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Dysfunction in Patients With Small-for-Size Grafts After Living Donor Liver Transplantation

Shozo Mori[1,2], Min-Su Park[1], Hyeyoung Kim[1], Youngrok Choi[1], Geun Hong[1], Nam-Joon Yi,[1] Kwang-Woong Lee[1], Kyung-Suk Suh[1]

[1]*Department of Surgery, Seoul National University College of Medicine, Seoul, Korea*

[2]*Department of Gastroenterological Surgery, Dokkyo Medical University, Tochigi, Japan*

The relationship between postoperative percentage fall of platelet (PLT) counts and graft dysfunction after living donor liver transplantation (LDLT) in recipients with small-for-size (SFS) graft has not been fully evaluated. We retrospectively studied 50 adult-to-adult LDLT recipients with a graft-to-recipient weight ratio of <0.8% between 1999 and 2011. Graft dysfunction was defined as the presence of hyperbilirubinemia, coagulopathy, or ascites on 3 consecutive days during the first postoperative week. Each clinical sign of dysfunction was assigned 1 point. Postoperative percentage fall in PLT counts, graft dysfunction score, and postoperative complications according to the Clavien-Dindo classification were investigated. Overall, 31 patients (62%) exhibited a PLT count fall of more than 50%, and 19 (38%) patients exhibited a PLT count fall of less than 50% at postoperative day (POD) 3. Receiver operating characteristic curve analysis indicated that at POD 3, the cutoff value of PLT count fall was 56% for a graft dysfunction score of 2 or 3 (sensitivity, 70%; specificity, 63.3%). Fourteen of 20 patients (70%) with a dysfunction score of 2 or 3 and 11 of 30 patients (37%) with a dysfunction score of 0 or 1 showed a fall in PLT count >56% at POD 3 ($P = 0.021$). Grade 2 to 5 complications were more observed in patients with a dysfunction score of 2 or 3 than in patients with a dysfunction score of 0 or 1 ($P < 0.001$). The fall of PLT count at POD 3 >56% is an ominous sign that can predict the graft dysfunction after LDLT in recipients with SFS graft.

Key words: Thrombocytopenia – Small-for-size graft – Portal hypertension – Small-for-size syndrome – Graft dysfunction

Corresponding author: Kyung-Suk Suh, MD, PhD, Department of Surgery, Seoul National University College of Medicine, Seoul National University Hospital, 28 Yongon-Dong, Chongno-Gu, Seoul 110–744, Korea.
Tel.: 82 2 2072 3789; Fax: 82 2 766 3975; E-mail: kssuh@snu.ac.kr

Since living donor liver transplantation (LDLT) has become widely accepted as a treatment of choice for end-stage liver disease (ESLD),[1] we often encounter a situation involving graft-size mismatching. A graft-to-recipient weight ratio (GRWR) of <0.8% has been demonstrated as a predictor of poor morbidity and mortality.[2] Patients with ESLD frequently suffer from thrombocytopenia and refractory ascites caused by portal hypertension before surgery. In LDLT, portal hypertension is not immediately relieved after surgery, especially when using a small graft; moreover, it leads to graft dysfunction and so-called small-for-size (SFS) syndrome.[2,3] Use of the smaller graft may contribute to slow recovery of platelet (PLT) counts or protracted thrombocytopenia.

In the clinical setting, the lowest PLT counts are usually observed during the first week after LDLT and recover with restoration of graft function. The delayed recovery of PLT counts may lead to increased morbidity and mortality resulting from bleeding-related complications and infections during the postoperative period.[4,5] Previous studies have not clearly demonstrated the relationship between postoperative thrombocytopenia and graft dysfunction following LDLT, especially in SFS graft recipients.

The aim of this study was to investigate whether the observed early postoperative percentage fall of PLT counts predicts the graft dysfunction of patients with an SFS graft (GRWR <0.8%) after LDLT.

