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Comparing Ten-Year Renal Outcomes in Deceased Donor and Living Donor Liver Transplants

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Abstract

Few studies have explored whether the type of LT, deceased donor LT (DDLT) or living donor LT (LDLT), impacts long term renal outcomes. We performed a retrospective analysis of 220 LT recipients at our institution to study their renal outcomes at 10 years. Exclusion criteria were, age 18 years, graft survival 6 months and multiorgan transplants; 108 DDLTs and 62 LDLTs were eligible. At baseline, DDLTs had a lower eGFR than LDLTs and 10.2% of DDLTs were on dialysis as compared to 0% of LDLTs. At 10 years, 7 DDLT and 3 LDLT recipients required dialysis or renal transplant ($p=0.75$). In recipients with graft survival >6 months, DDLTs had a slower decline in eGFR as compared to LDLTs ($p<0.01$). Amongst LDLTs the decline in eGFR continued over the entire 10-year period, whereas amongst DDLTs, the decline in eGFR slowed significantly after 6-months ($p=0.01$). This difference between the two groups was not seen amongst patients in the highest quartile of baseline eGFR. Patient and graft survival was similar. In conclusion, the incidence of end stage renal disease was similar in both DDLT and LDLT patients but LDLT recipients seem to have a more sustained decline in eGFR when compared to DDLT recipients.

Keywords

Renal outcomes; liver transplant; deceased donor; living donor

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Introduction

Chronic kidney disease (CKD) is the most frequent chronic complication after liver transplant (LT)(1). It is associated with decreased survival, especially if recipients develop end stage renal disease (ESRD) (1–5). Risk factors for the development of CKD after LT include cyclosporine use, dose and levels (6–8), recipient age (1, 2, 9), number of years elapsed after LT(1), acute kidney injury (AKI) post-LT (2), hepatitis C infection (2, 10, 11), pre- or post- transplant diabetes mellitus (2, 7, 9, 10), pre- or post- transplant hypertension (2, 8, 9, 11) pre-transplant eGFR (2, 3, 6), and decline in eGFR within the first year post-LT(1, 3, 6–8, 10). Long term outcomes of living donor LT (LDLT) as compared to deceased donor LT (DDLT) have yielded mixed results, often favoring the latter (12–17). However, increased center experience and improved surgical techniques are associated with improved LDLT outcomes (12, 14, 18).

Evidence exploring the impact of the type of graft on renal outcomes is scarce. To the best of our knowledge, only one study has reported similar incidence of CKD at 5 years, between both DDLT and LDLT recipients who had hepatorenal syndrome (19). Since CKD is the commonest complication in patients undergoing LT (1), our aim was to evaluate if the type of LT influences renal outcomes (development of ESRD and eGFR decline) in LT recipients over 10 years.

Methods

We performed a retrospective analysis of 220 patients who received a LT at the University of Rochester between January 2000 and December 2001. Exclusion criteria were age < 18 years, graft survival < 6 months and multi-organ transplants. Patients with graft survival less than 6 months were excluded from the analysis since the objective of our study was to explore long term renal outcomes in patients who had successful liver transplantation. Amongst eligible subjects, patient information was obtained from a prospectively maintained electronic database, medical records software (EPIC, ALLSCRIPTS and OTTR) and, when necessary, data was requested from the United Network for Organ Sharing. Institutional review board approval was obtained prior to study initiation (RSRB00046155). Statistical analysis was supported by the University of Rochester clinical and translational science award number UL1 TR000042 from the National Center for Advancing Translational Sciences of the National Institutes of Health.

Data

The following preoperative factors were analyzed: age, sex, race/ethnicity, body mass index (BMI), crossmatch status, wait days, baseline co-morbid conditions (diabetes, hypertension, coronary artery disease, cancer), social history (smoking, alcohol), viral serologies (cytomegalovirus [CMV], Epstein–Barr virus [EBV], varicella zoster virus [VZV], herpes simplex virus [HSV]), model for end-stage liver disease (MELD) score, total bilirubin, international normalized ratio (INR), albumin, presence of ascites, cause of liver failure, baseline eGFR and renal replacement therapy (RRT) needs. Post-operative factors analyzed included: biliary complications, graft survival, patient survival, cause of death and, renal parameters such as eGFR and RRT needs or kidney transplant (KT) at 3, 6, 12, 60 and 120

months. For some patients 3-month serum creatinine values were not available and thus values between 1–3 months were used in the analysis. MELD scores for those transplanted before February 2002 were calculated using laboratory values available within 48 hours of the transplant. The eGFR was calculated using the MDRD equation. Patients who received a KT or were receiving RRT were assigned an eGFR of 10 ml/min/1.73 m² BSA for statistical evaluation.

