipient right HV opening with the IVC cross-clamped. Veno-venous bypass was not used in all the recipients. Multiple graft HV were reconstructed either via direct caval anastomosis, graft venoplasty or by use of interposition grafts to the inferior vena cava (10). The graft was reperfused upon completion of portal vein anastomosis followed later by arterial reperfusion. The hepatic artery reconstruction was done using microsurgical techniques. Biliary reconstruction was performed via duct-to-duct anastomosis without stent whenever possible. Intra-operative Doppler ultrasound was performed to check vascular flow patterns and velocities after vascular reconstruction and before and after abdominal closure.

Doppler ultrasound of portal hemodynamics

Portal hemodynamic was measured after graft arterial reconstruction and reperfusion. Intraoperative Doppler ultrasound was performed using an Acuson 512 scanner (Acuson, Mountain View, CA) with a 7.0 scanner in the imaging and Doppler modes. The portal vein flow was measured by recording the angle with corrected flow velocity and the cross-sectional area of the right portal vein (total mL/ min) and expressed as mL/ min/ 100 g graft. Serial determinations of the recipient portal flow (3 recorded readings with a mean) were obtained postreperfusion immediately after skin closure and daily during the first and seventh post-operative days. In the recipients whose splenic artery ligation or splenectomy was done, the measurements of the portal flow were made before and after the splenic procedure.

Statistical analyses

All values were expressed as mean ± SD and median as appropriate. Univariable and multivariable analyses were used to determine the possible relationship between variables. Pearson correlation coefficient was used to determine relationship between total portal flow (TPF) and RPFV. Fisher's exact test was used to demonstrate the relationship between portosystemic shunting and RPVF. Coefficient of coefficient was used to determine relationship between portosystemic shunts, spleen size and TPF. Data were analyzed using statistics computer software STATA (STATA Corporation, College Station, TX).

Results

A total of 43 recipients were included in this study. There were 36 male and 7 female recipients. The mean age was 49.4 ± 10.0 years (range, 18–63). The mean body weight was 68.4 ± 10.4 kg (range, 49–94). The mean size of the recipient spleen was 804.3 ± 387.5 cm^3 (range, 187–1743). The mean liver graft weight was 720.0 ± 121.9 g (range, 412–1023). The mean GRWR was 1.1 ± 0.2 (range, 0.70–1.53). The GRWR was <1 in 12 recipients. The GRSSR was 1.2 ± 0.8 (range, 0.48–3.85). Thirty-five recipients received liver grafts without the middle HV; whereas, the middle HV was included in the graft in eight recipients. Table 1 summarized the demographic variables.

Condition of the portal flow after liver transplant

The portal flow was measured immediately after graft arterial anastomosis. The TPF was 1478.7 ± 441.6 mL/ min (range, 868–2748) and the RPVF was 211.3 ± 59.9 mL/

Table 1: Demographic characteristics of recipients

	No.	Mean ± SD	Range
Gender			
Male	36		
Female	7		
Underlying disease			
HBV-related end-stage liver disease	24		
HCV-related end-stage liver disease	11		
HBV and HCV liver cirrhosis	2		
Autoimmune-induced liver cirrhosis	2		
Primary biliary cirrhosis	3		
Alcoholic liver cirrhosis	1		
Cirrhosis with HCC	16		
Age (yrs)	43	50.74 ± 9.70	18–64
Recipient body weight (kg)	43	68.45 ± 10.45	49–94
Graft size (cm^3)	43	719.96 ± 121.94	412–1023
Spleen size (cm^3)	43	804.3 ± 387	187–1743
GRWR (%)	43	1.07 ± 0.22	0.70–1.55
Total portal flow (mL/ min)	43	1478.68 ± 441.56	868–2748
RPVF per 100 g liver graft (mL/ min)	43	211.27 ± 59.87	103–394
GRSSR (%)	43	1.18 ± 0.78	0.48–3.85

HBV = hepatitis B virus; HCV = hepatitis C virus; HCC = hepatocellular carcinoma; GRWR = graft-to-recipient weight ratio; RPVF = recipient portal venous flow; GRSSR = graft-to-recipient spleen size ratio.

