The postoperative hepatic artery resistance index after living donor liver transplantation can predict early allograft dysfunction

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Abstract

To investigate whether postoperative hepatic hemodynamics have an impact on graft function.

Using a retrospective cohort with 262 adult living donor liver transplantation (LDLT) recipients, we discussed the relationship between postoperative hepatic hemodynamics and patient outcomes.

According to the definition of early allograft dysfunction (EAD), the patients were classified into the EAD group (43 patients) and the non-EAD group (219 patients). In terms of postoperative hemodynamic parameters, there was no significant differences between these 2 groups regarding hepatic artery flow (HAF), hepatic artery velocity (HAV), portal vein flow (PVF), and portal vein velocity (PVV), except for the hepatic artery resistance index (HARI) which was somewhat higher in the EAD group on postoperative day 3 (POD3) (0.70 vs 0.61, $P < .05$). According to these results, we used a ROC curve and found that a HARI of 0.68 was the cutoff point (with 73.8% sensitivity and 58.3% specificity) for predicting EAD after LDLT. In addition, multivariate analysis showed that fulminant hepatic failure, pretransplant hepatorenal syndrome, and HARI ≥ 0.68 on POD3 were independent risk factors for postoperative EAD.

Our results showed that postoperative hemodynamics might influence graft function by altering hepatic artery flow.

Abbreviations: ALT = alanine aminotransferase, AST = aspartate aminotransferase, CI = confidence interval, EAD = early allograft dysfunction, GRWR = graft-to-recipient weight ratio, HAF = hepatic artery flow, HARI = hepatic artery resistance index, HAV = hepatic artery velocity, HR = hazard ratio, INR = international normalized ratio, LDLT = living donor liver transplantation, LT = liver transplantation, MELD = model for end-stage liver disease, POD = postoperative day, PVF = portal vein flow, PVV = portal vein velocity, ROC = receiver operating characteristic, SFSS = small-for-size syndrome.

Keywords: early allograft dysfunction, hepatic artery resistance index, hepatic hemodynamics, living donor liver transplantation

1. Introduction

Liver transplantation (LT) has been used as the curative option for patients with end-stage liver diseases for the past several decades. However, the scarcity of available organs can hardly reach the demand of patients in need.[1] Living donor liver transplantation (LDLT) is used extensively as a method to alleviate the problem of organ shortage and demonstrate a similar outcome as deceased donor LT.[2] However, some guidelines

defined the living donor as a "marginal donor" due to the insufficient volume of the partial liver that might cause early allograft dysfunction (EAD).[3] EAD is a negative predictor of patient postoperative survival because it leads to eventual graft loss.[4] In fact, EAD is associated with donor aspects, recipient characteristics, and introperative factors.[5]

The postoperative hemodynamics of the graft are related to graft regeneration, liver function recovery, and recipient survival.[6,7] However, whether postoperative hemodynamics have an impact on EAD is not well known. Therefore, we designed a retrospective study using color Doppler flow findings to analyze the effect of hepatic hemodynamic alternation on the development of EAD.

2. Methods

All patients provided signed informed consent before LT. This retrospective study was approved by the ethics committee of West China Hospital.

2.1. Patients

Between January 2002 and February 2018, a total of 356 consecutive patients underwent LDLT at the Department of Liver Surgery, West China Hospital, Sichuan University. Fourteen patients were excluded due to incomplete records, 8 patients for early hepatic artery thrombosis, and 72 patients for being less than 18 years old. Thus, a final population of 262 recipients of LDLT was enrolled. The demographic and clinical data were imported from the Chinese Liver Transplant Registry (CLTR:

http://cltr.cotr.cn) and analyzed among both the donors and the recipients. Recipient etiology, pretransplant complications, and peri-transplant conditions, such as ischemia time, blood loss, operative time, portal flow modulation, and renal replacement therapy, were described. All operations were performed by experienced teams on patients under general anesthesia.

