ORIGINAL ARTICLE

Risk factors for and outcomes of delayed graft function in live donor kidney transplantation – a retrospective study

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SUMMARY

Delayed graft function (DGF) in deceased donor kidney transplantation is associated with worse outcomes. DGF has been less well studied in live donor transplantation. We aimed to examine the risk factors for DGF, and associations between DGF and short- and long-term outcomes in live donor kidney transplant recipients. Using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, we included live donor kidney transplants performed in Australia and New Zealand over 2004–2015 and excluded pediatric recipients (n = 440), pathological donors (n = 97), grafts that failed in the first week (as a proxy for primary non function; n = 38), and grafts with missing DGF data (n = 46). We used multivariable logistic regression to identify the risk factors for DGF and the association between DGF and rejection at 6 months; Cox proportional hazards models to examine the relationship between DGF and patient and graft survival; and linear regression to examine the association between DGF and eGFR at 1 year. DGF occurred in 77 (2.3%) of 3358 transplants. Risk factors for DGF included right-sided kidney [odds ratio (OR) 2.00 (95% CI 1.18, 3.40)], donor BMI [OR 1.06 per kg/m^2 (95% CI 1.01, 1.12)]; increasing time on dialysis and total ischemic time [OR 1.09 per hour (1.00, 1.17)]. DGF was associated with increased risk of rejection at 6 months [OR 2.37 (95% CI 1.41, 3.97)], worse patient survival [HR 2.14 (95% CI 1.21, 3.80)] and graft survival [HR 1.98 (95% CI 1.27, 3.10)], and worse renal function at 1 year [Coefficient -9.57 (95% CI −13.5, −5.64)]. DGF is uncommon after live donor kidney transplantation, but associated with significantly worse outcomes. The only modifiable risk factors identified were kidney side and total ischemic time.

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Key words
immunosuppression, ischaemia, kidney, live donors, outcome, rejection, surgery

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Introduction

Kidney transplantation is the preferred modality of treatment for the majority of end-stage kidney disease patients. In general, live donor kidney transplantation (LDKT) has superior outcomes compared to deceased donor kidney transplantation (DDKT) [1]. Factors that contribute to the better function include superior organ quality and well organized surgical conditions to reduce ischemic time [1].

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Various studies used different definitions for delayed graft function (DGF), such as the requirement for dialysis within 1 week of transplantation, increase in serum creatinine by 43 μmol/l in the first 24 h or decline in urine output by 30 ml/h in first 24 h [2]. The most widely accepted definition for DGF is requiring dialysis in the first week post-transplantation.

The incidence of DGF in most studies ranges from 20% to 50% in deceased donor and 4–10% in LDKT recipients [1]. DGF in deceased donor transplants is associated with increased length of hospital stay and risk of premature graft dysfunction and rejection [3].

Various risk factors have been attributed to DGF in DDKT, and the impact of DGF on graft survival and rejections has been well documented [4,5]. Conversely, risk factors for DGF and the associations between DGF and rejection and graft survival in LDKT are less well established. It is not known if established risk factors for DGF in DDKT also apply to LDKT.

The aim of this study was to examine the risk factors for DGF, and associations between DGF and short- and long-term graft and patient outcomes in LDKT.

Materials and methods

Study population

All adult primary LDKT recipients in Australia and New Zealand between 2004 and 2015 were included. We excluded pathological donors, defined as kidneys transplanted after nephrectomy for a tumor ($n = 97$), grafts that failed in the first week (as a proxy for primary nonfunction; $n = 38$), and grafts with missing DGF data ($n = 46$). There were 3358 LDKT recipients after these exclusions (Fig. 1).

Data collection

Baseline donor characteristics in the ANZDATA registry included age; relationship to recipient (related versus unrelated); gender; ethnicity; country of birth; comorbid conditions (smoking, diabetes, hypertension, baseline GFR estimated with the use of Chronic Kidney Disease Epidemiology Collaboration Creatinine Equation [6]; kidney side; type of surgery; number of renal arteries; renal veins and ureters, HLA match, ABO match).

