

Original Article Urology and Renal Surgery

Long-term follow-up data of living kidney donors with pre-existing pre-diabetes

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Received: 20 November 2025

Accepted: 24 January 2026

Published: 13 March 2026

DOI

10.25259/WARM_30_2025

Quick Response Code:



ABSTRACT

Objectives: Renal transplantation is the best option for patients suffering from end-stage renal disease. The global epidemic of end-stage renal disease has resulted in a widening gap between the supply and demand of kidney donors. Thus, allowing donors from expanded donor criteria, which included a group of donors with pre-diabetes. We did a retrospective study to compare the long-term post-operative kidney functions, development of diabetes and hypertension, and proteinuria, of living donors having pre-diabetes at the time of donation with those donors without any evidence of pre-diabetes or diabetes at our center in the past 10 years.

Material and Methods: Retrospective data were collected for living donor kidney transplants between January 2014 and December 2024 at our center. Study population: We included all the Kidney donors with no comorbidities who underwent donor nephrectomy at our center. We did not consider pre-diabetes as a comorbidity, and donors diagnosed with pre-diabetes were included in our study. Donors with other co-existing comorbidities such as hypertension and hypothyroidism were excluded. Donors were divided into two groups based on the presence of pre-diabetes in them. Two hundred and fifty-four donors were included in our study and were divided into two groups. Group 1 included 36 donors who had pre-diabetes at the time of donation, and Group 2 included 218 donors those who had no comorbidities (no pre-diabetes also) at the time of donation. We measured serum creatinine, 24 h urinary creatinine clearance, urinary proteins, urinary albumin, blood pressure, and the development of diabetes mellitus.

Results: There was no significant difference observed for the change in serum creatinine or 24-h urinary creatinine clearance between Group 1, having pre-diabetes at the time of donation, and Group 2, which did not have pre-diabetes or any other comorbidity at the time of donation. Nor was there any significant difference in blood pressure in Group 1 and Group 2. There was a significant difference in the development of significant proteinuria and albuminuria between the two groups. Furthermore, the development of drug or insulin-dependent diabetes was significantly higher in Group 1.

Conclusion: Pre-diabetic donors may be acceptable but require closer monitoring. As our study did not show evidence of deterioration in kidney function in them post donor nephrectomy.

Keywords: Diabetes, Donor nephrectomy, Extended donor criteria, Long term followup, Pre-diabetes

INTRODUCTION

Renal transplantation is the best option for patients suffering from end-stage renal disease; transplant not only improves survival but also the quality of life when compared with dialysis. As there is a scarcity of cadaveric donors, a living donor transplant is a feasible option.^[1-4]

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The global epidemic of end-stage renal disease has widened the gap between the supply and demand of living kidney donors.^[5] Thus, allowing living kidney donation using expanded donor criteria, which also allows kidney donation from donors having pre-diabetes.

We wanted to evaluate the health of the donors in long-term follow-up, those who had pre-diabetes at the time of donation.

We did a retrospective study to compare the long-term post-operative kidney functions, development of diabetes, hypertension, and proteinuria, in living donors having pre-diabetes at the time of donation, and compared the results with those donors that did not have any evidence of pre-diabetes or diabetes at our center in the past 10 years.

MATERIAL AND METHODS

Between January 2014 and December 2024, we at the Institute of Liver and Biliary Sciences, New Delhi, did 367 living donor kidney transplants. We attempted to contact all 367 donors (or donor families). Then, we filled out our questionnaire regarding current health and medications (especially diabetes mellitus [DM] drugs), current serum creatinine and HbA1c (Hemoglobin A1c) levels, urinary protein excretion, and the development of hypertension.

As per the American Diabetes Association guidelines, the definition of Pre-diabetes is an HbA1c level that ranges from 5.7 to 6.4. But to make donor criteria stricter, we took the pre-diabetes range of 5.5-6.0 for our study.

In the future, if guidelines are made, then the stricter upper limit of HbA1c can be followed in kidney donor criteria.

Study population

Inclusion criteria

All the kidney donors with no comorbidities who underwent donor nephrectomy at our center. Donors with pre-diabetes were included in our study.

Exclusion criteria

Donors with other co-existing comorbidities such as hypertension and hypothyroidism were excluded.