2.2. Definition of early allograft dysfunction

The definition of EAD was the presence of 1 or more postoperative laboratory values, which are as follows: serum levels of alanine aminotransferase (ALT) or aspartate amino-transferase (AST) > 2000 U/L within the first week, total bilirubin $\geq$ 10 mg/dl or international normalized ratio (INR) $\geq$ 1.6 on postoperative day (POD)7.[8] According to this definition, the patients were classified into 2 groups: EAD and non-EAD groups.

2.3. Postoperative Doppler ultrasound examinations

During the first 7 days after LDLT, routine duplex ultrasonography (Aloka Co., Ltd., Japan) was performed daily. The site of measurements for the proper hepatic artery was the point adjacent to the hepatic hilum, and the site of measurements for the portal vein was the maximum diameter near the anastomosis for the portal vein. Every examination was repeated 3 times, and the maximum value was recorded. The following characteristics of Doppler imaging of the portal vein and hepatic artery were recorded:

1. diameter;
2. portal vein blood flow (PVF, ml/minute) and hepatic arterial blood flow (HAF, ml/minute);
3. portal vein peak velocity (PVV, cm/second) and hepatic artery peak systolic velocity (HAV, cm/second);and
4. HARI.

The HARI calculated as follows: (peak systolic velocity-peak end diastolic velocity)/ (peak systolic velocity).

2.4. Post-transplant follow-up

The recipient was followed up weekly during the first month after hospital discharge. If there were no abnormal findings, then the recipient would be followed up every 3 months. All patients were given immunosuppressive drugs, such as tacrolimus, mycophe-nolate mofetil, prednisolone, and basiliximab based on the institutional immunosuppression schedule.

2.5. Statistical analyses

SPSS 22.0 statistical software was used to perform all analyses in this study. $P < .05$ was considered statistically significant. Repeated measure analysis of variance and t-test were used to compare continuous variables. Pearson Chi-Squared test or Fisher exact test was used to compare categorical data. The receiver operating characteristic (ROC) curve was plotted to examine the predictive ability of HARI in EAD. The optimal cutoff value was set as the value of the Youden index, which was the maximizing sum of sensitivity and specificity. To acquire the prognostic factors for survival, univariate, and multivariate analyses were performed using the Cox proportional hazard model and were expressed as a hazard ratio (HR) with a 95% confidence interval (CI). The graft and patient survival curves were plotted using the Kaplan–Meier method and were compared using the log-rank test.

3. Results

3.1. Participant characteristics

A total of 262 LDLT recipients were included in this period, of which 214 were men and 48 were women. The average transplant age was 42.7 ($\pm$8.4, range 16–65) years old. The pretransplant etiologies included 114 with hepatocellular carcinoma, 79 with hepatitis B cirrhosis, 43 with fulminant liver failure, 4 with Budd-Chiari syndromes, 14 with cirrhosis and hepatitis C virus, and 8 with other diseases. A total of 243 (92.7%) recipients received right lobe liver grafts without the middle hepatic vein, 13 (4.9%) recipients received right lobe liver grafts with the middle hepatic vein and 6 (2.2%) recipients received a left liver lobe.

According to the definition of EAD, there were 43 (16.4%) patients with EAD as the EAD group, and another 219 (83.6%) patients without EAD as the non-EAD group. The parameters of these 2 groups are listed in Table 1. There was no significant difference in donor factors between the 2 groups. In terms of the recipient factors, the MELD score was 16.8 (10–31) in the EAD

Table 1

Baseline characteristics of patients with or without EAD after LDLT.