Immunosuppression

Per protocol, both DDLT and LDLT recipients received the standard immunosuppressive regimen of tacrolimus, mycophenolate mofetil (MMF), and steroids. Tacrolimus was given orally every 12 hours at a dosage of 0.1 mg/kg/d. Target 12-hour trough whole blood tacrolimus concentrations were 12 ng/mL in the first month, 10 ng/mL in the second month, 8 ng/mL in the third month, and 6 ng/mL beyond 3 months. MMF (1 g) was administered orally twice a day. One gram of methylprednisolone was given intra-operatively prior to reperfusion of the liver allograft, followed postoperatively by a steroid taper totaling 600 mg over the next 5 days. After 5 days, prednisone, 20 mg/day was given and gradually reduced to 5 mg/day by 3 to 6 months. Subsequent immunosuppressive adjustments were made based on the individual's clinical course considering the presence of signs of rejection, drug toxicity, or infection.

Statistical analysis

Two sample comparisons were performed using the Wilcoxon rank-sum test (quantitative data) or Fisher's exact test (categorical data). Survival times were estimated using Kaplan-Meier curves and compared using log-rank tests. A linear mixed effects model with subject level random effects was used to model log eGFR as a function of time (including a quadratic term to model nonlinearity), with transplant type main effect, age at transplant main effect, and transplant type/time interaction included as independent predictors. Statistical analyses were performed using R statistical computing environment (cran.r-project.org). Estimates of nonlinear functional relationships were further analyzed using a generalized additive model (20). Models were fit using the generalized additive mixed model function (gamm4), part of the R statistical computing environment (21). Age was included as an independent variable. This function allows inclusion of subject level random effects in the model. A p-value of <0.05 was considered statistically significant.

Results

Patient characteristics

Of the 220 LTRs, 170 were eligible for analysis (108 DDLTs and 62 LDLTs). Nine patients were excluded since they were <18 years of age and one patient had a combined liver and kidney transplant. In the DDLT group, 26 patients (19.4%) had a graft survival of less than 6 months and were excluded from the study. Of these, 8 died with a functioning graft, 7 died after graft failure, 6 had a re-transplant and re-transplant status of the remaining 5 was unknown. In the LDLT group, 14 patients (18.4%) were excluded from the study since their grafts survived less than 6 months. Of these, 7 died with a functioning graft, 3 died after graft failure and 4 had re-transplants.

Baseline characteristics of the recipients are summarized in Table 1. There were more LDLT recipients with a history of smoking (66.13% vs. 44.44% $p = 0.02$). When compared with DDLT recipients, LDLT recipients had lower mean MELD scores (20.11 ± 8.84 vs. 13.94 ± 4.16 $p < 0.01$), lower total bilirubin levels (5.56 ± 7.83 vs. 2.43 ± 1.66 $p = 0.02$), lower INR (1.73 ± 0.59 vs. 1.45 ± 0.31 $p < 0.01$), higher albumin levels (2.79 ± 0.63 vs. 3.21 ± 0.65 $p < 0.01$), and a lesser percentage of patients with ascites (63.9% vs. 46.8% $p = 0.04$). All patients with acute hepatic necrosis received a DDLT ($p < 0.01$). There was no difference in the total number of biliary complications between DDLTs and LDLTs (38.9% vs. 32.3% $p = 0.41$). Maintenance immunosuppression regimens were comparable [Table 2]. Overall, tacrolimus trough levels were higher in the DDLT recipients but were within the target range of the protocol.

Comparison of patient and graft survival

Patient and graft survival was similar in the two groups [Figure 1]. 10-year patient survival was 58.3% in the DDLT and 61.3% in LDLT recipients respectively. 10-year graft survival was 56.4% in the DDLTs and 54.8% in LDLTs respectively. Cause of death is summarized in Table 3. The most common cause of death in the DDLT recipients was sepsis (37.8%) and in the LDLT recipients was cancer (25%). Three DDLT deaths were attributed to renal failure.

Comparison of renal outcomes

Renal characteristics are summarized in table 4 and represent the median eGFRs. At baseline, DDLTs had a lower median transplant eGFR when compared with LDLTs (81.75 vs. 101.72 , $p = 0.01$). Also, 10.2% of the patients with the DDLT and none of the patients with LDLTs were on RRT prior to transplant ($p < 0.01$). Seven patients with DDLTs and three with LDLTs were on dialysis or had a KT by 10 years but this difference did not reach statistical significance ($p = 0.75$). Overall, 6% of the patients in our cohort had ESRD requiring RRT or KT.

