Long-Term Outcome of Kidney Transplantation in Recipients with Focal Segmental Glomerulosclerosis

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Abstract

Background and objectives FSGS can recur after kidney transplantation and is associated with poor graft outcomes. We aimed to assess the incidence of FSGS recurrence post-transplant and determine the effect of graft source on recurrence and graft survival in patients with biopsy-proven FSGS.

Design, setting, participants, & measurements Using the Australian and New Zealand Dialysis and Transplant Registry, we assessed incidence of FSGS, the influence of donor type on the risk of FSGS recurrence, and graft loss in recipients with ESRD caused by primary FSGS using Kaplan–Meier and logistic regression analyses.

Results Between 1992 and 2011, 736 first kidney transplants were performed in 666 adults and 70 children (≤20 years old) with biopsy–proven primary FSGS. FSGS recurred in 76 (10.3%) patients. Younger age ($P<0.001$), nonwhite ethnicity ($P=0.02$), and having a live donor ($P=0.02$) were independent risk factors associated with recurrence. Median graft survival was significantly better for live donor compared with deceased donor grafts (14.8 versus 12.1 years; $P<0.01$). Disease recurrence predicted poor graft outcomes, with 52% (95% confidence interval, 40% to 63%) 5-year graft survival in the recurrence group compared with 83% (95% confidence interval, 79% to 86%) in the group without recurrent disease ($P<0.001$).

Conclusions FSGS recurrence after kidney transplantation was more common in live donor kidneys. Despite this, graft survival in live donor recipients was significantly better for both children and adults with FSGS. We propose that live donor transplantation should not be avoided in patients with FSGS.

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Introduction

FSGS is a common cause of steroid–resistant nephrotic syndrome (1,2), with 50%–70% of patients eventually progressing to ESRD requiring kidney transplantation (3). FSGS has been reported as having poorer transplant outcomes than most other causes of ESRD (4), largely because of disease recurrence in the transplanted kidney, affecting 15%–40% of pediatric transplants (5–10) and 4%–66% of adult transplants (7,11–16). Recurrence often occurs early, with nephrotic-range proteinuria appearing hours to days after transplantation (5,17). Known risk factors for FSGS recurrence include race (decreased risk for blacks and Hispanics) (18,19), initial steroid response (10), and possibly, donor age over 40 years old (11,14). Genetic FSGS is much less likely to recur (6,9,19,20). Factors reported not to affect recurrence include HLA mismatch (5,11,19), histologic subtype (21), time on dialysis (7), and type of immunosuppression (22).

There has long been controversy over the source of donor kidneys for patients with primary FSGS (23,24). Although live donor (LD) kidney transplantation is generally associated with improved graft survival, some centers avoid LDs for patients with FSGS because of concerns of a higher risk of disease recurrence in the transplanted kidney, resulting in poorer transplant outcomes (4,25–28). More recent research suggests that donor source has no effect on recurrence of FSGS after transplantation, but there is limited information available on the effect of donor source on long-term outcomes (19). Recent studies suggest that LD kidney transplant rates remain lower among patients with FSGS in both Europe (29% for FSGS versus 41% for hypodysplasia) (20) and North America (44% for patients with FSGS versus 53% for patients without FSGS) (4).

In this study, we reviewed the association of graft source on recurrence and graft outcomes in pediatric and adult patients with primary FSGS in our region using the Australian and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Materials and Methods