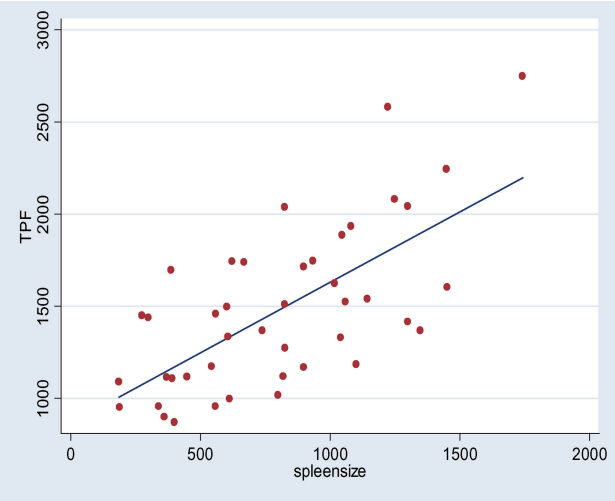

Figure 1: Relationship between pretransplant spleen size and total portal flow in the graft after adult living donor liver transplantation. TPF = total portal flow, in mL/ minute; spleen size, in cm^3 p = 0.001.

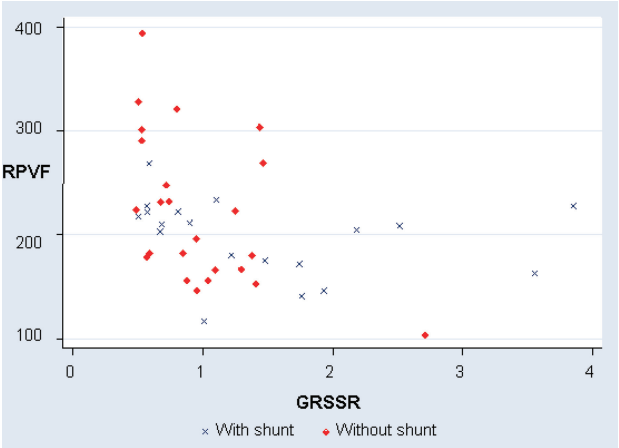

Figure 2: Relationship between GRSSR, RPVF and portosystemic shunting. RPVF = recipient portal venous flow in mL per 100 g liver graft weight; GRSSR = graft-to-recipient spleen size ratio.

min/ 100 g graft (range, 103–394). The spleen size was directly proportional to the total portal flow (p = 0.001) as demonstrated in Figure 1 and to the RPVF (p = 0.014). Using Pearson correlation coefficient between TPF and spleen size, the value was calculated to be 0.6721. The p-value for the coefficient was significant (p = 0.000).

Relationship between RPVF and GRSSR

An RPVF flow >250 mL/ min/ 100 g graft was found in eight recipients. The rest of the recipients demonstrated RPVF flows <250 mL. The GRSSR varied significantly (p = 0.021) with the RPVF as seen in Figure 2. The GRSSR was >0.6 in 32 recipients; whereas, the GRSSR was <0.6 in the remaining 11 recipients. In the group with GRSSR >0.6 (n = 32), three recipients were found to have an RPVF >250 mL. However, in the group with GRSSR <0.6 (n = 11), five recipients were found to have RPVF >250 mL. A GRSSR <0.6 was highly associated with posttransplant elevated RPVF (p = 0.017). The GRSSR value of <0.6 was determined using ROC curve and ROC analysis (see the Appendix, Table A1 and Figure A1). Figure A2 showed the relationship between GRSSR and RPVF.

Relationship between GRWR and RPVF

There was no statistically significant relationship noted between GRWR and RPVF (p = 0.552). In recipients with GRWR <1 (n = 12) two had RPVF >250 mL.