Patients and Methods

Recipients

A retrospective review of adult-to-adult LDLT was carried out from April 1999 to January 2011. This study proceeded after obtaining approval from the Institutional Review Boards of the Seoul National University College of Medicine. During this period, 51 recipients meeting the criteria of GRWR <0.8% and recipient age >18 years were identified. One patient was excluded from this study because a splenectomy to control bleeding had been simultaneously performed with LDLT. Therefore, the study included 39 men (78%) and 11 women (22%) with a median age of 49 years (range, 21–65 years) and a median body mass index (BMI) of 24.3 kg/m^2 (range, 16–35.9 kg/m^2). The indications for transplantation in these patients included virus-related liver cirrhosis (n = 43; hepatitis B and C virus, 39 and 4, respectively), cryptogenic liver cirrhosis (n = 3), alcoholic liver cirrhosis (n = 2), hepatitis A virus-related fulminant hepatic failure (n = 1), and Wilson disease (n = 1); of these, 18 (36%) suffered from hepatocellular carcinoma (HCC). Preoperative examination of the hepatic reserve demonstrated a median Child-Pugh score of 11 (range, 6–14) and a model for ESLD (MELD) score of 20 (range, 7–51). Overall, the preoperative median PLT count was $55.5 \times 10^3/\mu L$ (range, $10 \times 10^3/\mu L$ to $263 \times 10^3/\mu L$) (Table 1). The lowest PLT count was observed at postoperative day (POD) 3 ($24 \times 10^3/\mu L$), and recovery from thrombocytopenia occurred at POD 14 ($104 \times 10^3/\mu L$). However, low PLT counts persisted for 1 year after surgery (POD 365; $122 \times 10^3/\mu L$; Fig. 1).

The recipient operations have been described previously.[6] Regarding the surgical data, the median operation time and estimated blood loss were 528 minutes (range, 335–820 minutes) and 4000 mL (range, 500–50,000 mL), respectively. All recipients received red blood cell transfusions, with a median of 8 units (range, 1–59 units), and 13 recipients received platelet transfusions, with a median of 10 units (range, 2–12 units). For biliary anastomosis, 38 recipients (76%) underwent reconstruction with a ductal anastomosis, and 12 recipients (24%) underwent a hepaticojejunostomy. Neither preoperative or intraoperative prophylactic splenic artery modulation nor splenectomy to reduce portal hypertension was performed, nor were there any ABO-mismatched cases). The median intensive care unit (ICU) and hospital stays were 8 days (range, 3–78 days) and 27 days (range, 11–141 days), respectively. Patients were followed up for a median of 35.5 months (range, 0.4–145.1 months) (Table 1).

The definitions used for complications were adapted from the Clavien grading system for negative outcomes.[7] Grade 1 included minor risk events that required administration of analgesics, antipyretics, anti-inflammatories, and antiemetics, as well as drugs for urinary retention, lower urinary tract infection, arterial hypertension, hyperlipidemia, or transient hyperglycemia. Grade 2 complications were potentially life threatening but did not require invasive interventions. Complications that were greater than grade 3 were considered major complications requiring invasive interventions (grade 3) or leading to lasting disability or organ dysfunction that was either difficult to control or had a significant risk of leading to graft failure (grade 4) or death (grade 5).

Table 1 Clinical characteristics of recipients with SFS grafts

Variables	Total, n = 50
Age, y (range)	49 (21–65)
Sex, male (%)	39 (78%)
Underlying liver disease	
Hepatitis B/C (%)	39/4 (86%)
Others (%)	7 (14%)
HCC (%)	18 (36%)
Child-Pugh score (range)	11 (6–14)
MELD score (range)	20 (7–51)
PLT count, $\times 10^3/\mu L$ (range)	55.5 (10–263)
Graft type, right/left	29/21
Graft weight, g (range)	510 (250–815)
GRWR, % (range)	0.74 (0.49–0.79)
ICU stay, d (range)	8 (3–78)
Hospital stay, d (range)	27 (11–141)
Follow-up, mo (range)	35.5 (0.4–145.1)

Donors and grafts

Donor selection and surgical procedures were performed according to published procedures.[8] The donors included 32 men and 18 women with a median age of 30 years (range, 17–53 years) and a median BMI of 22.4 kg/m^2 (range, 16.6–29.7 kg/m^2). We performed 29 (58%) right lobe grafts and 21 (42%) left lobe grafts to the middle hepatic vein (MHV). Of the 29 right lobe grafts, 11 were extended right lobe grafts to the MHV and 14 were modified right lobe grafts in which the MHV or its tributaries were reconstructed by artificial vascular grafts. Four grafts were conventional right lobe grafts without reconstruction of the MHV. Graft weights ranged from 250 g to 815 g (median, 510 g), and the GRWR ranged from 0.49% to 0.79% (median, 0.74%). Six grafts (12%) were diagnosed as macrovesicular steatosis, ranging from 10% to 30% by intraoperative wedge biopsy. The median operation time was 298 minutes (range, 190–545 minutes), and the estimated blood loss of donor hepatectomy was 300 mL (range, 50–1000 mL). Donors were not administered transfusions. The median cold and warm ischemia times were 66 minutes (range, 15–166 minutes) and 41 minutes (range, 23–80 minutes), respectively.