Data Sources and Study Sample

Data were obtained from the ANZDATA Registry on all patients with primary FSGS who received their first kidney transplant during the 20-year period between January of 1992 and December of 2011. The ANZDATA Registry collects extensive information on all types of RRT in Australia and New Zealand. Data collection is mandatory and occurs yearly, with

significant events (death and graft loss) being reported monthly. For the purpose of our study, we collected deidentified data on donor characteristics (age, race, HLA typing, and deceased donor [DD] versus LD), recipient characteristics (age, race, HLA typing, diagnoses, time on dialysis, time of transplant, and prior grafts), and graft outcomes, including recurrence of primary disease (biopsy proven) and graft failure. Transplants performed before 1992, second or subsequent kidney transplants, and transplants for secondary FSGS were excluded from the analysis. The ANZDATA Registry does not record genetic testing results; hence, we were unable to exclude patients with underlying genetic mutations associated with FSGS from the cohort. The outcomes were assessed separately for adults and children, with pediatric recipients defined as ≤20 years of age. All recurrences were histologically proven, and the date of onset of recurrent disease was defined either clinically by the onset of nephrotic-range proteinuria with a decrease in serum albumin or as the date of histologic confirmation on tissue biopsy (renal biopsy or graft nephrectomy).

Ethics approval for this study was waived by the institutional ethics committee because of the deidentified nature of the collected data. The clinical and research activities being reported are consistent with the Principles of the Declaration of Istanbul as outlined in the Declaration of Istanbul on Organ Trafficking and Transplant Tourism.

Analytic Variables and Outcome Measures

Data are expressed as means with SDs unless otherwise specified for non–normally distributed variables. Chi-squared test was used for univariate analysis of categorical data, and two–tailed *t* test was used for continuous variables. Kaplan–Meier analysis was performed to compare graft survival according to recurrence of FSGS and donor or recipient characteristics. Independent predictors of FSGS recurrence were assessed by logistic regression and included the following variables: donor source, sex, HLA mismatch, donor and recipient ages, ethnicity, and transplant era. Transplant era, as determined at the date of transplantation, was defined according to the prevalent immunosuppressive regimen used within Australia and New Zealand as follows: era 1 (January of 1992 to December of 1998): cyclosporin and azathioprine; era 2 (January of 1998 to December of 2005): cyclosporin and mycophenolate; and era 3 (January of 2006 to December of 2011): tacrolimus and mycophenolate. Data were analyzed using Stata10 for Windows (StataCorp., College Station, TX), with P values <0.05 considered statistically significant.

Results

Cohort Demographics

In total, 13,903 kidney transplants were performed in Australia and New Zealand over the 20-year period between January of 1992 and December of 2011, with 904 transplants in 836 patients with biopsy-proven FSGS. After exclusion of patients with secondary FSGS, 736 recipients with a diagnosis of primary FSGS who underwent their first transplant were included in this study. Seventy (9.5%) were pediatric recipients.

LD rates for both adult and pediatric recipients with FSGS (34% in adults and 64% in pediatric patients) were comparable with LD rates for recipients with ESRD secondary to other diseases (33% in adults and 63% in pediatric patients). In adult patients with FSGS, LD recipients were significantly younger than recipients of a DD kidney, and their donors were significantly older than DDs. There was no significant difference in HLA mismatch between LDs and DDs for either pediatric or adult recipients (Table 1).

In the majority of patients, standard immunosuppression consisted of a calcineurin inhibitor, an antimetabolite, and prednisolone, with mycophenolate replacing azathioprine in 1998 and tacrolimus replacing cyclosporin in 2006 (29).

Predictors of FSGS Recurrence

FSGS recurred in 76 (10.3%) patients and was more common in LD recipients compared with DD recipients (14% versus 8%; chi square =0.03). On the basis of reported date (seven patients with date of recurrence on the basis of post–transplant graft nephrectomy excluded), recurrence occurred most commonly in the first 2 years post-transplant in both children and adults (Figure 1), with no significant difference in time between LD and DD grafts (P=0.78). Disease recurrence was significantly more common in pediatric patients than in adults (36% versus 8%, respectively; $P<0.001$), but there was no significant difference in recurrence rate within the pediatric cohort on the basis of the donor source (P=0.58) (Table 1).

