HHS Public Access

Author manuscript

Liver Transpl. Author manuscript; available in PMC 2016 September 01.

Published in final edited form as:

Liver Transpl. 2015 September ; 21(9): 1179–1185. doi:10.1002/lt.24166.

Acute Kidney Injury after Orthotopic Liver Transplantation Using Live Donor versus Cadaveric Donor Grafts: a Propensity Score-Matched Analysis

Ibtesam A. Hilmi, MBCHB, FRCA, **Daniela Damian, MD**, **Ali Al-Khafaji, MB, CHB**, **Tetsuro Sakai, MD, PhD**, **J. Donaldson, MS**, **Daniel G. Winger, MS**, and **John A. Kellum, MD**

Abstract

Introduction—Acute kidney injury (AKI) is a common complication after liver transplantation (LT). Few studies investigating the incidence and risk factors for AKI after live donor LT (LDLT) have been published.

Hypothesis—LDLT recipients have a lower risk for post-LT AKI than cadaveric donor LT (CDLT) recipients due to higher quality liver grafts.

Methods—We retrospectively reviewed LDLTs and CDLTs performed at the University of Pittsburgh Medical Center between Jan. 2006 and Dec. 2011. AKI was defined as a 50% increase in serum creatinine (SCr) from baseline (preoperative) values within 48 hours (1). One hundred LDLT and 424 CDLT recipients were included in the propensity score matching logistic model based on age, gender, MELD score, Child score, pre-transplant SCr, and pre-existing diabetes mellitus. Eighty-six pairs were created after one-to-one propensity-matching. The binary outcome of AKI was analyzed using mixed effects logistic regression, incorporating the main exposure of interest (LDLT versus CDLT) with the aforementioned matching criteria and post-reperfusion syndrome, number of units of packed red cells, and donor age as fixed effects.

Results—In the corresponding matched dataset, the incidence of AKI at 72 hours was 23.3% in the LDLT group, significantly lower than 44.2% in the CDLT group (p=0.004). Multivariable mixed effects logistic regression showed that live donor liver allografts were significantly associated with reduced odds of AKI at 72 hours post-LT (p=0.047, OR=0.307; 95% CI 0.096–0.984). The matched patients had lower body weights, better-preserved liver functions, and more stable intraoperative hemodynamic parameters. The donors were also younger for the matched patients than for the un-matched patients.

Conclusion—Receiving a graft from live donor has a protective effect against early post-LT AKI.

Introduction

Live donor liver transplantation (LDLT) has expanded, aiming to both increase the donor pool and provide excellent grafts from healthy donors with short cold ischemia time. The

Dr. Hilmi, Ibtesam, Presbyterian Hospital, 200 Lothrop Street, Pittsburgh, Pennsylvania, United States, 15312, hilmiia@anes.upmc.edu.

disadvantage of this procedure is that it puts two individuals at risk; it is important that both procedures proceed without serious perioperative complications. One of the most serious and common post-liver transplantation (LT) complications is the development of acute kidney injury (AKI), which can adversely affect patient and graft outcomes. AKI has been studied extensively in cadaveric donor liver transplant (CDLT) recipients, but not LDLT recipients. In this study, we investigated and compared the risk factors and incidence of AKI between living donor partial liver transplant recipients and cadaveric donor whole liver transplant recipients.

Methods

After approval from the University of Pittsburgh Institutional Review Board (PRO10050135), we retrospectively analyzed the data of LDLT and CDLT recipients over a six-year period (January 2006–December 2011). The LDLT group included adults with end-stage liver disease (ESLD) who received right lobe partial liver allografts from live donors. Patients with fulminant liver failure or history of previous LT were excluded from the study, as well as patients with chronic renal failure on dialysis and patients who died within the first 72 hours post-LT. We collected data at three time points: the preoperative, intraoperative, and postoperative periods. The preoperative and intraoperative data were used to predict AKI, while the postoperative data was used to define the endpoint (development of AKI) and outcome analysis. The following preoperative data were included: patient demographics, etiology of ESLD, Model for End-Stage Liver Disease (MELD) score, Child score, eGFR by using the Modification of Diet in Renal Disease (MDRD) equation (2), SCr, and preoperative co-morbidities. The intra-operative data were: duration of surgery, utilization of veno-venous bypass (VVBP), use of methylene blue and aprotinin, and cold and warm ischemia times. Hemodynamic parameters (systemic blood pressure and heart rate) and the use of vasopressor agents were used to document the development of postreperfusion syndrome (PRS). Volume and type of blood products, crystalloids, and colloids used were included in the analysis. Postoperative data included daily SCr for the first 72 hours post-LT, which was used to define post-LT AKI, as well as one-year patient and graft survivals.