Pre-diabetes was not considered as an exclusion criterion.

We divided donors into two groups based on the presence of pre-diabetes in them.

We had a total of 367 donors, out of whom 65 donors were lost to follow-up, as 4 of them died and 61 were either unable or unwilling to follow up at our center. The remaining 302 donors agreeing to participate in our study were included in the data calculations [Figure 1].

Out of these 302 donors, patients suffering from comorbidities (e.g., hypertension controlled on medication) other than pre-diabetes at the time of kidney donation were 48, and they were excluded from the study. The remaining 254 donors were divided into two groups. Group 1 included 36 donors who had pre-diabetes at the time of donation, and Group 2 included 218 donors those who had no comorbidities (no pre-diabetes also) at the time of donation. The flowchart depicts the donors.

Protocol: All the donors included in the study were called in for an outpatient department visit, and tests were done. Blood pressure was measured. The 24-h urinary was assessed for creatinine, total protein, and albumin. Blood was assessed for creatinine. The adequacy of the urine collection was determined by the total creatinine excretion. Acceptable specimens contained 1.0–2.0 g/24 h for men and 0.8–1.8 women.

To address the renal implications of pre diabetes after donation, we studied the association with incident hypertension, proteinuria, serum creatinine rise, and rate of estimated glomerular filtration rate (eGFR) change after donation between two groups of donors.

We defined proteinuria by a urinary albumin excretion rate >30 mg/g creatinine, 24 h urinary protein >200 mg/day, or ≥2+ on urine dipstick tests. To calculate the eGFR, we used the Chronic Kidney Disease Epidemiology Collaboration equation (14–15). Serum creatinine values after at least 3 months of donation were used because the goal was to model long-term trends in kidney function.

Statistical analysis

Continuous covariates were summarized as mean (standard deviation [SD]). To test univariable differences between donors with and without pre-diabetes, we used *t*-tests (continuous variables) and Chi-square or Fisher's tests (categorical variables).

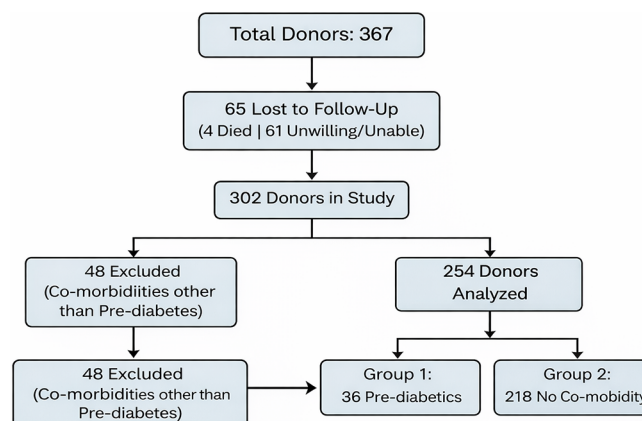


Figure 1: Flowchart representation of the study population criteria.

RESULTS

Renal function: Age-adjusted GFR for a solitary kidney, which was 46.8 (15.2)/1.73 m² in Group 1, and 48.2 (15.9) in Group 2, and did not have a significant difference. Cockcroft–Gault GFR was used for calculating GFR.

Urinary protein excretion: There was no difference in values for 24-h urinary protein and albumin excretion after donation between the two groups.

Blood pressure: For our study, hypertension was defined as blood pressure 140/90 mmHg or greater, or normotensive but managed with antihypertensive medication. No donors were hypertensive preoperatively. A total of 34 donors developed hypertension postoperatively, which was isolated systolic in 11 and diastolic in 4. Of the remaining 19 patients were normotensive on antihypertension medications.

Systolic blood pressure and diastolic blood pressure did not have a significant difference between Group 1 and Group 2. As 5 out of 36 donors of Group 1 developed hypertension in follow-up, out of which 4 were normotensive on antihypertension medications. Moreover, in Group 2, 29 patients out of 218 donors developed hypertension.

Development of diabetes

There was a significant difference in the development of drug/insulin-dependent diabetes in both groups. Eight patients out of those 36 pre-diabetics, i.e., Group 1 donors, were now dependent on a drug or insulin for DM. Moreover, out of 218 donors in Group 2, 15 donors had developed drug-dependent DM [Tables 1 and 2].