Logistic regression analysis showed a significantly higher risk of recurrence in younger patients ($P<0.001$), nonwhite patients (P=0.02), and recipients of an LD transplant (P=0.02). HLA mismatch (at A, B, and DR loci) and sex were not statistically significantly associated with recurrence (Table 2). Transplantation after 1998 (eras 2 and 3) was associated with a lower risk of recurrence (era 2: P=0.02; era 3: P=0.06).

Predictors of Graft Survival

Patients with a diagnosis of FSGS had poorer graft survival compared with the general transplant population. For all transplants in adults in Australia and New Zealand over this time period, 5-year graft survival in recipients with FSGS was significantly worse than that in recipients without FSGS (81% versus 88%, respectively; $P<0.001$). For pediatric patients, 5-year graft survival of recipients with FSGS was much worse compared with that in non-FSGS recipients (68% versus 93%, respectively; P=0.001).

For those with FSGS, donor source was significantly associated with graft survival. Median graft survival for the whole transplant cohort with primary FSGS was significantly better for LD versus DD grafts (14.8 versus 12.1 years; $P<0.01$), with 5-year graft survival of 85% for LD grafts and 76% for DD grafts ($P<0.001$) (Figure 2). In keeping with data from the whole cohort, pediatric recipients of LD grafts also had better outcomes, with 80% 5-year graft survival compared with 46% 5-year graft survival in DD recipients (P=0.03) (Figure 3).

Recurrence of FSGS post kidney transplantation was a strong predictor of graft outcomes. Disease recurrence predicted poor graft outcomes, with 52% (95% confidence interval, 40% to 63%) 5-year graft survival in the recurrence group compared with 83% (95% confidence interval, 79% to 86%) 5-year graft survival in the group without recurrent

Table 1. Patient demographics

Baseline characteristics	Pediatric Recipients		Adult Recipients	
	Deceased Donor	Live Donor	Deceased Donor	Live Donor
Total, *n* (%)	25 (36)	45 (64)	437 (66)	229 (34)
Men, *n* (%)	14 (56)	24 (53)	303 (69)	150 (66)
White, *n* (%)	15 (60)[a]	40 (89)[a]	357 (82)	192 (84)
Recipient age at transplant, yr, mean (SD)	13.6 (4.1)	14.4 (4.9)	47.0 (12.2)[b]	44.7 (13.6)[b]
Donor age, yr, mean (SD)	39.0 (21.2)	43.1 (10.0)	42.4 (17.3)[c]	49.2 (11.7)[c]
Mean HLA mismatches				
HLA-A	1.1	0.8	1.1	1.0
HLA-B	1.3	0.9	1.2	1.1
HLA-DR	0.9	0.8	0.7	1.0
FSGS recurrence, *n* (%)	10 (40)	15 (33)	29 (7)	22 (10)

P values compare deceased donor versus live donor within the cohort of only pediatric or adult patients.
[a]$P<0.01$.
[b]$P=0.03$.
[c]$P<0.001$.

disease ($P<0.001$) (Figure 4). The majority of graft loss occurred in the first 2 years postdisease recurrence, predominantly from DDs.

Discussion

In this large 20-year retrospective cohort study, we confirm findings from previous studies, which showed that patients transplanted for FSGS have poorer long–term graft survival compared with patients transplanted for other causes of ESRD. Multivariate analysis revealed that donor source was weakly, albeit significantly, associated with disease recurrence in adults and children. Importantly, despite this higher recurrence risk, LD recipients had significantly better long–term graft survival. As previously reported, we also found that recurrence of FSGS was more common in children than in adults, occurring in approximately one third of transplanted children. Disease recurrence occurred largely within the first 2 years after transplantation and remains a serious post–transplant complication, with one half of affected transplants failing within 5 years.