AKI was defined according to the most recent definition: a 50% increase in SCr from baseline (pre-transplant value) within 48 hours without urine output (1). A modified definition of PRS was used, defining PRS as the presence of hemodynamic instability with persistent hypotension (< 30% of the anhepatic level) requiring continuous vasopressor support intraoperatively and/or post-operatively (3). Of note and as the standard of care, all patients were optimized in cardiovascular, hematocrit, and acid-base balance prior to reperfusion. Patients were followed for one-year from the date of their transplant surgery. The main outcome was 72 hours post-LT AKI with possible pre-operative and intraoperative factors associated with AKI; secondary outcomes were one-year patient and graft survivals. To compare incidence of AKI between the LDLT and CLDT groups, propensity score matching was used to substantially reduce allocation bias. The only factor that determines whether a patient will receive a graft from a live donor versus a cadaveric donor is the availability of such a donor; however, in our study, LDLT recipients were younger and had lower MELD scores than the CLDT recipients. For this reason, we included age, gender, and

MELD score in the propensity matching scoring together with preoperative SCr value and pre-existing diabetes mellitus because of the possible consequences of such factors on the incidence of post-LT AKI. We did not include intraoperative variables for the propensity matching process despite significant differences, since these variables are not determined before transplantation. However, the intraoperative variables were used to predict the development of post-LT AKI.

Statistical analysis

The statistical analysis was performed using Stata Statistical Software: Release 12 (College Station, TX: StataCorp LP), along with psmatch2 command for propensity score matching (4). Pearson's chi-square test was used to compare the categorical variables; for normally distributed continuous variables, the un-paired Student's t-test (presented as mean ± standard deviation) was used. If normality was violated, the Mann-Whitney U test was used (presented as median with interquartile range). A p value of <0.05 was considered statistically significant. Prior to examining the effects of liver graft type, univariate and multivariable logistic regressions were performed to identify independent risk factors for AKI. The p-value for inclusion in the multivariable logistic regression was 0.1. Log Rank (Mantel-Cox) and Kaplan- Meier survival curves were used to analyze liver graft and patient survival.

For propensity score matching, we generated propensity scores for the binary exposure of "receiving a transplant from a live donor" (vs. cadaveric, or unexposed). The logistic model for this process included MELD score, SCr, age, gender, and pre-LT diabetes mellitus as variables in the propensity score generation process. One-to-one optimal matches were assigned without replacement using a caliper of 0.05 maximum distances between propensity scores. A matched dataset of n=172 (86 pairs of exposed and unexposed) was established. In order to examine how greatly this matched dataset differed from the general patient population, these 172 patients were compared to the 352 patients (14 from the LDLT group and 338 from the CDLT group) who were not matched. The nonparametric Mann-Whitney U test was used to compare continuous variables and chi-squared tests were used to compare categorical variables.

Finally, using the propensity-matched dataset of 86 pairs, we ran mixed effects logistic regression to predict the binary outcome of AKI. The main exposure of interest (live vs. cadaveric donors) and several patient variables (gender, weight, MELD score, Child score, pre-LT SCr, pre-existing diabetes mellitus, PRS, number of packed red blood cell units, and donor age) were incorporated as fixed effects. A random effects intercept for each pair was used along with clustered robust standard errors, which accounts for the paired structure of the dataset. Log-rank test and Kaplan-Meier survival curves were used to analyze liver graft and patient survivals.

Results

A total of 107 LT patients received liver grafts from live donors. Seven patients were excluded, six were on pre-transplant dialysis, and one patient was re-transplanted with a

cadaveric graft in the first 24 hours post-LT due to primary graft failure. The etiology of liver failure was 20% non-alcoholic steatohepatitis, 11% alcoholic cirrhosis, 24% hepatitis C cirrhosis, 31% biliary cirrhosis, and miscellaneous etiologies in 14% of the group. Nineteen percent (19%) of patients in both groups had eGFR >60ml/min/1.73 m^2 as calculated by the MDRD equation. Twenty-one percent (21%) of the 100 patients developed AKI within the first 72 hours post-LT. The AKI and non-AKI groups' preoperative and intraoperative data with donor characteristics are listed in Table 1. The development of PRS, non-use of VVBP, and number of units of platelets significantly impacted the development of post-LT AKI (p= 0.009, 0.045, and 0.014, respectively).