Recipient characteristics in ANZDATA included age; gender; ethnicity; cause of end-stage kidney disease; preemptive transplants; percentage peak panel reactive antibody (PRA); waiting time before live donor transplant; comorbidities (diabetes, coronary artery disease, peripheral vascular disease, cerebrovascular disease and smoking history).

Transplant-related characteristics in ANZDATA included HLA mismatches; ABO incompatibility; total ischemic time (hours); and use of antibody induction immunosuppression therapy.

In this study, DGF was deemed to be present if the registry data demonstrated that there was no spontaneous fall in serum creatinine by 10% in 72 h and dialysis was required during the first 72 h after transplantation.

Clinical outcomes

The outcomes we studied were risk factors for DGF; rejection at 6 months; graft function (CKD-EPI eGFR) at 1 year after transplantation; graft survival (defined as survival without return to dialysis or re-transplantation) and patient survival.

We assessed the following factors for confounding: primary disease; predictors of rejection – HLA mismatch, Peak PRA, donor and recipient's age; re-transplantation; total ischemic time; duration and modality of renal replacement therapy (RRT); comorbidities of recipient such as diabetes, hypertension, coronary artery disease, peripheral vascular disease, cerebrovascular disease and chronic lung disease; body mass index; antibody induction immunosuppression and era of transplantation (2004–2009, 2010–2015).

Statistical analyses

The baseline characteristics of the study cohort were expressed as the number and proportion, mean plus or minus standard deviation (SD), or median and interquartile range (IQR).

Associations among covariates and DGF were analyzed by multivariable logistic regression analysis, and results were expressed as odds ratios (ORs) with 95% confidence intervals (CI). The association of DGF with eGFR at 1 year was modeled by linear regression models and results were expressed as the mean difference with 95% CIs. Association between DGF and rejection at 6 months was analyzed with multivariable logistic regression analysis. We used Cox proportional hazards models to examine the relationship between DGF and patient and graft survival. Follow-up was until an outcome event occurred, and was censored at 31 December 2015, loss to follow-up or re-transplantation from a live donor.

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For each outcome, variables significant at the 0.05 level on univariable analysis were included in a base multivariable model. We sequentially removed the least significant variables until all remaining variables were significant at the 0.05 level. DGF was forced into every model since it was the primary exposure. We performed standard model diagnostics to ensure models met assumptions.

Statistical analyses were performed using STATA version 14 (Stata Corp LLC, College Station, TX, USA). *P*-values <0.05 were considered statistically significant.

Results

Live donor kidney transplant donor, recipient, and transplant characteristics

Baseline characteristics of our study population are described in Table 1. There were 3358 live donor transplant recipients between 2004 and 2015 who were followed for a median of 5.8 years resulting in 19 126 person–years of follow-up. Sixty-three (63) recipients lost to follow-up after a median of 2.1 years and only two of them had DGF.

A total of 77 of 3358 recipients (2.3%) experienced DGF. Distribution of donor characteristics such as age; gender; BMI; eGFR (CKD-EPI); and donor diabetes was similar in each group whereas donor hypertension was more common in the DGF group. A right-sided donor kidney was observed more in the DGF group whereas other graft characteristics such as number of arteries; veins or ureters; or surgical procedure technique (laparoscopic surgery) were similar across both groups.

There were 940 pre-emptive kidney transplant recipients in our cohort and only six experienced DGF. DGF was observed in only two out of 110 kidney exchange recipients included in this study.

Risk factors for DGF

Risk factors for DGF are shown in Table 2, and included donor BMI [OR 1.06 per kg/m^2 (95% CI 1.01, 1.12)]; right kidney [OR 2.00 (95% CI 1.18, 3.40)], increasing time on dialysis (mainly 6 months or more) and total ischemic time (OR 1.09 (95% CI, 1.00, 1.17) per hour.

DGF and rejection

The rejection rate at 6 months was 20% in those without DGF and 40% in those with DGF. On multivariable analysis, DGF was associated with an increased risk of rejection at 6 months [OR, 2.37 (95% CI 1.41, 3.97)] (Table 3). Other risk factors included HLA mismatch; induction treatment; and peak PRA (Fig. 1).