Clinical characteristics	EAD (N=43)	Without EAD (N=219)	P value
Donor factors			
Age (years), mean (SD)	37.2 (11.6)	36.4 (10.2)	.616
Sex (male/female)	28/15	128/91	.498
BMI (kg/m^2), mean (SD)	23.8 (2.8)	23.2 (2.5)	.208
HBcAb positive (%)	6 (13.9%)	44 (20.1%)	.404
GRWR (%), mean (SD)	0.95 (0.21)	0.91 (0.19)	.405
Graft weight(g), mean (SD)	554 (112.1)	584.4 (105.2)	.648
Recipients factors			
Age (years), mean (SD)	43.4 (9.4)	42.6 (8.5)	.584
Sex (male/female)	32/11	182/37	.583
BMI ((kg/m^2), mean (SD)	22.8 (3.2)	23.2 (2.6)	.836
HBsAg positive (%)	33 (76.7%)	162 (73.9%)	.849
HBeAg positive (%)	12 (27.9%)	45 (20.5%)	.075
HCV-Ab positive (%)	4 (9.31%)	10 (4.57%)	.266
MELD score, median (SD)	16.8 (8.9)	12.2 (9.5)	.02
Cirrhosis (%)	32 (74.4%)	169 (77.2%)	.183
Medical condition			
Cold ischemia time (minutes), median (SD)	85 (100.4)	76 (88.2)	.609
Operation time (minutes), median (SD)	675 (185.6)	640 (154.8)	.186
Blood loss (ml), median (SD)	2000 (1200)	1500 (1400)	.196
PRBCs transfusion (U), median (SD)	8 (6.2)	6 (5.4)	.246
Plasma transfusion (mL), median (SD)	1800 (1100)	1000 (1200)	.01
Portal flow modulation (%)	7 (16.2%)	19 (8.6%)	.127
Post-reperfusion syndrome	12 (27.9%)	39 (17.8%)	.096
In ICU (%)	3 (6.9%))	8 (3.7%)	.415
Fulminant hepatic failure	15 (34.9)	28 (12.8%)	<.001
SFSS (%)	6 (13.9%)	13 (5.9%)	.062
Renal replacement therapy (%)	14 (32.6%)	22 (10.4%)	<.001
Pre-transplant complications			
GI bleeding (%)	2 (4.7%)	18 (8.2%)	.545
Peritonitis (%)	2 (4.7%)	14 (6.4%)	.494
Uncontrolled ascites (%)	3 (6.9%)	42 (19.2%)	.067
pretransplant hepatorenal syndrome	8 (18.6%)	17 (7.8%)	.042
Encephalopathy (%)	7 (16.3%)	18 (8.2%)	.592

BMI = body mass index, EAD = early allograft dysfunction, GI = gastrointestinal bleeding, GRWR = graft-to-recipient weight ratio, HBcAb = hepatitis B c antibody, HBeAg = hepatitis B e antigen, HBsAg = hepatitis B surface antigen, HCV-Ab = hepatitis C antibody, ICU = intensive care unit, LDLT = living donor liver transplantation, MELD = model for end-stage liver disease, PRBCs = packed red blood cells, SD = standard deviation, SFSS = Small-for-size syndrome.
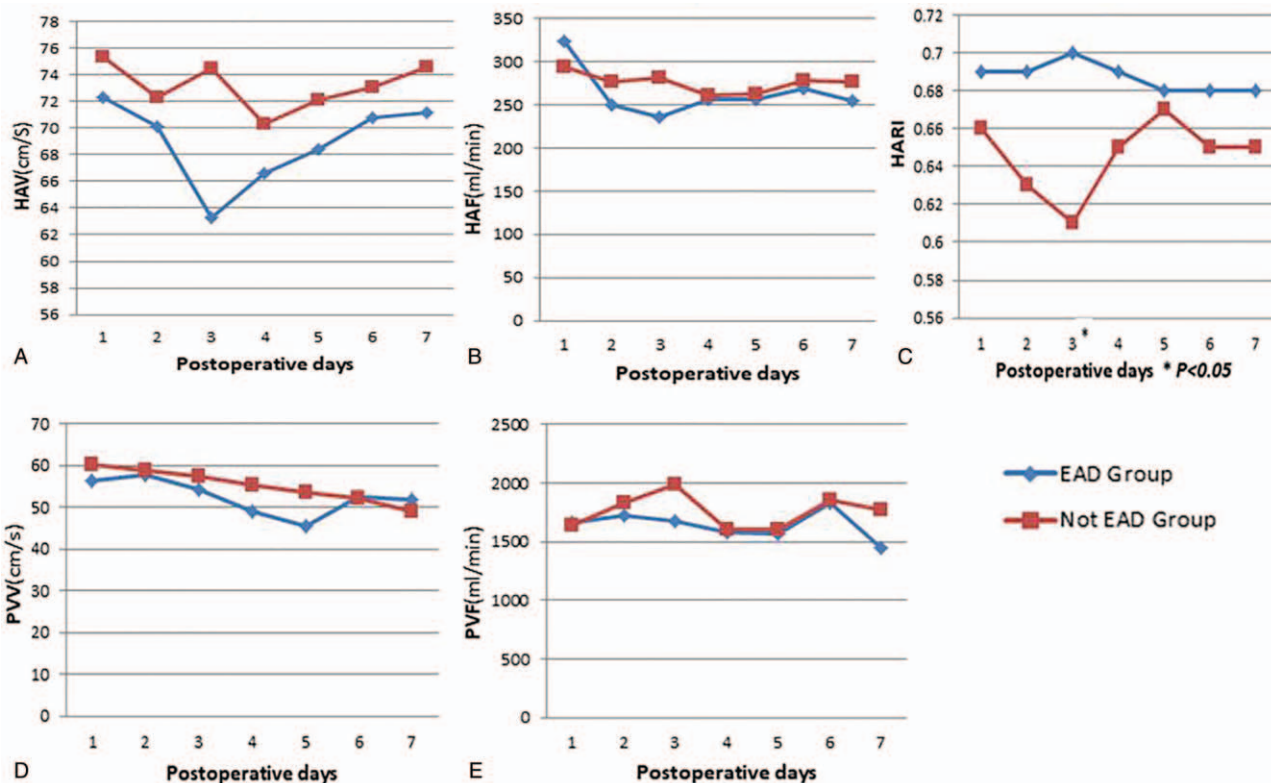
Figure 1. Continuous hepatic hemodynamic change in the 2 groups in the first week after living donor liver transplantation. (A HAV; B HAF; C HARI; D PVV; E PVF). HAF = hepatic artery flow, HARI = hepatic artery resistance index, HAV = hepatic artery velocity, PVF = portal vein flow. PVV = portal vein velocity. $^{*}P < .05$.