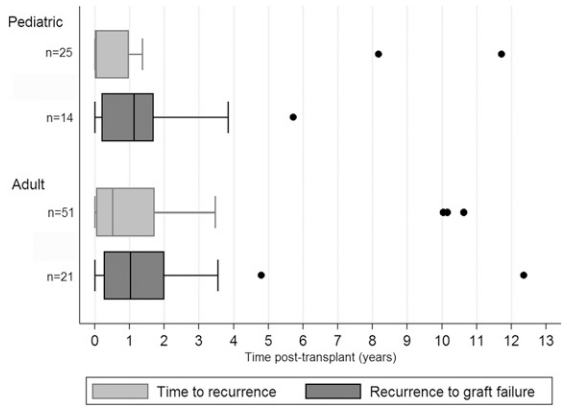

Figure 1. | Time to recurrence and time from recurrence to graft failure. Time to recurrence and time from recurrence to graft failure for pediatric and adult recipients with recurrent FSGS.

In our adult population, we found a recurrence rate of 7.5%. This is by far the largest published adult transplant cohort with biopsy–proven recurrent FSGS. Other smaller studies (n=18–77) report recurrence rates in adults of 4%–66% (7,11–16). Unfortunately, many of these studies do not differentiate between first and subsequent transplants, which may bias results given the significantly higher risk of recurrence after loss of a first transplant with recurrent disease. The data in this study include a large cohort followed during their initial transplant, which allows us to more accurately predict the risk of FSGS recurrence for adults in this geographic region.

Recurrence in the pediatric cohort was higher than that reported in a large United Network for Organ Sharing (UNOS) Study (34% versus 15%, respectively) (19). We postulate that this may be related to the large black and Hispanic cohort in the latter study, which had a lower recurrence rate compared with in other populations. The recurrence rate of FSGS in our pediatric population was comparable with that in smaller European studies with predominantly white populations (17). We found that recurrence was much more likely to occur in children than adults. Few studies have directly compared recurrence in adults and children, and they have involved smaller sample sizes, ranging from 25 to 107 patients (21,30,31). A small Korean study of 107 patients found no difference in recurrence rates between adults and children (32). In keeping with some other studies, we found white ethnicity to be associated with lower risk of disease recurrence. As a corollary, the nonwhite population in this cohort, which consists of mainly Asian and Indigenous patients, seems to be at higher risk of disease recurrence. We hypothesize that racial differences and variable sample size may account for the different findings of our study compared with those of previously published reports.

The pediatric subgroup had a much higher incidence of recurrence than adults, but in this group, there were no

Table 2. Multivariate analysis of risk factors for FSGS recurrence postkidney transplant

Covariate and Variable Level	Odds Ratio	95% CI	*P* Value
Recipient age	0.95[a]	0.93 to 0.97	<0.001
Recipient race			
White	1.0		
Nonwhite	2.10	1.14 to 3.87	0.02
Recipient sex			
Women	1.0		
Men	1.194	0.69 to 2.06	0.52
Donor age	0.99[a]	0.97 to 1.00	0.14
Donor type			
Deceased donor	1.0		
Live donor	2.04	1.11 to 3.72	0.02
HLA mismatch A			
0	1.0		
1	1.60	0.83 to 3.10	0.16
2	1.31	0.53 to 3.21	0.56
HLA mismatch B			
0	1.0		
1	1.06	0.51 to 2.21	0.87
2	1.46	0.60 to 3.57	0.40
HLA mismatch DR			
0	1.0		
1	0.56	0.31 to 1.01	0.06
2	0.57	0.23 to 1.40	0.22
Transplant era			
Era 1	1.0		
Era 2	0.48	0.25 to 0.91	0.02
Era 3	0.54	0.28 to 1.03	0.06

95% CI, 95% confidence interval.
[a]Age variable increment is 1 year.

significant differences in FSGS recurrence rates on the basis of donor source. Our results are similar to those of a UNOS Study that showed no association between donor source and risk of FSGS recurrence in children (19). In contrast, for the whole cohort, we found a modest increase in recurrence in LD compared with DD grafts on univariate analysis. This association remained significant after adjusting for a number of additional covariates, including recipient age, donor age, race, and HLA mismatch. Furthermore, transplant era was an important determinant of risk of recurrence, with a lower risk after the introduction of mycophenolate– and tacrolimus–based immunosuppressive regimens. Although we cannot exclude another unmeasured factor related to patient selection that would affect these results (for example, an underlying genetic cause for the FSGS), on the basis of this analysis, there does seem to be a weak, albeit significant, association between donor source and FSGS recurrence.