Relationship between recipient age, body weight and native liver disease

Using univariable and multivariable analyses, there were no statistically significant relationships between the development of portal hyperperfusion in the recipient with regards to recipient corrected age, body weight and native liver disease.

Effect of portosystemic shunting

Nineteen recipients with a large portosystemic shunt were identified during the pretransplant survey. A coronary vein was assessed to be engorged if its size was >5 mm by CT measurement in a hemodynamically stable patient. The size of the coronary vein was considered to be a direct evidence for a large portosystemic shunt. The evaluation of an engorged vein in all the CT scans was made by the same radiologist in this series. This evaluation was based on expert opinion (Level V evidence).

The portosystemic shunts included engorged coronary vein (n = 17), gastrorenal shunt (n = 1) and splenorenal shunt (n = 1) based on pretransplant CT angiographic studies. There was no statistically significant relationship noted between the spleen size and the presence or absence of shunting (p = 0.149). However, 1 of 19 showed an RPVF >250 mL/ min/ 100 g graft. In the 24 recipients without significant shunting, 7 had an RPVF >250 mL. A near statistically significant relationship existed between the absence of portosystemic shunting and excessive RPVF (p = 0.059) as demonstrated in Table 3.

When the relationship between GRSSR, RPVF and presence or absence of shunting was analyzed, 4 of 19 patients with spontaneous shunts have a GRSSR of <0.6. In these 4 patients, only 1 developed portal hyperperfusion; whereas in the 24 patients without shunts, 7 have a GRSSR of <0.6. In these 7 patients, 4 developed portal hyperperfusion. These findings were graphically illustrated in Figure 2.

Table 2: Relationship between GRSSR and RPVF

	GRSSR < 0.6	GRSSR > 0.6	Total
RPVF > 250	5	3	8
RPVF < 250	6	29	35
Total	11	32|	43

GRSSR = graft-to-recipient spleen size ratio; RPVF = recipient portal venous flow (in mL per 100 g liver graft weight).
2-sided Fisher's exact test, p value = 0.017.

Table 3: Relationship between portosystemic shunting and RPVF

	With shunt	Without shunt	Total
RPVF >250	1	7	8
RPVF <250	18	17	35
Total	19	24	43

RPVF = recipient portal venous flow in mL per 100 g liver graft weight.
Fisher's exact test, p value = 0.059.

Prognosis of the graft

An RPVF >250 mL/ min/ 100 g graft was found in eight recipients (309.6 ± 40.2 mL/ min/ 100 g graft; range, 269–394). Intra-operative splenic artery ligation was performed in four of eight and splenectomy in one of eight of these recipients. A decrease in RPVF to <250 mL was achieved in the eight recipients who received intervention. The preintervention RPVF mean was 346.4 ± 46.6 mL; and the postintervention RPVF mean was 193.4 ± 32.9 mL. There was no mortality or operative complication; likewise, there was no primary nonfunction or delayed non-function of the graft in these five recipients.

In the authors' early experience, there were no interventions performed to attempt to decrease the portal flow in the first three recipients with RPVF >250 mL. Poor initial function of the graft developed in one of three of these recipients. This same recipient was the only mortality in this series. In two other recipients with an RPVF of 260–270 mL where no intervention was performed, the RPVF decreased gradually over a few days. Portal hyperperfusion did not worsen and initial poor function or primary nonfunction of the graft did not develop.

Discussion

ALDLT is an effective approach to decrease the number of transplant candidates in the waiting list. However, size mismatch is a major obstacle in ALDLT. Its incidence varies significantly among western and Asian countries and it may represent up to >50% in most active centers (11). However, the consequences of using a small graft in a cirrhotic patient with severe portal hypertension are largely unpredictable. The so-called small-for-size syndrome seems to be present in most worldwide series (12). This syndrome may have different etiologies, including those related to the graft, such as size, actual functional mass, anatomic variability and the presence of severe portal hypertension. It is also well known that small donor grafts for recipients may induce postoperative hyperbilirubinemia and liver injury that may result in liver regeneration failure (13).