group compared to 12.2 (9–18) in the non-EAD group ($P < .05$). For the preoperative conditions, the presence of fulminant hepatic failure was significantly higher in the EAD group than in the non-EAD group (34.9% vs 12.8%, $P < .001$). In addition, the presence of pretransplant hepatorenal syndrome was 18.6% in the EAD group and 7.8% in the non-EAD group ($P = .042$). Additionally, plasma transfusion was higher in the EAD group (1800 ml) than in the non-EAD group (1000 ml). There was a significant association ($P < .001$) of EAD with renal replacement therapy (10.4%) when compared to that of non-EAD with renal replacement therapy (32.6%). No significant difference was observed regarding the other analyzed characteristics.

3.2. Hemodynamic changes

The intrahepatic hemodynamic changes in the graft were shown by averaging the values (Fig. 1). There was a significant difference in HARI on POD3 between the EAD group and the non-EAD group (0.70 vs 0.61, $P < .05$). These 2 groups did not differ with

regard to HAV, HAF, PVV, PVF, or other hemodynamic parameters. The mean HARI of the entire population ranged from 0.61 to 0.70 during the first week after transplantation (Table 2). The HARI in the EAD group remained within the higher level (range, 0.68–0.70), which reached its highest point on POD3 when it was significantly higher than that in the non-EAD group. According to the results, the ROC curve was used to analyze the cutoff point of HARI in these 2 groups. The results showed that a cutoff point for HARI (with 73.8% sensitivity and 58.3% specificity, Fig. 2) was found to predict EAD after LDLT. Patients with HARI ≥ 0.68 on POD3 have a higher risk of EAD.

3.3. Risk factors for EAD

In these 2 groups, the univariate analysis showed that 5 variables had a significant association with the development of postoperative EAD: postoperative HARI ≥ 0.68, fulminant hepatic failure, plasma transfusion, MELD score, and pretransplant hepatorenal syndrome (Table 3). Furthermore, we used a multivariate logistic

Table 2

HARI Between patients with or without EAD after liver transplantation.