Prior to examining the effects of liver graft type, multivariable logistic regression was used to identify possible preoperative, intra-operative, and donor factors associated with AKI at 72 hours post-LDLT. In the multivariable logistic model, an entry p value of <0.1, MELD score, PRS, use of VVBP, and number of platelet units were used to fit a forward stepwise procedure. In the resulting model, only severe PRS remained significantly associated with developing AKI within 72 hours post-LT with an odds ratio (OR) of 4.9 and a 95% confidence interval (CI) of 1.3–17.2 (p=0.014).

LDLT and CLDT recipients' pre-operative and intraoperative variables before propensity score matching are illustrated in Table 2. The incidence of AKI at 72 hours post-LT before propensity score matching was 21% in the LDLT group and 52.1% in the CDLT group (p <0.001). There were significant differences between four of the variables used for the propensity score matching (age, MELD score, gender, and pre-transplant SCr). The presence of pre-transplant diabetes mellitus was evenly distributed between the two groups. After the matching process, no differences were found between the selected 86 pairs (Table 3). The matching process reduced the overall mean bias from 46.7 to 4.5, and the differences between the two groups, based on the variables listed on Table 3, became statistically insignificant (p=0.987). After propensity score matching, the LDLT and CDLT recipients' incidence of AKI at 72 hours post-LT statistically significantly differed: 23.3% and 44.2% respectively, with p=0.004. The mixed effects logistic regression analysis incorporated our main exposure of interest (live donor versus cadaveric donor liver grafts) along with covariates, which were found to be statistically linked to AKI and were cited previously in the literature as being associated with AKI. Those additional covariates included: patient age, gender, weight, MELD score, Child score, pre-transplant SCr, pre-existing diabetes mellitus, PRS, and number of PRBC units, platelets, and fresh frozen plasma administered. In the full multivariable model, only liver graft type was significantly associated with AKI at 72 hours post-LT (Table 4). More specifically, receiving a liver from a live donor had a protective effect against AKI in the first 72 hours post-LT with OR=0.307 (95% CI 0.095–0.98; p=0.047).

In order to determine how greatly this propensity-matched dataset of 172 patients differs from the general patient population, the variables used in the final mixed effects logistic regression model were compared between the matched pairs (86 LDLT+86 CDLT) and the remaining un-matched patients (14 LDLT+338 CDLT) (Table 5). The matched set of patients weighed less, had better-preserved liver functions, had less eventful intraoperative courses, and had younger donors than the rest of the un-matched patients.

Overall one-year survival of the LDL grafts and the CLD grafts after propensity score matching showed no statistically significant difference (p=0.613). Recipients' one-year survivals based on type of liver graft after propensity score matching did not differ (p=0.505).

Discussion

LDLT was developed to expand the critical organ pool and reduce the prolonged waiting period or death on the waiting list before receiving the appropriate liver allograft. The advantage of LDLT, apart from being an elective well-planned procedure, is that an organ taken from a healthy volunteer comes with a very short cold ischemia time and hopefully with less ischemic insult to the graft. However, because the LDLT liver is a partial graft, recipients have an increased incidence of certain complications such as bile leak, stricture of the bile duct, vascular thrombosis, and small-for-size (SFS) syndrome (5).