Definition of graft dysfunction and dysfunction score

Graft dysfunction was defined as the presence of hyperbilirubinemia [serum total bilirubin >100 μmol/L (5.8 mg/dL)], coagulopathy [prothrombin time–international normalized ratio (PT-INR) >2], or ascites >1000 mL/d on 3 consecutive days during the first postoperative week. Each clinical sign of dysfunction was assigned 1 point, and the final graft dysfunction score (range, 0–3) consisted of the sum of the assigned points.[9,10]

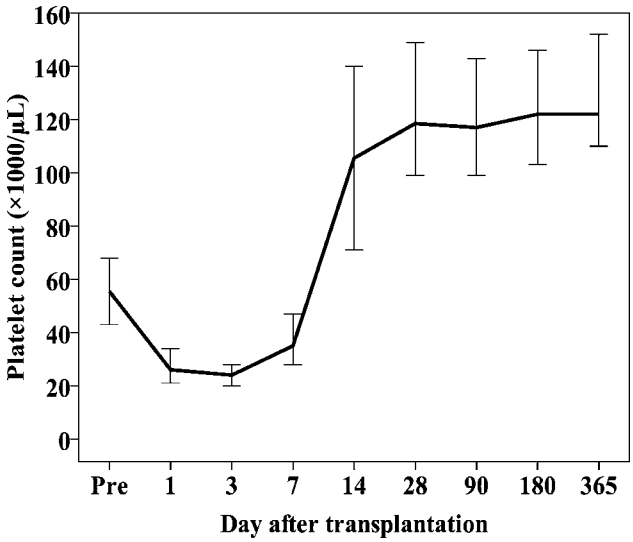

Fig. 1 Evolution of PLT counts up to 1 year after LDLT. The data represent the median values, and the vertical "I" bars indicate 95% confidence intervals (CI). The median preoperative PLT count was $55.5 \times 10^3/\mu L$. The lowest PLT count was observed at POD 3 ($24 \times 10^3/\mu L$), and recovery from thrombocytopenia occurred at POD 14 ($104 \times 10^3/\mu L$). However, low PLT counts persisted for 1 year after surgery (POD 365; $122 \times 10^3/\mu L$).

Statistical analysis

Data are expressed as the median and range. Thrombocytopenia was defined as a PLT count of <100,000/μL. Categoric variables were compared with the χ^2 test, whereas continuous variables were compared using the Mann-Whitney *U* test.

Statistical analyses were performed using the SPSS statistical software package, Version 17.0 (SPSS Inc, Chicago, Illinois). A P value <0.05 was considered statistically significant.

Results

The percentage fall of PLT count at POD 3 for each patient is shown in Fig. 2. Overall, 31 patients (62%) exhibited a PLT count fall of more than 50%, and 15 (30%) patients exhibited a PLT count fall of less than 50%. Four (8%) patients showed a PLT count rise, ranging from 0% to 50%.

Receiver operating characteristic (ROC) curve analysis is shown in Fig. 3A. ROC curve analysis indicated the cutoff value of PLT count fall at POD 3

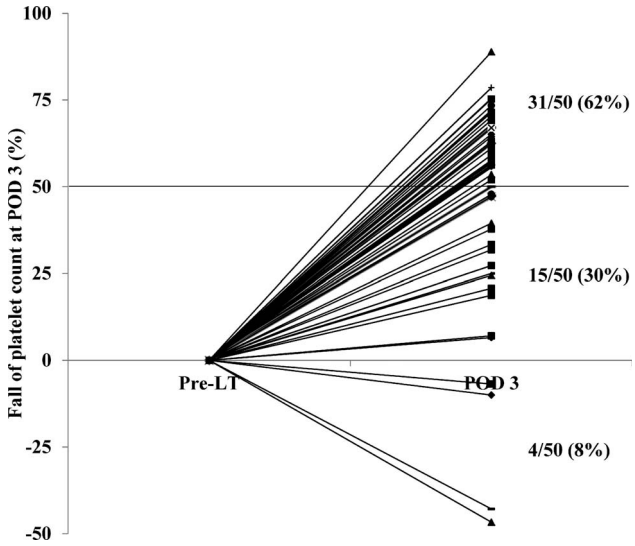

Fig. 2 Percentage fall of PLT count at POD 3. Of 50 patients, 31 (62%) and 15 (30%) exhibited a PLT count fall of more than 50% and less than 50%, respectively. In contrast, 4 (8%) patients exhibited a PLT count rise ranging from 0% to 50%.

of 56% for graft dysfunction score of 2 or 3 (sensitivity, 70%; specificity, 63.3%). Evolution of PLT count according to the fall of PLT count at POD 3 >56% or ≤56% up to 1 year after LDLT is shown in Fig. 3B. No significant difference was observed between the 2 groups in terms of the amount of intraoperative platelet transfusion ($P = 0.333$). The preoperative PLT count showed no statistically significant difference between the 2 groups. The fall of PLT count in the >56% group showed a less steep increase in the PLT count after LDLT compared with that of the ≤56% group.