	POD1	POD2	POD3	POD4	POD5	POD6	POD7
Without EAD	0.66	0.63	0.61	0.65	0.67	0.65	0.65
EAD	0.69	0.69	0.70	0.69	0.68	0.68	0.68
P-value	0.378	0.186	0.047*	0.218	0.634	0.309	0.656

EAD = early allograft dysfunction, HARI = hepatic artery resistance index, POD = postoperative day.
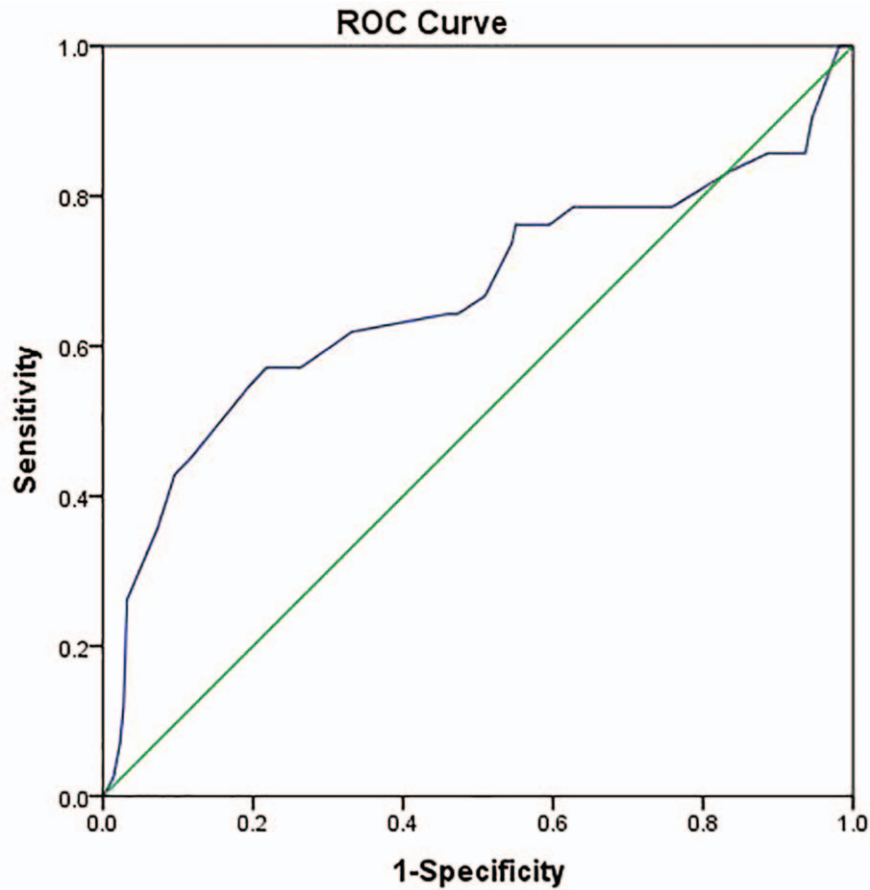
Figure 2. ROC curve for HARI on POD3 associated with postoperative early allograft dysfunction (area under the curve = 0.665, $P < .001$). A HARI of 0.68 was the most accurate cutoff value with the highest Youden index (Youen index = 0.319, sensitivity = 0.738, specificity = 0.583) for predicting postoperative early allograft dysfunction. ROC = the receiver operating characteristic.

regression model including these significantly different variables to identify whether HARI $\geq$ 0.68 on POD3 is an independent risk factor for EAD. The results showed that fulminant hepatic

Table 3

Univariable analysis comparing baseline clinical characteristics.