As shown in figures 2a, for those patients that had graft survival > 6 months, there was a steeper decline in the eGFR in the LDLTs when compared with the DDLTs ($p < 0.01$). Figure 2b and c represent the eGFR decline over time in the form of boxplots. In the DDLT recipients, eGFR decline appears to slow down after 6 months, whereas there appears to be a sustained eGFR decline in the LDLT recipients over the entire 10 years [figure 3 b, c]. The drop in eGFR between 6 months and 10 years was significantly higher for the LDLT recipients when compared with the DDLT recipients ($p = 0.01$).

Impact of pre-transplant eGFR

Lastly, we compared the effect of pre-transplant eGFR on eGFR decline over 10 years between DDLTs and LDLTs. To do this we grouped recipients into 4 quartiles each: 1st quartile represents recipients with the highest baseline eGFR and 4th represents those with the lowest eGFR [Supplementary figure 1a–d]. For the quartile of patients with the highest baseline eGFR in each group, the overall decline in eGFR appears to be similar. For recipients in the 2nd and 3rd eGFR quartiles, within the DDLT group the decline in eGFR begins to stop by 6 months, whereas the decline for LDLTs continues over the entire 10

years. In the 4th quartile which has the patients with the lowest eGFR at baseline, in the DDLT recipients, eGFR increases in the first few months and then stabilizes. By contrast, for the same quartile for the LDLT recipients, eGFR shows a steady decline beyond 12 months.

Figure 3a and b analyzes the change in eGFR between each consecutive time period using boxplots. For the first quartile, overall there is no statistically significant decline in eGFR after 6 months in both LDLT and DDLT recipients. LDLT recipients in quartiles 2–4, overall demonstrate a statistically significant drop in eGFR in most time intervals [Figure 3c]. DDLT recipients, on the other hand, demonstrate no statistically significant drop in eGFR at each consecutive time interval after 6 months [Figure 3d].

Discussion

Our study demonstrates that amongst long term survivors of LT, incidence of ESRD is similar in both groups. However, LDLT recipients appear to have a more rapid and sustained decline in eGFR as compared with DDLT recipients. Patients with a higher pre-transplant eGFR appear to have a similar decline in both DDLT and LDLT groups. Similar findings were noted by Nishi and colleagues who demonstrated that LDLTs have a more rapid deterioration of renal function, especially in the first year (22). A study from Toronto, specifically looking at patients who had undergone LT for hepato-renal syndrome, showed similar creatinine levels in between the two groups at 5 years but the rate of eGFR decline was not reported (19).

The reasons for our findings could be several. First, liver complications occur at a higher rate in LDLTs. A meta-analysis of perioperative outcomes after LT showed that in comparison to DDLTs, LDLTs were associated with a significantly longer duration of recipient operation time, biliary complications, vascular complications and re-transplantation (18). It is possible that these peri- and post-operative complications lead to hemodynamic changes, ischemic and toxic injury to the kidneys and cause AKI at a higher rate in the LDLTs. AKI after LT has a reported incidence of 12% to 95% and the predominant etiology is acute tubular necrosis (4, 23, 24). Since AKI is a well-known risk factor for CKD we speculate this might lead to a higher eGFR decline in the LDLTs.

Second, a study by Fujinaga and colleagues of 98 LDLTs demonstrated that small graft-to-recipient weight ratio (GRWR) was predictive of late renal dysfunction (11). A significant relationship between low GRWR and early postoperative renal dysfunction is also described (25). Also, male recipients with female donors have a higher likelihood of graft failure (26). Smaller graft size is thought to affect the balance between vasoconstriction and vasodilatory factors and lead to AKI (11). Also, AKI after LDLT is speculated to occur due to persistent portal hypertension and a hyperdynamic state in patients with a smaller graft (25).

Lastly, an interesting study by Iglesias and colleagues revealed that worsening renal function while awaiting LT was protective against the development of AKI post-LT (24). One of the many possible reasons proposed was ischemic preconditioning, which is a well-known phenomenon in the contrast nephropathy literature. The DDLTs in our group did have lower

eGFR when compared with LDLTs and 10.2% required dialysis. Additionally, for DDLT recipients in the lowest eGFR quartile, we did note an increase in eGFR in the first few months post-LT. Thus, it is possible that ischemic preconditioning pre-transplant might have led to better renal outcomes post-transplant in the DDLT recipients. We did not have peri-operative data to study the impact of AKI and GRWR on 10-year renal outcomes.