DGF and eGFR at 1 year

At 1 year the mean (SD) eGFR was 57.4 (16.9) ml/min/1.73 m^2 in those without DGF and 47.4 (18.4) ml/min/1.73 m^2 in those with DGF. After adjusting for other variables, DGF was associated with a lower eGFR at 1 year [Coefficient −9.57 (95% CI −13.5, −5.64)] (Table 4), whereas donor characteristics such as age; sex; eGFR or recipient characters age; sex; race; BMI at transplantation; and induction treatment were not statistically associated with eGFR at 1 year and were adjusted for the calculation in the model (Table 4).

DGF and graft survival

Delayed graft function was associated with poor graft survival [HR 1.98 (95% CI 1.27, 3.10)] (Fig. 2 and Table 5). Diabetic nephropathy as a primary cause of renal disease [HR 1.84 (95% CI 1.34, 2.52)] and renal replacement therapy duration (1 to <5 years) [HR 1.40 (95% CI 1.09, 1.80)] were also significantly associated with graft survival (Table 5). Recipient comorbidities, mainly coronary artery disease (HR 1.35 [95% CI 1.04, 1.74]) and cerebrovascular events (HR 1.44 [95% CI 1.02, 2.05]) were associated with poorer graft survival. Peak PRA and duration of renal replacement therapy were not significantly associated with poor graft survival in this cohort.

DGF and patient survival

Delayed graft function was associated with decreased patient survival [HR 2.14 (95% CI 1.21, 3.80)] (Fig. 3 and Table 6). Longer renal replacement therapy duration (mainly more than 1 year) [HR 1.84 (95% CI 1.27, 3.67)]; diabetic nephropathy as a primary renal disease [HR 2.86 (95% CI 0.97, 4.15)]; and cerebrovascular events [HR 1.61 (95% 1.07, 2.40)] were also significantly associated with poor patient survival among the LDKT recipients. Recipient age, peak PRA had no association with poor patient survival.

Discussion

In our study involving 3358 LDKT recipients with a median follow-up of 5.8 years, we demonstrated that

Table 1. Baseline characteristics of live donor kidney transplant recipients stratified by categories of delayed graft function (n = 3358)

Factor	No DGF	DGF	*P*-value
Number	3281	77	
Donor characteristics			
Donor age (years), median (IQR)	50.0 (42.0, 58.0)	51.0 (43.0, 59.0)	0.90
Donor male sex	1341 (40.9%)	29 (37.7%)	0.57
Donor diabetes	9 (0.3%)	1 (1.3%)	0.10
Donor hypertension	663 (20.4%)	23 (30.7%)	0.030
Donor body mass index, median (IQR)	26.3 (23.7, 29.0)	27.7 (24.5, 29.8)	0.018
Donor eGFR (CKD-EPI), median (IQR)	92.5 (81.5, 102.4)	90.0 (84.1, 101.6)	0.89
Right-sided kidney	560 (17.2%)	20 (26.3%)	0.038
Laparoscopic surgery	2891 (88.7%)	64 (84.2%)	0.23
Number of arteries			
1	2732 (84.0%)	64 (85.3%)	0.96
2	484 (14.9%)	10 (13.3%)	
3	33 (1.0%)	1 (1.3%)	
4	4 (0.1%)	0 (0.0%)	
Number of veins			
1	3125 (96.2%)	72 (96.0%)	0.98
2	116 (3.6%)	3 (4.0%)	
3	6 (0.2%)	0 (0.0%)	
4	1 (<1%)	0 (0.0%)	
Number of ureters			
1	3209 (98.9%)	75 (100.0%)	0.36
2	36 (1.1%)	0 (0.0%)	
Recipient characteristics			
Age at transplant, median (IQR)	47.0 (36.0, 56.0)	50.0 (38.0, 58.0)	0.28
Recipient male	2083 (63.5%)	56 (72.7%)	0.096
Ethnicity			
Caucasian	2768 (84.4%)	67 (87.0%)	0.75
Aboriginal/Torres Strait Islander	20 (0.6%)	0 (0.0%)	
Asian	278 (8.5%)	5 (6.5%)	
Māori	79 (2.4%)	1 (1.3%)	
Pacific	72 (2.2%)	2 (2.6%)	
Other	36 (1.1%)	2 (2.6%)	
Not reported	28 (0.9%)	0 (0.0%)	
Primary renal disease			
GN	1551 (47.3%)	29 (37.7%)	0.012
Polycystic	546 (16.7%)	9 (11.7%)	
Reflux	336 (10.3%)	8 (10.4%)	
Hypertension	144 (4.4%)	5 (6.5%)	
Diabetes	245 (7.5%)	14 (18.2%)	
Other	454 (13.9%)	12 (15.6%)	
Recipient BMI at transplant, median (IQR)	25.6 (22.7, 29.1)	26.5 (24.1, 29.7)	0.054
Diabetes	508 (15.5%)	17 (22.1%)	0.12
Coronary disease	400 (12.2%)	18 (23.4%)	0.003
Peripheral vascular disease	180 (5.5%)	8 (10.4%)	0.065
Cerebrovascular disease	149 (4.6%)	5 (6.5%)	0.42
Chronic lung disease	191 (5.8%)	5 (6.5%)	0.81
RRT duration			
Pre-emptive	1012 (30.8%)	9 (11.7%)	0.002
<6 months	437 (13.3%)	8 (10.4%)	
6 months to <1 year	388 (11.8%)	13 (16.9%)	
1 to <5 years	1065 (32.5%)	32 (41.6%)	
5+ years	379 (11.6%)	15 (19.5%)	
Re-transplantation	258 (7.9%)	9 (11.7%)	0.22