Variables	OR	95% CI	P value
Donor factors			
Sex male	0.76	0.37–1.63	.498
HBcAb positive	0.66	0.26–1.86	.404
Recipients factors			
Sex male	0.50	0.20–1.29	.583
MELD score	2.64	1.52–3.21	.02
HBsAg positive	1.86	0.42–2.78	.849
HBeAg positive (%)	1.09	0.56–2.36	.075
Cirrhosis	0.47	0.26–1.07	.183
Medical condition			
Cold ischemia time	1.1	0.94–1.30	.99
Fulminant hepatic failure	5.35	2.24–12.6	<.001.
Plasma transfusion	2.86	1.64–3.78	.034
Portal flow modulation	0.51	0.17–1.26	.072
Pretransplant hepatorenal syndrome	5.34	1.71–15.6	.042
HARI on POD3 $\geq$0.68	3.86	1.54–8.57	<.001

EAD = early allograft dysfunction, HARI = hepatic artery resistance index, HBcAb = hepatitis B c antibody, HBeAg = hepatitis B e antigen, HBsAg = hepatitis B surface antigen, POD = postoperative day.

failure, HARI$\geq$0.68 on POD3 and pretransplant hepatorenal syndrome were independent risk factors for postoperative EAD (Table 4).

3.4. Graft and patient survival

Kaplan–Meier curves were plotted to show the graft survival and patient survival of these 2 groups (Figs. 3 and 4). There were significant differences in graft survival and patient survival between the EAD group and non-EAD group ($P < .001$). The 12-month mortality of these groups was significantly different: 23.2% (10/43) of the recipients died in the EAD group as opposed to 11.8% (26/219) in non-EAD group. The primary causes of death in patients were graft failure, sepsis, and malignancy.

Table 4

Multivariate analysis of independent risk factor for EAD.

Variables	Odds ratio	95% CI	P value
HARI on POD3$\geq$0.68 vs <0.68	2.86	1.22–6.82	.016
Fulminant hepatic failure (yes)	3.63	1.54–8.60	.004
Pretransplant hepatorenal syndrome (yes)	3.65	1.08–12.3	.003

EAD = early allograft dysfunction, HARI = hepatic artery resistance index, POD = postoperative day.
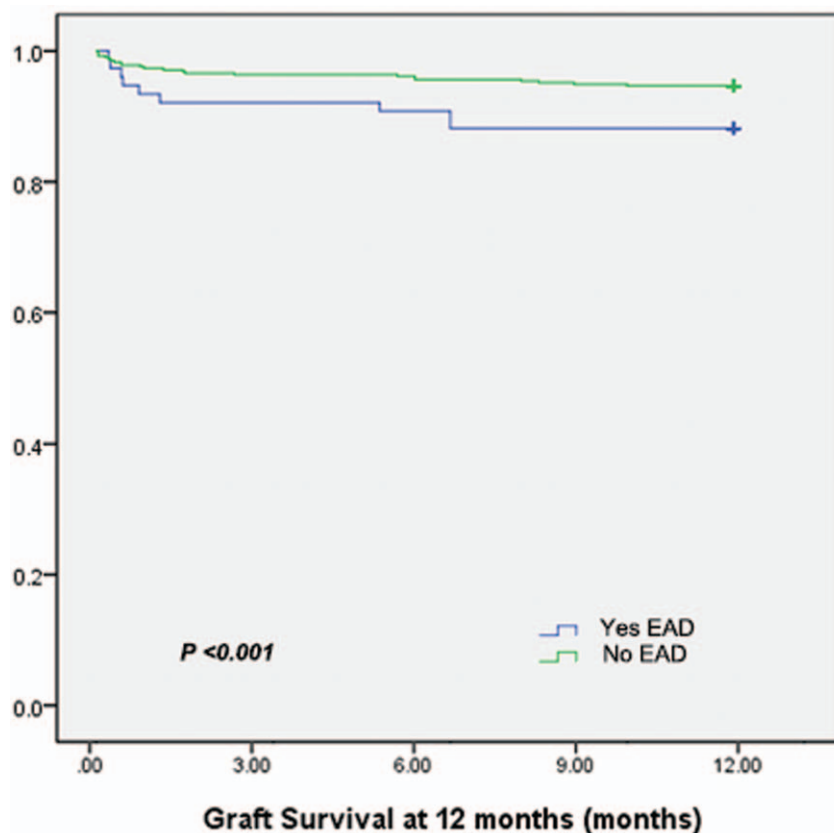
Figure 3. Kaplan–Meier curves for graft survival in the first year after living donor liver transplantation.