The etiology of CKD after LT is varied and is often assumed to be due to calcineurin inhibitor (CNI) toxicity (6). However, in a study of 81 LTs with creatinine >1.5mg/dl, all biopsies demonstrated glomerular abnormalities, 42% showed primary glomerular diseases, and only 16% had evidence of CNI toxicity (27). Hence, CKD in patients with LT is attributable to different pathologies. We did note that LDLTs had lower mean tacrolimus trough levels when compared to DDLTs at days 3, 7 and 21. Hence, at least in the immediate post-operative period, CNI toxicity cannot explain our findings.

Our findings may be important when evaluating and counseling patients for a LT, particularly those with a compromised renal function. Nevertheless, LDLT is still an excellent alternative to DDLT because it facilitates access to LT (18). LDLTs at one center had a 10% 1-year survival advantage compared with DDLTs or patients on a waiting list (16). Also, LDLT is advantageous to those with lower MELD scores who are not eligible for a DDLT (28).

Our study has a number of limitations. Being retrospective in nature, this study cannot account for a number of confounding factors that may have influenced the outcomes reported such as cumulative CNI dose, impact of donor variables, surgical technique, rejection episodes, graft size and use of other potential nephrotoxic medications. This is a single-center study and our results may not be generalizable to other centers. Also, for the reasons stated earlier, we excluded patients with early graft failure (less than 6 month graft survival) from our study. Our analysis does not have the same power as a prospective, intention-to-treat, randomized and controlled clinical trial. We used the MDRD equation for eGFR calculation that is less accurate in cirrhotic patients even though, our analysis entailed trending correlation between changes in eGFR over time rather than absolute values. We did not examine other parameters of renal dysfunction such as albuminuria or proteinuria, and neither did we have data on histologic analyses of the kidneys. Lastly, this study included LTs in 2000 and 2001 prior to using MELD for organ allocation. A prospective study might be able to address all these limitations. Nevertheless, since renal dysfunction is the most common complication after LT (1) and is associated with lower survival (2–5, 8), our findings may be relevant to counsel patients and prospective donors.

In conclusion, amongst liver transplant recipients with allograft survival of >6 months, our study showed that LDLT recipients have a similar incidence of ESRD but a more rapid and sustained decline in eGFR as compared with DDLT recipients over a period of 10 years. Patients with a higher baseline eGFR appear to have similar long term renal outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Figure 1. Kaplan-Meier curves of 10-year graft and patient survival of deceased donor and living donor liver transplant recipients. Number at risk for each treatment group is given at the times indicated on the plot.

Figure 2.

a) Comparison of the decline in eGFR over time between deceased donor and living donor liver transplant recipients (DDLTL and LDLTL respectively). A linear mixed effects model with subject level random effects was used to model log eGFR as a function of time (including a quadratic term), with transplant type main effect, age at transplant main effect, and transplant type/time interaction included as independent predictors. Log eGFR was then expressed as its numerical equivalent for ease of interpretation. Age is controlled at median = 52. Also, boxplot representation of eGFR at study time-points for DDLTL (b) and LDLTL (c) recipients are presented.

Figure 3.

Boxplot representation of Δ eGFR (delta eGFR) calculated between consecutive study time-points, by treatment group and transplant eGFR quartile. Using a generalized additive model, decline in eGFR was plotted over time for deceased donor and living donor liver transplant recipients (DDLT and LDLT respectively). Recipients were grouped into 4 quartiles based on their transplant eGFR, 1st quartile represents recipients with the highest baseline eGFR and 4th represents those with the lowest eGFR. Age is controlled at median = 52. The median eGFR in the DDLT and LDLT for 1st, 2nd, 3rd, and 4th quartiles were 170.04, 106.23, 74.67, 36.89 and 144.39, 103.24, 78.84, 50.08, respectively. An asterisk * indicates Δ eGFR < 0 with $p < 0.05$.