The elective nature of the procedure and excellent donor criteria raise the expectation that such a procedure will improve post-operative renal function and will significantly reduce the incidence of this serious complication (6). In this study, the incidence of AKI was still found to be high and occurred in 23.3% of the LDLT recipients within the first 72 hours post-LT; however, this is still significantly less than that of their counterpart standard CDLT recipients (44.2%). A study by Lin *et al.* (7) reported a comparable incidence of AKI (36.2%) in their LDLT recipients during the first three months post-LT, which shows that AKI in this group is not an uncommon occurrence. The development of SFS grafts can have detrimental effects on post-LT renal functions in LDLT recipients (8). Lee *et al.* (9) reported that the development of SFS syndrome was a significant risk factor for post-LT AKI. None of the 100 patients in our study group developed SFS syndrome; this may be explained by the strict rule of utilizing ≥ 800 grams from the live donor liver and possibly low-risk recipients. The same study by Lee *et al.* concluded that SFS syndrome, MELD score, and pre-LT renal function were significant risk factors for postoperative AKI in LDLT recipients, conclusions that we could not prove in our study. One of the reasons for arriving at different conclusions can be explained by the use of different definitions of AKI. Lee *et al.* used the International Ascites Club's Diagnostic Criteria, which defines AKI as SCr level > 1.5 mg/dL at three months following liver transplant. MELD score was identified as a risk factor for post-LT AKI (10) in multiple previous studies, but in our study, MELD score was not a risk factor, which can be explained by the fact that our patient population was very homogenous and with very comparable MELD scores (MELD scores for the whole cohort were 13.3±5.05). However, Selzner *et al.* (11) also found that MELD score was not a risk factor for post-LT AKI in LDLT recipients. In our study, the univariable analysis of the risk factors for post-LT were number of platelet units transfused, the development of PRS, and non-use of VVBP. Overall, the transfusion of blood products seemed to be a strong marker for number of complications and can affect overall survival following LT (12). Although the mechanism of injury is still not fully understood, blood transfusion had been identified as a risk factor for AKI in many previous studies (13). In particular, platelets may have non-hemostatic properties, which can contribute to ischemia/reperfusion injury of the graft via induction of sinusoidal endothelial cell apoptosis (14). By the same token, platelets can induce vascular endothelial dysfunction with end-organ damage, which has a reach far

beyond the liver allograft. VVBP was implemented to improve hemodynamic stability and renal perfusion and may lower the incidence of post-LT AKI (15). This was clear in the LDLT recipients, but the use of VVBP had no impact on the incidence of post-LT AKI in the CDLT recipients (16). These conflicting results may be related to the study group's characteristics or graft-specific factors. The last factor which impacted the incidence of post-LT AKI in the LDLT recipients was the development of PRS. PRS is the only risk factor for post-LT AKI that remained significant in multivariable regression analysis. The presence of hemodynamic instabilities and the requirement of vasopressor agents may compromise renal perfusion and can result in renal injury with post-LT AKI. PRS was not significant as a factor in post-LT AKI in the CDLT recipients (16), which may be related to better preload optimization in this group than in the LDLT group. Intraoperative volume restriction in the LDLT recipients prevents congestion of the partial liver allografts, in particular during the neohepatic phase, a rule which does not apply to the full cadaveric liver allografts.

We demonstrated that LDLT recipients have a lower risk for post-LT AKI than CDLT recipients. The incidence of AKI in the 100 LDLT recipients was 21%, which is significantly lower than the incidence of 52% in the 424 CLDT recipients. However, since the two groups were very heterogeneous with different patient demographics, severity and etiology of ESLD, and levels of preoperative renal function, we decided to use the propensity match score test to homogenize the groups and minimize bias in the selection process. Interestingly, the incidence of AKI in the LDLT recipients remained significantly lower (23.3%) than the incidence in the CLDT recipients (44.2%), suggesting that the type of the graft was the only factor that contributed to this difference. To our knowledge, the use of propensity match score to investigate and accurately compare the incidences of AKI in LDLT recipients and CDLT recipients has not been reported in previous publications. Our interesting findings came from the survival analysis; one-year patient and graft survivals did not differ between the LDLT group and the corresponding matched-control group. This may have resulted from the fact that these matched groups were very similar in all aspects (preoperative variables and patient demographics), which placed them in the low risk category for postoperative mortality and graft loss. When the survival analysis based on the development of post-LT AKI in the first 72 hours was conducted, the differences in graft and patient survival between these matched groups were not statistically significant. The preoperative parameters (Table 3) for the matched groups (172 patients) were superior in most aspects to those of the remaining un-matched group (352 patients), which probably influenced the outcome results in a favorable way. The selection of candidates to receive LDL grafts at our center is not entirely based on the availability of the donor but the inclusion of low-risk criteria candidates; this factor is very important to ensure successful outcomes. These general rules include low MELD scores (in the 20s) and absence of comorbidities (cardiac, renal, or pulmonary diseases), as well as recipient age (maximum age: early 60s).