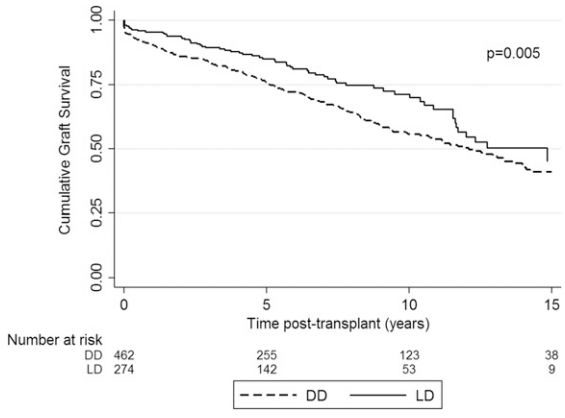

Figure 2. | Improved graft survival in patients with primary FSGS with living donors. Graft survival in patients with primary FSGS stratified by donor type. DD, deceased donor; LD, live donor.

We also investigated predictors of graft survival in patients with FSGS. In keeping with general reports on graft survival in both children and adults, recipients of LD kidneys had better median and 5-year graft survival rates compared with DD recipients. Short-term studies of outcomes of kidney transplantation for FSGS would only capture the adverse effects of early recurrence and graft loss, whereas long–term follow-up reveals the overall better outcomes for LD grafts. The finding of improved graft outcomes with LD grafts is in direct contrast to some previous studies (4). A large North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS) Study reported the loss of the usual LD survival advantage in FSGS. Our cohort differs from the American cohort, which had a large (approximately 30%) black population (4). Black children are less likely to have disease recurrence and more likely to receive a

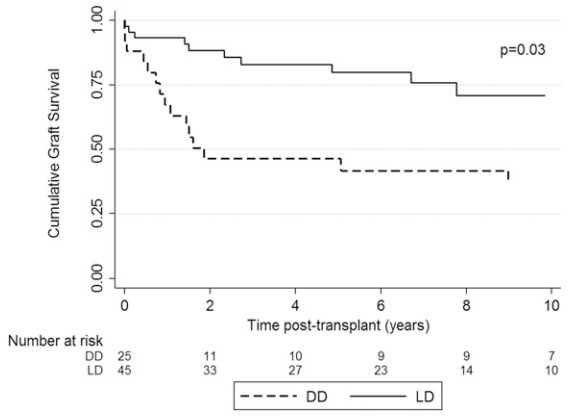

Figure 3. | Improved graft survival in pediatric patients with primary FSGS with living donors. Graft survival in pediatric patients with primary FSGS stratified by donor type. DD, deceased donor; LD, live donor.

DD kidney. We hypothesize that this may have been responsible for the improved DD outcomes observed in the NAPRTCS Study.