Table 1

Characteristics of the study population

Characteristics	DDLT (108)	LDLT (62)	P value
Age (mean in years)	52.03 $\pm$ 10.58	52.93 $\pm$ 9.39	0.83
Race/Ethnicity n (%)			0.76
African American	6 (5.5)	1 (1.6)	
Asian	2 (1.9)	1 (1.6)	
Caucasian	92 (85.2)	55 (88.7)	
Hispanic	7 (6.5)	5 (8.1)	
Native American	1(0.9)	0	
Sex n (%)			0.13
Female	32 (29.6)	26 (41.9)	
Male	76 (70.4)	36 (58.1)	
BMI (mean in kg/m ²)	29.40 $\pm$ 6.52	29.23 $\pm$ 5.78	0.93
Positive Crossmatch	5	4	0.74
Wait days (mean)	314.85 $\pm$ 354.86	295.58 $\pm$ 391.72	0.90
Medical History n (%)			
Diabetes	45 (41.7)	18 (29)	0.10
Hypertension	42 (38.9)	27 (43.5)	0.63
Coronary artery disease	3 (2.8)	7 (11.3)	0.11
Cancer (prior)	14 (13)	8 (12.9)	0.87
Social History n (%)			
Smoking	48 (44.4)	41 (66.1)	0.02
Alcohol	53 (49.1)	27 (43.5)	0.53
Positive viral serologies n (%)			
Cytomegalovirus	67 (62)	30 (48.4)	0.11
Epstein Barr virus	100 (92.6)	59 (95.2)	0.75
Varicella Zoster virus	98 (90.7)	58 (93.5)	0.58
Herpes Simplex virus	76 (70.4)	42 (67.7)	0.32
Liver characteristics			
MELD (mean)	20.11 $\pm$ 8.84	13.94 $\pm$ 4.16	<0.01
Total bilirubin (mean in mg/dl)	5.56 $\pm$ 7.83	2.43 $\pm$ 1.66	0.02
INR (mean)	1.73 $\pm$ 0.59	1.45 $\pm$ 0.31	<0.01
Albumin (mean in mg/dl)	2.79 $\pm$ 0.63	3.21 $\pm$ 0.65	<0.01
Ascites n (%)	69 (63.9)	29 (46.8)	0.04
Cause of liver failure n (%)			
Acute hepatic necrosis	11 (10.2)	0	<0.01
Cholestatic liver disease	8 (7.4)	8 (12.9)	0.28
Fulminant	2 (1.9)	0	0.53
Hepatitis B	15 (13.9)	10 (16.1)	0.82
Hepatitis C	12 (11.1)	3 (4.8)	0.26
Malignant Neoplasm	0	2 (3.2)	0.13

Characteristics	DDLT (108)	LDLT (62)	P value
Metabolic/toxic	5 (4.6)	0	0.16
Non-cholestatic	82 (75.9)	51 (82.3)	0.44
Biliary Complications n (%)	42 (38.9)	20 (32.3)	0.41

Table 2

Immunosuppression regimen

Characteristics	DDLT (108)	LDLT (62)	P value
Maintenance Immunosuppression			
Steroids	108	61	0.36
Tacrolimus	100	57	>0.99
Azathioprine	1	0	>0.99
Sirolimus	4	0	>0.99
Generic cyclosporine	1	0	>0.99
Cyclosporine (Neoral)	3	3	>0.99
Cyclosporine (Sandimmune A)	2	2	>0.99
Mycophenolate Mofetil	91	59	>0.99
Tacrolimus trough levels in ng/ml			
Day 3 Mean	12.57±7.57	6.04±3.79	<0.01
Day 7 Mean	11.17±3.89	9.19±3.98	<0.01
Day 14 Mean	10.55±3.60	8.83±3.83	<0.01

Table 3

10-year patient survival and cause of death

	DDLT	LDLT
Alive % (n)	58.3 (63)	61.3 (38)
Deceased % (n)	41.7 (45)	38.7 (24)
Cause of death % (n)		
Cancer	8.9 (4)	25 (6)
Heart	11.1 (5)	12.5 (3)
Liver	4.4 (2)	8.3 (2)
Renal failure	6.7 (3)	0
Sepsis	37.8 (17)	20.8 (5)
Other	20 (9)	8.3 (2)
Unknown	11.1 (5)	25 (6)

Table 4

Renal characteristics of the study population

Characteristics	DDLT	LDLT	P value
eGFR* at transplant, median	81.75+/-11.34	101.72+/-11.39	0.01
RRT prior to or at transplant, % (n)	10.2 (11)	0	<0.01
Hemodialysis or renal transplant, % (n)			
1-year	4.6 (5)	1.6 (1)	0.42
5-year	4.6 (5)	1.6 (1)	0.42
10-year	6.5 (7)	4.8 (3)	0.75
eGFR*, median (n)			
3-month	70.91+/-10.56 (101)	93.28+/-8.41 (61)	<0.01
6-month	56.99+/-3.65 (98)	74.48+/-5.68 (57)	<0.01
1-year	55.14+/-3.84 (95)	71.37+/-9.61 (57)	<0.01
5-year	51.29+/-5.96 (71)	55.69+/-11.53 (46)	0.55
10-year	52.20+/-7.71 (50)	49.78+/-17.16 (30)	0.89

* eGFR is in ml/min/1.73 m²