The dysfunction score in the fall of PLT count at POD 3 for the >56% or ≤56% groups is shown in Fig. 4A. The dysfunction score was significantly higher in the fall of PLT count of the >56% group than that of the ≤56% group (1.7 ± 1.2 versus 1 ± 1.1; $P = 0.032$). Plot of each percentage fall of PLT count at POD 3 used for the graft dysfunction score, ranging from 0 to 3, is described in Fig. 4B. In patients who had a dysfunction score of 2 or 3 (n = 20), 14 (70%) patients had shown a fall of PLT count >56% at POD 3. However, in patients who had a dysfunction score of 0 or 1 (n = 30), 11 (37%) patients had shown a fall of PLT count >56% at POD 3 ($P = 0.021$).

Morbidities within 3 months after LDLT are shown in Table 2. Of 20 patients with a dysfunction score of 2 or 3 (n = 20), 19 (95%) patients had Clavien grade 2 to 5 complications. In contrast, 13 of 30 patients (43%) with a dysfunction score of 0 or 1 had Clavien grade 2 to 5 complications ($P < 0.001$).

Discussion

Our results show that a reduction in the PLT count occurred in all recipients with SFS graft. Thrombocytopenia occurred frequently and persisted in 43 of 50 recipients (86%) at POD 7, and recovery from thrombocytopenia occurred at POD 14. However, low PLT counts persisted for 1 year after surgery.

Thrombocytopenia after liver transplantation can be caused by hemodilution, immunologic reactions, consumption, or sequestration or entrapment of PLTs in the liver graft.[4,5,11] In patients with ESLD, an increased sequestration of PLTs in the spleen owing to portal hypertension, which results in splenomegaly, hypersplenism, and reduced production of

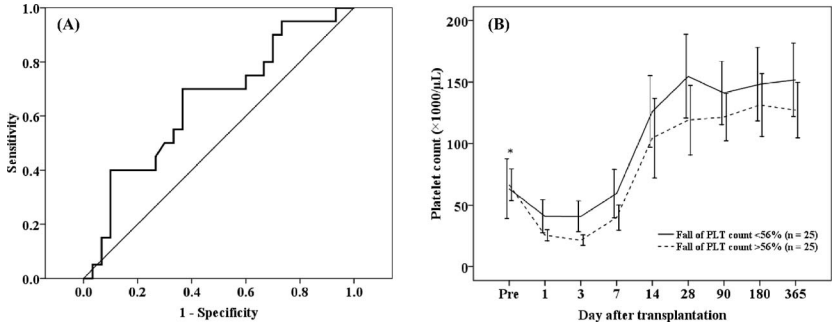

Fig. 3 (A) Receiver operating characteristic (ROC) curve analysis. ROC curve analysis indicated a cutoff value of PLT count fall at POD 3 of 56% for graft dysfunction score of 2 or 3 (sensitivity, 70%; specificity, 63.3%). (B) Evolution of PLT count according to the fall of PLT count at POD 3 >56% or ≤56% up to 1 year after LDLT. The data represent the median values with the 95% CI indicated as described in Fig. 1. The preoperative PLT count did not show a statistically significant difference between the 2 groups (*). The fall of PLT count >56% group showed a less steep increase in the PLT count after LDLT compared with that of the ≤56% group.