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Table 1. Continued.

Factor	No DGF	DGF	*P*-value
Transplant characteristics			
Year of transplant			
2004–2009	1724 (52.5%)	36 (46.8%)	0.31
2010–2015	1557 (47.5%)	41 (53.2%)	
HLA mismatch, median (IQR)	3.0 (2.0, 5.0)	4.0 (2.0, 5.0)	0.069
Peak PRA (%), median (IQR)	0.0 (0.0, 7.0)	2.0 (0.0, 6.0)	0.59
ABO incompatibility	289 (9.1%)	11 (14.3%)	0.12
Total ischemia (to nearest hour), mean (SD)	2.9 (1.8)	3.5 (1.9)	0.003
Any induction therapy	2724 (83.0%)	64 (83.1%)	0.98

BMI, body mass index; CKD-EPI, chronic kidney disease epidemiology collaboration; GN, glomerulonephritis; HLA, human leukocyte antigen; IQR, interquartile range; PRA, panel reactive antibody; RRT, renal replacement therapy; SD, standard deviation.

Table 2. Risk factors associated with delayed graft function

	Odds ratio	95% CI	*P*-value
Donor body mass index	1.06	1.01, 1.12	0.03
Right kidney	2.00	1.18, 3.4	0.01
Renal replacement therapy duration			
Pre-emptive	1 (referent)		0.01
<6 months	1.76	0.65, 4.77	
6 months to <1 year	3.89	1.64, 9.18	
1 to <5 years	3.26	1.54, 6.91	
5+ years	3.21	1.31, 7.84	
Total ischemia time (per hour)	1.09	1.00, 1.17	0.034

Results of final multivariable model.

Table 3. : Risk factors for rejection at 6 months

	Odds ratio	95% CI	*P*-value
Delayed graft function	2.37	1.41, 3.97	0.001
Donor age (per year)	1.02	1.01, 1.02	0.00
Donor male	0.80	0.66, 0.97	0.02
Primary renal disease			
Glomerulonephritis	1 (referent)		0.003
Polycystic	0.63	0.48, 0.83	
Reflux nephropathy	0.90	0.66, 1.25	
Hypertension	1.28	0.85, 1.91	
Diabetes	1.17	0.84, 1.63	
Other	0.78	0.58, 1.05	
Recipient body mass index at transplant (per kg/m^2)	1.04	1.02, 1.06	<0.001
Human leukocyte antigen mismatch	1.23	1.16, 1.30	<0.001
Peak panel reactive antigen assay	1.01	1.00, 1.01	0.005
Induction treatment	0.52	0.42, 0.66	<0.001

Results of final multivariable model.

DGF was independently associated with poor graft function, higher risk of rejection, and poorer graft and patient survival. We identified longer total ischemia time, right-sided kidney, and increasing time on dialysis as risk factors for DGF, which are not different from those described for DDKT.