Our study had some limitations. First, the analysis was retrospective, which may have impacted the identification of some confounding factors. Second, the use of SCr values as the main criterion in the definition of AKI in accordance with the most recent definition of AKI (1) and eliminating urine output from the equation may have caused us to underestimate the incidence of AKI. Finally, patients who were able to be propensity

matched differed in several ways from the patients who were not chosen by the matching process (Table 5). This can affect generalizability in the same way that randomized clinical trials lose some generalizability as a result of inclusion and exclusion criteria.

In conclusion, the incidence of post-transplant AKI in LLDT recipients was 21% and 23.3% with propensity matching testing, which was much lower than the incidence in the corresponding matched-control group of CDLT recipients (44.2%). In the propensity match test, type of graft (LDL graft) was the determining factor in lowering the incidence of post-LT AKI; receiving a graft from live donor had a protective effect against early post-LT AKI.

References

1. Wong F, Nadium MK, Kellum JA, et al. Working Party proposal for a revised classification system of renal dysfunction in patients with cirrhosis. Gut. 2011 May; 60(5):702–709. [PubMed: 21325171]
2. Poge U, Gerhardt T, Palmedo H, Klehr HU, et al. MDRD equations for estimation of GFR in renal transplant recipients. Am J Transplant. 2005; 5:1306–11. [PubMed: 15888034]
3. Hilmi IA, Horton CN, Planinsic RM, et al. The impact of post-reperfusion syndrome on short-term patient and graft outcome. Liver Transpl. 2008 Apr; 14(4):504–508. [PubMed: 18383079]
4. Leuven, E.; Sianesi, B. PSMATCH2. Stata module to perform full Mahalanobis and propensity score matching, common support graphing, and covariate imbalance testing. 2003. http://ideas.repec.org/c/boc/bocode/s432001.html. Version 4.0.10
5. Kiuchi T, Tanaka K, Ito T, et al. Small-for-size graft in living donor liver transplantation: how far should we go? Liver Transpl. 2003; 9:S29. [PubMed: 12942476]
6. Karapanagiotou A, Dimitriadis C, Papadopoulos S, et al. Comparison of RIFLE and AKIN criteria in the evaluation of the frequency of acute kidney injury in post-liver transplantation patients. Transplant Proc. 2014 Nov; 46(9):3222–7. [PubMed: 25420865]
7. Lin CC, Chuang FR, Wang CC, Chen YS, Chen CL, Liu YW, Cheng YF, Lee CH, Jawan B. Early postoperative complications in recipients of living donor liver transplantation. Transpl Proc. 2004; 36:2338–2341.
8. Yamamoto S, Sato Y, Ichida T, Kurosaki I, Nakatsuka H, Hatakeyama K. Acute renal failure during early postoperative period in adult living-related donor liver transplantation. Hepatogastroenterology. 2004 Nov-Dec;51(60):1815–19. [PubMed: 15532833]
9. Lee SK, Park JB, Kim GS, Choi GS, Kim DJ, Kwon CH, Lee SK, Joh JW. Early postoperative renal dysfunction in the adult living donor liver transplantation. Transpl proce. 2007; 39:1517–19.
10. Karapanagiotou A, Kydona C, Dimitriadis C, et al. Acute kidney injury after orthotopic liver transplantation. Transpl Proc. 2012; 44:2727–2729.
11. Selzner M1, Kashfi A, Cattral MS, Selzner N, McGilvray ID, Greig PD, Levy GA, Renner EL, Grant DR. Live donor liver transplantation in high MELD score recipients. Ann Surg. 2010 Jan; 251(1):153–7. [PubMed: 19858705]
12. Boin IF, Leonardi MI, Luzo AC, et al. Intraoperative massive transfusion decreases survival after liver transplantation. Transpl Proc. 2008 Apr; 40(3):789–91.
13. Hod EA, Zhang N, Sokol SA, et al. Transfusion of red blood cell after prolonged storage procedures harmful effects that are mediated by iron and inflammation. Blood. 2010; 115:4284–92. [PubMed: 20299509]
14. Pereboom IT1, Lisman T, Porte RJ. Platelets in liver transplantation: friend or foe? Liver Transpl. 2008 Jul; 14(7):923–31. [PubMed: 18581510]
15. Shaw BW, Martin DJ, Marquez JM, Kang YG, Bugbee AC Jr, Iwatsuki S, et al. The advantages of veno-venous bypass during orthotopic liver transplantation. Semin Liver Dis. 1985; 5:344–8. [PubMed: 3909428]