There was no significant difference observed for the change in serum creatinine or 24-h urinary creatinine clearance between the values of Group 1 and Group 2 after donation. Nor is there a significant difference in blood pressure between the groups.

There was a significant difference in the development of proteinuria and albuminuria; it was significantly higher in Group 1 when compared to Group 2.

The percentage of donors developing drug or insulin-dependent diabetes after the donation was significantly higher in group 1 when compared to group 2.

We tried to follow up on these 254 renal donors for at least 10 years. The mean time of 6.2 years' follow-up post donor nephrectomy is presented in this study.

DISCUSSION

Our study showed that there was no significant difference in serum creatinine or 24-h urinary creatinine clearance in

Table 1: Donor baseline characteristics.

Factor	Group 1 (n=36)	Group 2 (n=218)	P-value
Age, median (IQR)	51 (43–58)	54 (43–59)	0.50
Sex, male (n, %)	9 (25%)	47 (21%)	0.59
Ethnicity (n, %)			
Indian	35 (97%)	210 (96%)	
Others	1 (3%)	8 (4%)	
Smoking (n, %)			
Never	34 (94%)	204 (93%)	0.36
Current	Nil	Nil	
Former	2 (6%)	14 (7%)	
Family history of diabetes mellitus (n, %)	8 (22%)	43 (19%)	0.63
BMI (kg/m ²), median (IQR)	26.5 (23.9–29.2)	26.2 (23.9–29.0)	0.75

p < 0.05 is significant. IQR: Interquartile range, BMI: Body mass index

Table 2: Results of donors.

	Group 1 (n=36)	Group 2 (n=218)	P-value
Serum creatinine (mg/dL)	0.94 (0.17)	0.90 (0.16)	0.19
Creatinine clearance (mL/min/1.73 m ²)	68.2 (19.1)	66.4 (18.8)	0.60
Cockcroft–Gault glomerular filtration rate (mL/min/1.73 m ²)	46.8 (15.2)	48.2 (15.9)	0.61
Systolic blood pressure (mm Hg)	119.8 (13.4)	118 (13)	0.45
Diastolic blood pressure (mm Hg)	74.2 (10.2)	73.8 (9.8)	0.82
Urinary protein excretion (g/24 h)	2.48 (5.2)	1.18 (3.4)	<0.01
Urinary albumin excretion (mg/day)	214 (45.2)	38 (8.4)	<0.01
Development of DM (Drug/Insulin dependent)	8 (22%)	15 (6%)	0.0096 (<0.01)

p < 0.05 is significant (bold). Unit mean ± SE; SE: Standard error, DM: Diabetes mellitus

future follow-up of the donors who had pre-diabetes at the time of donation than those donors who did not have pre-diabetes or any other comorbidity at the time of donation. These findings of serum creatinine and 24 h urinary creatinine clearance are similar and in line with other studies that have assessed renal function >10 years after renal donation.^[6–13] In summary, our current study demonstrates that renal function is well preserved in both groups after donor nephrectomy.

In our study, urinary protein and albumin excretion were significantly higher in future follow-up in donors having pre-diabetes at the time of donation than in donors who did not have pre-diabetes or any other comorbidity at the time of donation. Many previous studies with a mean follow-up of 10–15 years^[6-13] showed similar prevalence post donor nephrectomy.

The significance of proteinuria and albuminuria in our present study is unclear, as we could not correlate increased proteinuria with any deterioration of renal function. As there was no significant difference in renal function in the follow-up period between the two groups. Moreover, as per our study, both proteinuria and albuminuria had no correlation with any deterioration of renal function.

Nevertheless, this finding suggests that patients with pre-diabetes before donation represent a subgroup at risk for the development of significant proteinuria after donation. The probable causes for proteinuria in our cases can be due to DM, hyperfiltration injury, nephrosclerosis, or new and unrelated renal disease.

There was no significant difference in the development of hypertension in our study groups. Our findings are similar to the meta-analysis and a few studies that studied the prevalence of hypertension post renal donation or nephrectomy due to other causes.^[6-13]

In our study, the development of drug or insulin-dependent diabetes was significantly higher in donors having pre-diabetes at the time of donation.

