Outcomes of renal transplantation in adult patients with primary focal segmental glomerulosclerosis: a single-centre experience over 5 decades

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Introduction

Primary focal segmental glomerulosclerosis (FSGS) is a common cause of nephrotic syndrome in adults and children and often leads to end-stage kidney disease.[1] The aetiology may be immune mediated or due to genetic mutations affecting glomerular proteins. Primary FSGS frequently recurs following renal transplantation, with published reports of 30–50% of patients being affected.[2,3] A number of genetic mutations confer a very low risk of recurrent disease, and a more detailed understanding of the patient population is required in order to individualise pre-transplant counselling, and to identify patients at high risk of recurrence.[4]

Materials and methods

We performed a retrospective database search of all patients who received renal transplants at our centre since 1981 (n=3,908 transplants in n=3,533 patients) with end-stage renal failure (ESRF) due to primary FSGS. A detailed case-note review was undertaken to exclude patients with secondary FSGS. We evaluated the course of their native kidney disease, and their transplant outcomes including the incidence of recurrent FSGS and graft survival. The diagnosis of recurrent FSGS was made in patients with supportive transplant histology and proteinuria.[1]

Results and discussion

We identified 106 patients with primary FSGS who received renal transplants, representing approximately 3% of the transplant population. Detailed follow-up data were available for 75 patients, with a median follow-up time of 84 (±82) months. Of these patients, 48 (63%) were male and 27 (37%) were female, which reflects the higher preponderance of FSGS (1.5 times higher) in men in the general population. Median age was 43 (±18) years at time of transplantation and, where known, 30.5 (±67.5) years at the time of FSGS diagnosis. 66.7% (50) of the patients were Caucasian and 33.3% (25) were non-Caucasian (including Pakistani, Indian, Bangladeshi, Black Caribbean or other Black, or other Asian background, or did not state ethnicity). Genetic analysis revealed mutations in six patients: ACTNS4, NPHS2, ACTN4, INF-2 (n=2) and NUP107, but this analysis was not available for the majority of patients in our cohort. 52% of transplants were from deceased donors and 48% were from live donors.

In all patients with functioning grafts, the median graft estimated glomerular filtration rate (eGFR) was 46 mL/min and urine albumin to creatinine ratio (ACR) was 9.3 at median 96 months post-transplant.

We identified recurrent FSGS in 13 (17.3%) of patients. Recurrent disease was more common in young Caucasian men and typically occurred early post-transplant (median 1 month), but was observed as late as 3 years post-transplant. Recurrent disease was treated with plasma exchange (nine times) and/or rituximab (three times) in addition to maintenance immunosuppression with calcineurin inhibitor, anti-proliferative and corticosteroids. Despite treatment, recurrent disease led to graft failure in 10/13 cases, within median <1 month. No cases of recurrent disease occurred in patients with an identified genetic mutation.

Conclusion

Our study shows that the rate of recurrent FSGS observed in our centre over 5 decades is much lower than published rates (16.3%), but that recurrent disease is likely to lead to graft loss. Recurrent FSGS occurred more commonly in young Caucasian men. This information will guide more individualised risk counselling to our multi-ethnic urban population prior to renal transplantation.[4] ■

Conflicts of interest

None declared.

References

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