16. Hilmi IA, Damian D, Al-Khafaji A, et al. Acute kidney injury following liver transplantation: incidence, risk factors, and effects on patient and graft. BJA. 2015 Feb. Page 1 of 8. 10.1093/bja/aeu556

Table 1

Preoperative and intraoperative acute kidney injury (AKI) and non-AKI group data of the live liver donor transplant (LDLT) recipients

LDLT recipient data	AKI group N=21	Non-AKI group N=79	*p≤0.05
Pre-operative Variables			
Patient age (years)	52.4±11.9	52.8±12.1	0.895
Patient sex (male)	13 (61.9%)	40 (50.6%)	0.358
Patient weight (kg)	80.1±17.7	79.5±16.8	0.868
Patient race (Caucasian)	19 (90.5%)	75 (94.9%)	0.273
MELD score	14 [10; 18]	12 [9; 15]	0.086
Child score	8 [8; 11]	8 [7; 10]	0.166
Serum creatinine (mg/dl)	0.9 [0.7; 1.1]	0.9 [0.7; 1.1]	0.905
Modification of Diet in Renal Disease (MDRD) formula	90.7±41.2	85.1±30.2	0.486
eGFR (<60 ml/min/1.73 m2)	4 (19%)	15 (19%)	0.995
Pre-existing diabetes mellitus	7 (33.3%)	21 (26.6%)	0.54
Pre-existing coronary artery disease	1 (4.8%)	5 (6.3%)	0.788
Intra-operative Variables			
Total operative time (h)	7.7 [7.1; 10]	7.9 [6.8; 8.9]	0.939
Cold ischemic time (min)	135 [109; 166]	118 [96; 147]	0.238
Warm ischemic time (min)	31 [26; 42]	32 [30; 38]	0.703
Veno-veno bypass use	18 (90%)	76 (98.7%)	0.045*
Methylene blue use	14 (70%)	49 (63.6%)	0.595
Aprotinin use	1 (5%)	2 (2.6%)	0.58
Post-reperfusion syndrome	6 (28.6%)	6 (7.6%)	0.009*
Crystalloid (liters)	5.8±2.6	5.3±1.9	0.392
Colloid (liters)	2.8±1	2.6±1.2	0.528
Packed red cell (units)	5 [3; 8]	4 [2; 7]	0.114
Fresh frozen plasma (units)	2 [0; 4]	2 [0; 4]	0.321
Platelets (units)	1 [0.5; 2]	0 [0; 1]	0.014*
Cryoprecipitate (units)	0 [0; 0]	0 [0; 0]	0.223
Donor Characteristics			
Donor race (Caucasian)	17 (81%)	73 (92.4%)	0.281
Donor age (years)	39±11.9	36.9±10.7	0.432
Donor height (cm)	1.71±11	1.71±11	0.907
Donor weight (kg)	73.3±16.9	79.5±15.8	0.121

Table 2

Initial comparison of pre-operative and intraoperative variables between live liver donor transplant (LDLT) recipients and cadaveric donor transplant (CDLT) recipients before the propensity match test