4. Discussion

In this study, we demonstrated the tendency and range of hepatic hemodynamics parameters of recipients in the first week after LDLT and investigated the effect of hepatic hemodynamic alternation on the development of EAD. The results reveal that postoperative hepatic hemodynamics were associated with the development of EAD after LDLT: patients with relatively high HARI on POD3 were significantly more likely to develop EAD. In addition, we found that fulminant hepatic failure, HARI $\geq$ 0.68 on POD3 and pretransplant hepatorenal syndrome were independent risk factors for postoperative EAD.

The present study demonstrated the interaction between recipient characteristics, donor characteristics and intraoperative factors associated with EAD. These risk factors include donor steatosis, amount of blood transfused, combined organ transplantation, and MELD score.[9–12] In our study, we found that fulminant hepatic failure, MELD score, hepatorenal syndrome, and plasma transfusion were significantly related to the development of EAD. These factors were primarily associated with the severity of liver fibrosis caused by hepatitis B or hepatitis C infection, and have been systematically analyzed in previous studies.[8,13] However, few studies have evaluated the effect of postoperative graft hemodynamics on liver function. Therefore, we compared postoperative graft hemodynamics between EAD and non-EAD patients in this study. As mentioned above, we found that HARI on POD3 was associated with the occurrence of EAD after LDLT. Patients with relatively high HARI were

significantly more likely to develop EAD than those with lower HARI levels. In addition, the logistic regression analysis showed that HARI $\geq$ 0.68 on POD3 was an independent risk factor for postoperative EAD. In this study, there was no significant difference in the occurrence of SFSS between the 2 groups. One of the reasons is that the mean graft-to-recipient weight ratio (GRWR) reached 0.95% in EAD group and 0.91% in non-EAD group (Table 1), resulting in a lower overall incidence of Small-for-size syndrome (SFSS) comparing with previously published reports (7.2% vs 8%–13%).[14,15] It was difficult to find a significant difference with such a low incidence of SFSS.

The characteristics of hepatic hemodynamics were different before and after transplantation, and a significant increase in the total hepatic flows was observed in both cadaver and live donor grafts.[16,17] The arterial supply is essential for graft function. For example, hepatic artery thrombosis after liver transplantation can cause early loss of the graft and retransplantation or the death of the recipient. In other cases, recipients with acute histopathological rejection showed a continuously high incidence of arterial complications. However, the relationship between hepatic hemodynamics and graft function recovery has not been thoroughly investigated in LT. Accumulating evidence from clinical studies has indicated that hepatic artery flow can improve liver function by mediating of liver regeneration.[18] In addition, other studies have also proven that increasing portal vein flow can stimulate regeneration after liver resection in rats.[19] These phenomena showed that hemodynamics is significantly associated with graft regeneration and that hepatic inflow has some degree of predetermined relationship with EAD.
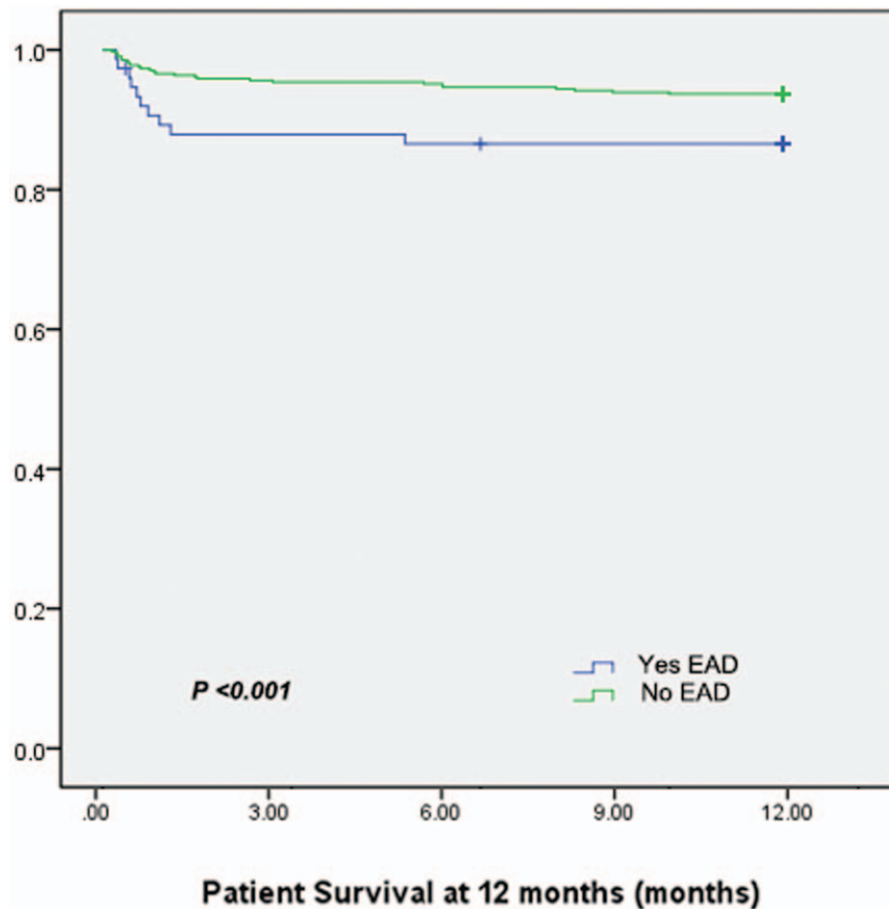
Figure 4. Kaplan–Meier curves for patient survival in the first year after living donor liver transplantation.