Although the use of the GRWR has been well accepted as an important predictor of the adequacy of post-transplant liver function with a safety ratio range of >0.8, there have been exceptions reported in which this ratio was clearly lower than 0.8 (range, 0.6–0.8) and yet satisfactory graft function was observed (14). It is then clear that graft function and survival are influenced not only by graft size but also by pretransplant disease severity and the associated portal hypertension (15). Consistently, a graft with a GRWR as low as 0.6 can be used safely for patients without advanced cirrhosis or for patients in Child's class A (3). Since the splenic component accounts for up to 52% of the total portal venous flow (16), its contribution to portal hypertension cannot be ignored. As a result, it is not unreasonable to propose that spleen size may reflect the severity of pretransplant portal hypertension. In this study we found that the size of the spleen is in linear correlation with the amount of the portal flow. If the GRSSR is <0.6, there is a high possibility of excessive portal flow. Therefore, other than the GRWR that predicts the occurrence of small-for-size syndrome based on the static factors of the liver graft and recipient body weight, the GRSSR seems to be another useful parameter that takes into account the recipient's portal hemodynamic status in predicting posttransplant portal hyperperfusion syndrome which may eventually lead to a small-for-size syndrome. Since the size of the spleen and the liver graft from the living donor can be easily obtained through noninvasive imaging studies as CT or magnetic resonance imaging, this parameter appears particularly valuable in assessing the possible development of posttransplant portal hyperperfusion during the pretransplant survey.

The implication of this study is that for recipients who have a GRSSR <0.6, interventions to modulate and prevent excessive portal graft flow may be considered such as splenic artery ligation or splenectomy as previously reported (16–18). Based on the same theory, the Boillot group developed a new transplant technique in ALDLT using a small-for-size graft by diverting the superior mesenteric venous flow with a mesocaval shunt and downstream ligation of the superior mesenteric vein to avoid graft congestion and failure by over-perfusion (19). The importance of splenic arterial inflow in affecting the portal venous flow is reflected in the finding that ligation of the former leads to a 45% drop in the latter, improvement in the recipient hepatic arterial flow and resolution of refractory ascites (16–18).

Portosystemic shunting used to be a common surgical practice in alleviating severe portal hypertension and its associated life-threatening complications such as esophageal and gastric varices. On the other hand,

portosystemic shunts significantly reduce portal venous flow. Based on the theory of excessive portal flow as a cause of small-for-size syndrome, spontaneous portosystemic shunts should be left in place when small grafts with hyperkinetic portal flow are used in ALDLT. The total portal flow towards the liver is markedly reduced in patients with large portosystemic shunts as compared with those without shunting. In the 19 recipients who had large portosystemic shunts, only 1 was found to have an excessive portal flow (>250 mL / min/ 100 g graft) when compared with the nonshunt group in which 7 of 24 cases were found to have an excessive portal flow ($p = 0.059$). Therefore, our results suggest that portosystemic shunting may be of benefit in decreasing portal flow in ALDLT when portal hyperperfusion is present.

Although pretransplant parameters such as GRWR and GRSSR can be used as reasonable predictors for the prevention of possible portal hyperperfusion syndrome in ALDLT, excessive portal venous flow can only be confirmed after the actual graft reperfusion using either direct measurement of the portal venous pressure or portal venous blood flow. Information obtained during the pretransplant survey provides valuable guidance in evaluating possible pretransplant or intraoperative measures to prevent portal hyperperfusion syndrome which include preservation of existing native portosystemic shunting, splenic artery ligation, splenectomy or diversion of the superior mesenteric venous flow through mesocaval shunting.

In conclusion, the hemodynamic patterns after right lobe ALDLT are predictable based on the GRWR and the GRSSR. The GRWR is important but an incomplete parameter in predicting the magnitude of hemodynamic changes in ALDLT because it does not take the portal hemodynamic factor into consideration. On the other hand, the size of the spleen is strongly associated with excessive portal venous flow; whereby, it serves as a useful parameter in the calculation of the GRSSR. The spleen size also showed a statistically significant linear relationship with the RPVF. A GRSSR <0.6 may predict the development of post transplant portal hyperperfusion.