In a United network for organ sharing (UNOS) study, Redfield *et al.* [3] demonstrated cold ischemia time (CIT) and African American donor race were significant risk factors for DGF. CIT stratified at greater or lower than 12 h was found to be the strongest risk factor for DGF in LDKT. The average CIT for shipped kidneys that experienced DGF was 9.0 h compared to 6.8 h for shipped kidneys that did not have DGF ($P = 0.04$) [3]. Our data support similar finding of longer total ischemia time associated with a higher risk of DGF. Krishnan *et al.* 2016 observed CIT >4 h was associated with lower eGFR (a reduction >10 ml/min per 1.73 m) and twofold increased risk of graft loss and death-censored graft loss compared with CIT of 1–2 h [7]. Cold ischemia time varied from 4 to 22 h in the Australian Kidney Exchange owing to long distances and other unclear reasons, but we observed DGF in only two kidney exchange recipients out of 110 in our data. A possible explanation is that very stringent selection criteria for the Kidney exchange program led to a lower risk of DGF.

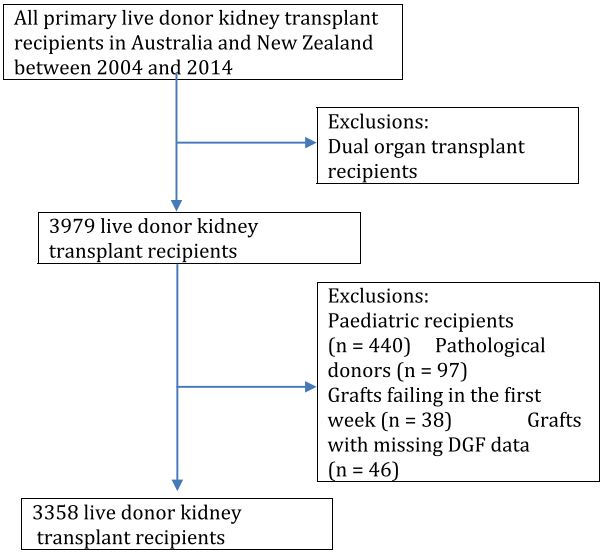

Figure 1 Study design: data collection

Right donor kidney accounts to 17% of DGF cohort in our study and the reasons for selection of right kidney are not collected by ANZDATA. Redfield *et al.* 2016 demonstrated a similar finding of higher risk of DGF in right-sided kidney (16.8%) versus left kidney (12.3%). Possible explanations for this finding include shorter renal vein and longer renal artery, which could be

Table 4. Predictors of eGFR at 1 year

	Coefficient	95% CI	*P*-value
Delayed graft function	−9.57	−13.5, −5.64	0.001
Donor characteristics			
Age (years)	−0.38	−0.44, −0.31	<0.001
Male sex	3.10	1.93, 4.26	<0.001
eGFR	0.16	0.12, 0.21	<0.001
Number of arteries	−3.16	−4.53, −1.80	<0.001
Recipient characteristics			
Age at transplant	−0.12	−0.17, −0.08	<0.001
Male sex	−1.2	−2.40, −0.01	0.049
Race category			
Caucasian	0 (referent)		0.01
Aboriginal/Torres Strait Islander	0.33	−8.01, 8.72	
Asian	0.30	−1.76, 2.37	
Māori	−6.12	−10.06, −2.18	
Pacific	−5.76	−10.23, −1.29	
Other	2.00	−3.65, 7.67	
Not reported	2.21	−8.50, 12.92	
Body mass index at transplant	−0.41	−0.54, −0.28	<0.001
Transplant characteristics			
Transplant Era			
−2004 to 2009	0 (referent)		<0.001
−2010 to 2015	2.16	0.96, 3.36	
Induction treatment	2.55	1.02, 4.08	0.001

Results of final multivariable model.

Figure 2 Overall graft survival by DGS status. Graft survival was not censored at death.