Patient Data	LDLT N=100	CDLT N=424	p
Pre-operative variables			
Patient age (years)	52.7±12	56.7±9.5	0.002
Patient gender (male)	53%	32.5%	<0.001
Patient weight (kg)	80 [66; 90]	85 [72; 97]	0.006
Patient race (Caucasian)	94%	94.60%	0.94
(MELD) score	13 [9; 15]	19 [14; 22]	<0.001
Child score	8 [7; 10]	9 [7; 10]	0.106
Pre-transplant serum creatinine (mg/dl)	0.9 [0.8; 1.1]	1 [0.8; 1.3]	<0.001
Pre-existing diabetes mellitus	28%	31.8%	0.456
Pre-existing coronary artery disease	6%	5%	0.67
Intraoperative variables			
Total operative time (h)	7.8 [6.8; 87]	7.2 [6; 8.3]	<0.001
Cold ischemic time (h)	2 [1.6; 2.7]	11 [8.8; 12.]	<0.001
Warm ischemic time (min)	32 [29; 38]	27 [23; 31]	<0.001
Veno-venous bypass use	97%	92%	0.075
Methylene blue use	65%	80%	0.002
Aprotinin use	3.0%	14.5%	0.002
Post-reperfusion syndrome	12%	45.80%	<0.001
Crystalloid use (l)	5 [3.7; 6.5]	4.6 [3.5; 5.7]	0.026
Colloid use (l)	2.5 [2; 3.3]	2.5 [1.5; 3.5]	0.199
Red blood cells (units)	4 [2; 7]	6 [4; 10]	<0.001
Fresh frozen plasma (units)	2 [0; 4]	5 [2; 14]	<0.001
Platelets (units)	1 [0; 1]	1 [0; 2]	<0.001
Cryoprecipitate (units)	0 [0; 0]	0 [0; 1]	<0.001

Table 3

Comparison of mean, bias, and t-test results between the live liver donor transplant (LDLT) recipients and cadaveric donor transplant (CDLT) recipients before and after propensity score matching of the variables of interest

		Mean		% bias	t-test	
Variables	**unmatched matched**	LDLT	CDLT		t	p
Patient age	**unmatched**	52.7	56.7	−37	−3.6	0.002
	matched	55.3	55.4	−1.2	−0.09	0.932
Patient gender	**unmatched**	0.47	0.67	−42.1	−3.87	<0.001
	matched	0.5	0.52	−4.8	−0.3	0.762
MELD score	**unmatched**	13.1	19.3	−105.7	−8.72	<0.001
	matched	13.7	13.4	5.5	0.46	0.646
Pre-transplant serum creatinine	**unmatched**	0.99	1.28	−40.2	−3.08	0.002
	matched	1.001	0.99	1	0.11	0.912
Pre-existing diabetes mellitus	**unmatched**	0.28	0.32	−8.4	−0.75	0.457
	matched	0.29	0.33	−10.1	−0.65	0.514

Table 4

Multivariable mixed effects logistic regression model for outcome of acute kidney injury (AKI) at 72 hours post liver transplantation in a propensity-matched dataset (n=172, 86 pairs)

Variables	Odds Ratio (OR)	Robust Std Err	P-value	95% Confidence interval for OR
Live donor liver graft (vs. cadaveric)	0.31	0.18	0.047	0.096–0.984
Patient age (per year)	0.97	0.26	0.314	0.924–1.026
Male gender (vs. female)	0.58	0.25	0.198	0.255–1.327
Patient weight (per kg)	1.02	0.01	0.205	0.991–1.044
MELD score (per unit)	1.03	0.06	0.542	0.928–1.153
Child score (per unit)	1.22	0.22	0.26	0.863–1.723
Pre-transplant SCr (mg/dl)	1.48	0.88	0.509	0.462–4.743
Pre-existing diabetes mellitus	1.21	0.62	0.709	0.446–3.275
Postreperfusion syndrome	1.78	0.88	0.243	0.676–4.696
Packed red blood cells (per unit)	1.04	0.05	0.477	0.939–1.143
Donor age (per year)	1.00	0.01	0.952	0.975–1.027
(constant)	0.05	0.12	--	--

Model contains a different random effects intercept for each matched pair of subjects

Table 5

Univariate variable comparison between matched and un-matched patients

	Matched (N=172)	Un-matched (N=352)	p
Age (years)	55.5 [49; 62]	56 [50; 64]	0.335
Female gender (%)	51.20%	69.60%	<0.001
Weight (kg)	67.5 [46.4; 79.9]	73.6 [45; 85.1]	0.001
MELD score	13 [10; 16]	22 [15; 24]	<0.001
Child score	8 [7; 9]	9 [7; 10]	0.001
Pre-transplant SCr (mg/dl)	0.9 [0.7; 1.2]	1 [0.8; 1.5]	<0.001
Diabetes mellitus (%)	31.40%	30.97%	0.921
Postreperfusion syndrome (%)	27.30%	45.30%	<0.001
Packed red blood cells (units)	4.5 [3; 8]	7 [4; 10]	<0.001
Donor age (years)	44.5 [33.5; 55]	51 [37; 65]	0.003