Acknowledgments

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Appendix

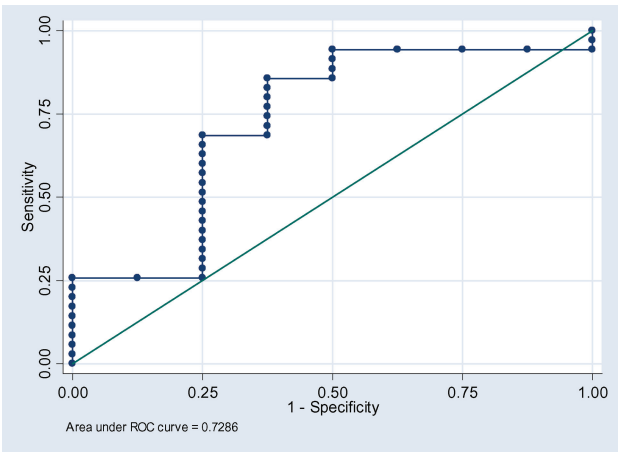

Figure A1: ROC curve detailing the GRSSR value (n = 43). GRSSR = graft-to-recipient spleen size ratio.

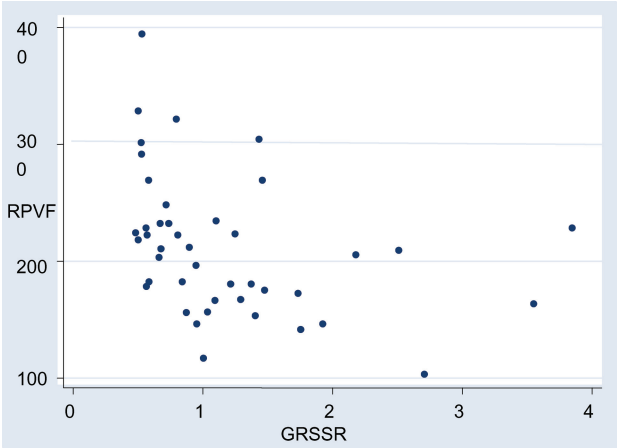

Figure A2: Relationship between GRSSR and RPVF. RPVF = recipient portal venous flow in mL per 100 g liver graft weight; GRSSR = graft-to-recipient spleen size ratio. p = 0.021.

Table A1: ROC analysis detailing sensitivity and specificity of GRSSR values

Cut point	Sensitivity	Specificity	Classified	LR+	LR–
(>= .4862)	100.00%	0.00%	81.40%	1.0000	
(>= .5044)	97.14%	0.00%	79.07%	0.9714	
(>= .5064)	94.29%	0.00%	76.74%	0.9429	
(>= .5299)	94.29%	12.50%	79.07%	1.0776	0.4571
(>= .5317)	94.29%	25.00%	81.40%	1.2571	0.2286
(>= .53469)	94.29%	37.50%	83.72%	1.5086	0.1524
(>= .567)	94.29%	50.00%	86.05%	1.8857	0.1143
(>= .568)	91.43%	50.00%	83.72%	1.8286	0.1714
(>= .5738)	88.57%	50.00%	81.40%	1.7714	0.2286
(>= .5869)	85.71%	50.00%	79.07%	1.7143	0.2857
(>= .5898)	85.71%	62.50%	81.40%	2.2857	0.2286
(>= .6675)	82.86%	62.50%	79.07%	2.2095	0.2743
(>= .675)	80.00%	62.50%	76.74%	2.1333	0.3200
(>= .68245)	77.14%	62.50%	74.42%	2.0571	0.3657
(>= .721)	74.29%	62.50%	72.09%	1.9810	0.4114
	ROC		Asymptotic Normal		
	Obs	Area	Std. Err.	[95% Conf. Interval]	
	43	0.7286	0.1148	0.50351	0.95364

GRSSR = graft-to-recipient spleen size ratio