In keeping with our findings, a number of previous large European and North American studies have reported poorer graft survival for those with FSGS compared with other causes of ESRD (4,20). The main determinant for poorer outcomes in these studies is recurrence of FSGS post-transplantation with subsequent graft loss, a finding that was confirmed in this study. In our cohort, most recurrence occurred within the first 2 years post-transplant, and one half of those grafts were lost within 5 years. For those with recurrence who still had their grafts at 5 years, the rate of graft loss was similar for the recurrent and nonrecurrent groups, suggesting that the excess graft loss from recurrent disease manifests in the early post–transplant period. We found that Australian and New Zealand transplant centers do not avoid LDs for FSGS, with LD rates the same as in the non-FSGS cohort. In contrast, European and North American transplant cohorts have previously reported significantly lower LD rates for recipients with a primary diagnosis of FSGS compared with other causes of ESRD (4,20). This may have been driven by a number of factors, including (*1*) prior studies reporting poor outcomes for LD in FSGS related to the negative effect of disease recurrence on outcomes (4); (2) an argument that, given the poorer outcomes for FSGS grafts, using LDs in FSGS is an inefficient utilization of a scarce resource; or (*3*) the potential risk of related LDs of patients with genetic FSGS developing FSGS themselves in the future. In this cohort, whereas LD grafts were associated with a higher risk of recurrence, in the longer term, there were better graft outcomes. Thus, although the risk of the donors developing FSGS themselves remains a risk (meaning that all potential related LDs for recipients with FSGS should be considered for genetic testing), we suggest that arguments *1* and *2* above are no longer valid, meaning that there are significant benefits to LD transplantation.

This study has several strengths and some potential weaknesses. The major strength of the study is the size of the cohort. This is the largest adult cohort examined for incidence of post-transplant recurrence of FSGS. Another strength is that, in using the ANZDATA Registry, we have accessed all patients with biopsy-proven FSGS requiring renal transplantation in Australia and New Zealand. Previous work has validated the coding and reporting of data using the ANZDATA Registry. Most previously published studies assessing FSGS recurrence look only at graft loss caused by recurrence and do not report on which of these were biopsy-proven recurrences. Most previous large studies are unable to identify patients with recurrence that does not result in graft failure. One of the strengths of our study is that, in using the longitudinal data collected by the ANZDATA Registry, we were able to accurately estimate biopsy–proven recurrence rates in a large Australian and New Zealand cohort. Limitations of our study are common to any retrospective registry analysis and include inability to inspect individual patient records to identify other potential risk factors, such as treatment of recurrence. Additionally, capturing only biopsy-proven recurrence may lead to misclassification bias and underestimate the true incidence of FSGS recurrence, because some patients are diagnosed on clinical grounds alone. In Australia and New Zealand, standard therapy for FSGS recurrence generally consists of plasma exchange, and more recently, rituximab has been used as a supplementary agent. However, the ANZDATA Registry does not collect information on treatment of recurrent disease, and therefore, we were unable to explore the effect of treatment on graft outcomes.

In keeping with other studies, the outcomes of kidney transplants in patients with FSGS transplanted in Australia and New Zealand are inferior to those of patients transplanted for other causes of kidney disease. In those with FSGS, LD kidney transplant recipients have better graft outcomes compared with DD recipients. Children are at higher risk of FSGS recurrence and graft loss compared with the adult population. We propose that, after exclusion of genetic abnormalities that may predispose to FSGS, LD grafts should not be avoided in transplant recipients with primary FSGS.

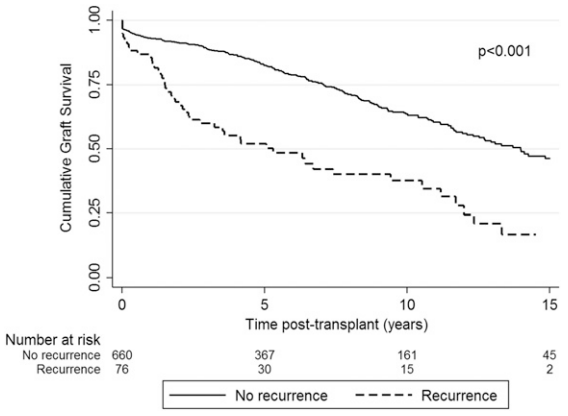

Figure 4. | Improved graft survival for FSGS recipients without recurrent disease. Graft survival in patients with primary FSGS stratified according to recurrence of FSGS.

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Disclosures

None.

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