Table 5. Risk factors associated with overall graft survival

	Hazard ratio	95% CI	*P*-value
Delayed graft function	1.98	1.27, 3.10	0.003
Donor age	1.01	1.00, 1.02	0.02
Recipient characteristics			
Disease category			
Glomerulonephritis	1 (referent)		<0.001
Polycystic	0.70	0.51, 0.95	
Reflux nephropathy	0.74	0.51, 1.07	
Hypertension	1.01	0.63, 1.62	
Diabetes	1.84	1.34, 2.52	
Other	1.47	1.13, 1.92	
Ischemic heart disease	1.35	1.05, 1.74	0.02
Cerebrovascular accident	1.44	1.02, 2.05	0.04
Renal replacement therapy duration			
Pre-emptive	1 (referent)		0.008
<6 months	0.80	0.55, 1.16	
6 months to 1 year	1.14	0.81, 1.59	
1–5 years	1.40	1.09, 1.80	
5+ years	1.27	0.90, 1.80	
Peak panel reactive antigen assay	1.01	1.00, 1.01	0.003

Results of final multivariable model.

technically more complex anastomoses [3]. Vacher-Coponat *et al.* 2012 demonstrated right-sided deceased donor kidneys were associated with higher incidence of DGF and poor renal function, but this did not carry after 12 months post-transplant period [8].

Brook *et al.* [9] found longer ischemia time in laparoscopic donor nephrectomy group but no significant difference in DGF among various surgical techniques such as open versus laparoscopic donor nephrectomy, which is consistent with our analysis. The average length of hospital stay for LD kidney recipients with DGF was significantly longer, 13.9 days versus 5.9 days for recipients without DGF [3]. One suggestion could be two teams operating on donor and recipient simultaneously to reduce the ischemia time to less than 1 h in a recipient with other risk factors.

Keith *et al.* [10] demonstrated longer duration of renal replacement therapy prior to transplant is associated with more risk of DGF in DD transplants, which is similar to our analysis on LDKT recipients. Possible

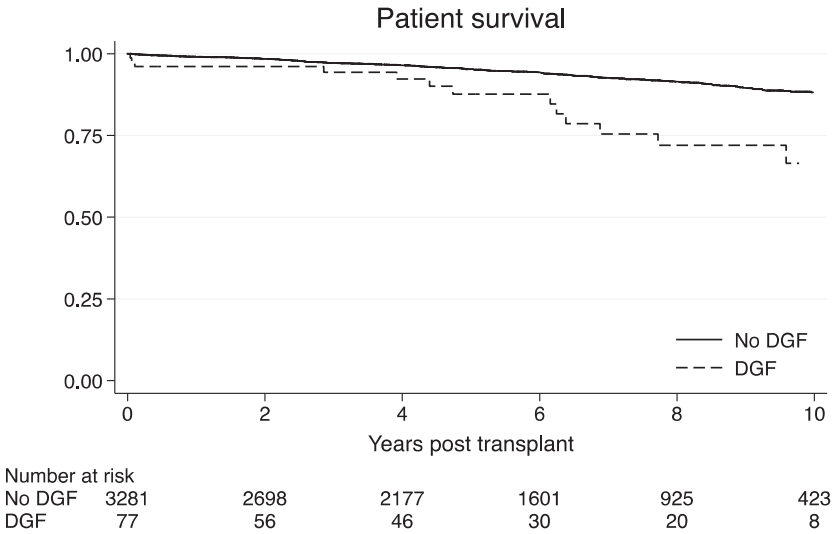

Figure 3 Overall patient survival by delayed graft function status.

Table 6. Risk factors associated with patient survival

	Hazard ratio	95% CI	*P*-value
Delayed graft function	2.14	1.21, 3.80	0.009
Recipient characteristics			
Age at transplant	1.05	1.04, 1.06	<0.001
Disease category			
Glomerulonephritis	1 (referent)		<0.001
Polycystic kidney disease	0.90	0.60, 1.34	
Reflux nephropathy	0.65	0.35, 1.23	
Hypertension	1.41	0.80, 2.52	
Diabetes	2.86	0.97, 4.15	
Other	1.78	1.22, 2.62	
Cerebrovascular accident	1.61	1.07, 2.40	0.02
Renal replacement therapy duration			
Pre-emptive	1 (referent)		0.001
<6 months	1.17	0.70, 1.98	
6 months to 1 year	0.93	0.53, 1.64	
1–5 years	1.84	1.27. 2.67	
5+ years	2.05	1.28, 3.30	
Peak panel reactive antigen assay	1.01	1.00, 1.01	0.01

Results of final multivariable model.

explanations for this finding include loss of residual native renal function, increased cardiovascular risk, unstable hemodynamics, and calcified atherosclerotic vessels.