Some studies have demonstrated that HARI has a positive relationship with the degree of liver fibrosis: patients with severe liver fibrosis have a higher level of HARI, which may result in increased flow resistance and arterial rigidity.[20] Other studies have found that HARI may be influenced by the different tissue composition of the liver in different hepatic disorders. A study with a small number of pediatric liver transplantation patients found that HARI was associated with biliary complications: patients with HARI < 0.57 were more likely to develop biliary complications.[20,21] In our study, marked differences in hemodynamic values were observed only at POD3. We propose that there are several possible reasons for this consequence: in the early postoperative period, there is hemodynamic instability caused by anesthesia, transplantation, and venous transfusion, which may alter hepatic hemodynamics; and in the late postoperative period, various regulatory mechanisms contribute to the control of hepatic flow, which makes it difficult to find a significant difference caused by 1 single factor. As mentioned above, the reasons for EAD remain unclear. It is difficult to decipher whether the marked difference in hepatic hemodynamics observed in the EAD group is a cause or a result of EAD. In terms of timing, the alteration of hepatic hemodynamics occurred at POD3 before developing EAD, which indicates that this factor may be a cause of EAD.

The primary purposes of studies on EAD are to establish a reliable index for the early detection of acute rejection and primary graft failure. Using the current results, numerous techniques have proven to be effective for the early detection of EAD after liver transplantation. Recent studies have found that the indocyanine green disappearance rate after reperfusion and postoperative MELD appropriately predicted EAD.[22] Additional techniques include the use of sequential organ failure assessment scores and MELD-lactate values.[10,23] Compared with these methods, using HARI is a cost-effective, noninvasive, and accessible method to predict EAD. Thus, clinicians can more readily detect alterations at the bedside of the patient.

We found several limitations to our study. First, the nature of the retrospective study reduces the strength of our hypothesis. Second, blood circulation state, hemodynamic detection bias, and perioperative treatment might influence the outcome. Third, we lacked pathologic data regarding donor graft steatosis and fibrosis, which has been shown to be related to the risk of developing EAD after LDLT. Fourth, we lacked preoperative, intraoperative, and long-term hepatic hemodynamic values for comparison of patients with and without EAD after LDLT.

In conclusion, our study found that HARI ≥ 0.68 on POD3 was an independent risk factor for EAD in adult LDLT patients. Postoperative hemodynamics might influence graft function by altering hepatic artery flow. However, further research is needed to explore the mechanism of the effect of hemodynamics on graft function.

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