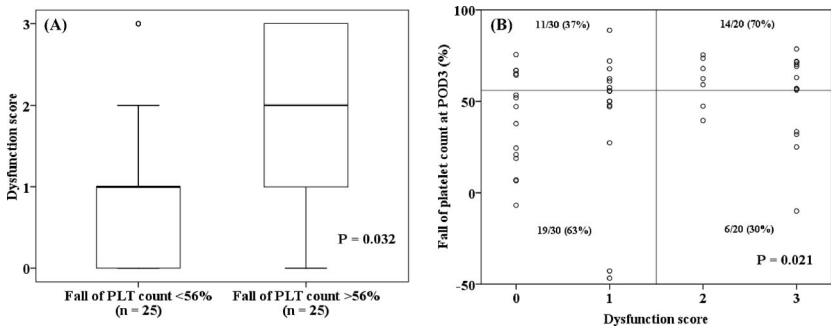

Fig. 4 (A) The dysfunction score in the fall of PLT count at POD 3 >56% or ≤56%. The data are presented as mean ± SD. The dysfunction score was significantly higher in the fall of PLT count >56% group than in the ≤56% group (1.7 ± 1.2 versus 1 ± 1.1; $P = 0.032$). (B) Plot of each percentage fall of PLT count at POD 3 used for the graft dysfunction score ranging from 0 to 3. In the fall of PLT count >56% (n = 25), 14 (56%) patients showed the dysfunction score of 2 or 3. However, in the fall of PLT count ≤56% (n = 25), 6 (24%) patients showed the dysfunction score of 2 or 3 ($P = 0.021$).

thrombopoietin, are also potential causes of thrombocytopenia.[12]

Portal hypertension after reperfusion is observed more commonly in recipients of smaller than of larger grafts.[3,13] It is thought that consumption, or sequestration or entrapment of PLTs into the graft and spleen may be accelerated to a greater extent in SFS grafts in comparison with non-SFS grafts. Marubashi *et al* demonstrated that a high portal venous pressure (≥25 mmHg) and a small graft (graft volume to standard liver volume ratio <0.55) correlated with posttransplantation thrombocytopenia.[14]

Regarding postoperative morbidities within 3 months after LDLT, 19 of 20 patients with a dysfunction score of 2 or 3 showed Clavien grade 2 to 5 complications (Table 2). Infection, intraperitoneal bleeding, and prolonged massive ascites and/or pleural effusion frequently developed as major complications. PLTs are thought to play a key role in antimicrobial host defenses, which inhibit and kill bacterial and fungal pathogens and potentiate the antimicrobial activity of conventional antibiotics.[5] Chang *et al* demonstrated that thrombocytopenia precedes infections and identified patients with nadir PLT counts of $\leq 30 \times 10^3/cm^2$ who were susceptible to early major infections.[5] This persistent thrombocytopenia portended a poor outcome in liver transplant recipients. Other studies report that severe thrombocytopenia may lead to increased morbidity and mortality resulting from bleeding complications during the postoperative period.[15,16]

Table 2 Morbidities within 3 months after LDLT

Clavien classification	Dysfunction score 0 or 1 (n = 30)	Dysfunction score 2 or 3 (n = 20)	*P* value
0–1	17 (57%)	1 (5%)	<0.001
2–5	13 (43%)	19 (95%)	
2	4	3	
3a	6	5	
3b	2	5	
4	0	2	
5	1	4	

Patients with SFS grafts (GRWR <0.8%) may suffer from specific symptoms, including prolonged hyperbilirubinemia, coagulopathy, and massive ascites from so-called small-for-size syndrome (SFSS).[9,10,17,18] The pathogenesis of SFSS involves, in part, portal hypertension resulting from impairment of liver nonparenchymal cells.[13,19] This mechanism also induces PLT activation, aggregation, consumption, and endothelial activation, and consequently, actively contributes to the pathogenesis of graft damage and dysfunction.[11] In the present study, 14 of 20 (70%) patients with a dysfunction score of 2 or 3 had a fall of PLT count at POD 3 >56% in comparison to 11 of 30 (37%) patients with a dysfunction score of 0 or 1, and the dysfunction score of 2 or 3 reflected a dismal outcome.

To relieve portal hypertension leading to graft dysfunction, perioperative splenic artery ligation, embolization, and splenectomy have been used. These procedures will be useful for avoiding or treating SFSS.[20–23]

Thrombotic microangiopathy (TMA) patients develop thrombocytopenia after solid organ transplantation, which is accompanied by hemolytic anemia, and this can result in life-threatening

complications.[24,25] TMA is characterized by microvascular occlusive disorder induced by endothelial damage and primary PLT aggregation. Many studies have focused on a disintegrin-like domain and metalloproteinase with thrombospondin type 1 motifs (ADAMTS)–13, a metalloproteinase produced exclusively in hepatic stellate cells. This enzyme specifically cleaves the highly multimeric von Willebrand factor, which plays a pivotal role in TMA.[26] Ko *et al* reported that a marked decrease of ADAMTS13 activity with concomitant thrombocytopenia was seen in recipients who developed early graft dysfunction in LDLT.[27]

As stated above, the percentage fall of PLT count at POD 3 may be a key clinical manifestation indicating a delayed restoration of graft function and portosystemic circulation as well as the inception of sequelae after LDLT.

In conclusion, the fall of PLT count at POD 3 >56% may be an ominous sign that can predict the graft dysfunction after LDLT in recipients with an SFS graft.

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