In our analysis, DGF was associated with twofold increase in acute rejection at 6 months (odds ratio 2.37). Transplantation initiates a cascade of ischemia-reperfusion injury (IRI), results in activation of both innate and adaptive immune systems which further leads to an intense inflammatory response [11–13]. Multiple studies have suggested that IRI triggers an immune response through recognition of injury not only recognition of specific non self-antigens; which can lead to acute rejection [14]. Cell injury and/or apoptosis are important in the development of DGF and may also be responsible for subsequent acute rejection. Cold storage of donor kidneys might attenuate the inflammatory response but cannot abolish it completely [13].

Yarlagadda *et al.* [15] demonstrated 1.64-fold increase in risk of biopsy proven acute rejection in the DGF group compared to non-DGF group in DDKT, which is similar to our finding in LDKT. We could not

demonstrate a statistically significant association between DGF and immunological risk factors (i.e., re transplant, PRA, HLA matching, ABO incompatibility) or year of transplantation and immunosuppression.

Delayed graft function is associated with lower graft function in terms of eGFR in DD kidney transplant recipients [16]. We demonstrated a negative association between DGF and low eGFR at 1 year. The association between lower eGFR at 1 year with number of renal arteries more than 1[−3.16 ml/min (95% CI −4.53, −1.80)] may be explained by the decreased perfusion of watershed areas during transplantation. Recipients of Pacific Islander descent and Māori ethnicity had lower eGFR at 1 year [(−6.12 ml/min, 95% CI −10.6, −2.18) and (−5.76 ml/min, 95% CI −10.23, −1.29) respectively]. This may be because of their higher muscle mass in recipients of Pacific Islander descent and Māori ethnicity [17].

Delayed graft function associated with poor graft and patient survival is documented in DD kidney transplants [4,15,18,19]. Redfield *et al.* 2016 demonstrated DGF associated with inferior patient survival (hazard ratio – 2.3, 95% CI 2.1, 2.6; $P < 0.001$), and inferior allograft survival compared to no DGF (65% vs. 85%, $P < 0.001$) [3]. We found similar outcomes in our LD transplant recipients with DGF being associated with double the risk of graft failure [HR 1.98] and inferior patient survival (HR 2.14).

This study includes a large dataset from a transnational registry, a dataset from Australia and New Zealand which will let us generalize the findings to transplants in Australia and New Zealand; a comprehensive list of confounders in the dataset that allowed for adjustment for these confounders using multivariable models.

Despite these strengths, there were some limitations in our study. Owing to the variability in the definition of DGF, comparison with other studies is not easy. The DGF definition requiring dialysis might underestimate the actual rate of DGF in pre-emptive kidney transplants, as they might not require dialysis due their residual renal function. The total number of kidney transplant recipients with DGF included in this study was quite low hence some of the analyses are underpowered and confidence intervals are quite broad.

While the shortcomings of this study as with any registry study is that multiple confounding factors such as the intensity of immunosuppression, the presence of donor-specific anti-HLA antibody, systematic differences in the management of kidney transplant recipients between transplanting centers and clinicians, complexity of donor kidneys and practical aspects including theater availability, timing of surgery (e.g., concurrent vs. simultaneous operation), type of perfusion fluid, intraoperative technical complications, and prolonged anastomotic times were not collected.

Conclusions

Even though DGF is less frequent in LDKT than in DDKT, it is associated with a significant increase in poor short- and long-term outcomes. Modifiable risk factors include total ischemia time and the right kidney. Transplant units should take these results into consideration when planning a live kidney donor transplant procedure.

Authorship

MRM: Principal author- 85% contribution - Designed study, performed research and wrote the paper. SB: Co-author - Contributed important inputs to the study and paper. PC: Co-author - Analyzed data, performed research and contribution to the writing of the paper

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Conflict of interest

The authors have declared no conflict of interest.

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