Still, the donor with pre-diabetes should not be denied from kidney donation if they are compliant enough to follow up regularly and get treated at the earliest sign of developing DM in the future. There is a lack of data regarding their long-term risks. This is evident in our study data, which show no significant difference in serum creatinine at long-term follow-up between the two groups.

Although DM has been clearly established as a major risk factor for the development of kidney disease and is the leading cause of end-stage renal disease,^[13] there are few studies examining the effects of pre-diabetes on the kidney.

The term “pre-diabetes” is applied in the setting of impaired glucose tolerance or HbA1c values between 5.5 and 6.5. They indicate a relatively high probability for the development of diabetes, microvascular, and macrovascular disease.^[3,14,15] Studies have shown that impaired insulin sensitivity and worsening glycemic status have been associated with an increased risk of new renal dysfunction, leading to increased odds of developing chronic kidney disease in diabetic patients when compared with normoglycemic individuals.^[16-20] Although the effects of pre-diabetes in the setting of a single kidney are not known, the presence of diabetes is associated

with an increased risk of developing renal disease in live kidney donors.^[21-25]

None of the studies till now have been able to prove a definite link between “pre-diabetes” and the risk of future kidney dysfunction. Suggesting that the absolute risk of developing a glycemia-related kidney abnormality in the donor having pre-diabetes was probably not high.^[26,27]

Our study showed that diabetes developed in 22% of pre-diabetic donors after a mean time of 6.2 years post kidney donation. However, there was no significant deterioration of renal function in these donors.

Limitations of the study

Our study is retrospective in nature, has a shorter duration of follow-up, and has attrition bias. Furthermore, there was a timely start to diabetes treatment among donors in our study who developed DM on follow-up.

Therefore, a longer follow-up study can rule out these biases and help formulate guidelines for kidney donation in pre-diabetic donors.

CONCLUSION

Pre-diabetic donors may be acceptable but require closer monitoring. As our study did not show evidence of deterioration in kidney function in them post donor nephrectomy.

Author contributions: ASJ: Contributed to the conceptualization and study design and was responsible for data collection and the overall conduct of the research work; CC: Performed the statistical analysis.

CTR number: 2108/41.

Ethical approval: Institutional Review Board approval is not required as it is a retrospective study.

Declaration of patient consent: Patient’s consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship: Nil.

Conflict of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Cosio FG, Alamir A, Yim S, Pesavento TE, Falkenhain ME, Henry ML, *et al.* Patient survival after renal transplantation: I. The impact of dialysis pre-transplant. *Kidney Int* 1998;53:767-72.
2. Meier-Kriesche HU, Port FK, Ojo AO, Rudich SM, Hanson JA, Cibrik DM, *et al.* Effect of waiting time on renal transplant outcome. *Kidney Int* 2000;58:1311-7.
3. Papalois VE, Moss A, Gillingham KJ, Sutherland DE,

- Matas AJ, Humar A. Pre-emptive transplants for patients with renal failure: An argument against waiting until dialysis. *Transplantation* 2000;70:625-31.
4. Bay WH, Hebert LA. The living donor in kidney transplantation. *Ann Intern Med* 1987;106:719-27.
 5. Anderson RG, Bueschen AJ, Lloyd LK, Dubovsky EV, Burns JR. Short-term and long-term changes in renal function after donor nephrectomy. *J Urol* 1991;145:11-3.
 6. Hakim RM, Goldszer RC, Brenner BM. Hypertension and proteinuria: Long-term sequelae of uninephrectomy in humans. *Kidney Int* 1984;25:930-6.
 7. Najarian JS, Chavers BM, McHugh LE, Matas AJ. 20 years or more of follow-up of living kidney donors. *Lancet* 1992;340:807-10.
 8. Talseth T, Fauchald P, Skrede S, Djøseland O, Berg KJ, Stenstrøm J, *et al.* Long-term blood pressure and renal function in kidney donors. *Kidney Int* 1986;29:1072-6.
 9. Vincenti F, Amend WJ Jr., Kaysen G, Feduska N, Birnbaum J, Duca R, *et al.* Long-term renal function in kidney donors. Sustained compensatory hyperfiltration with no adverse effects. *Transplantation* 1983;36:626-9.
 10. Watnick TJ, Jenkins RR, Rackoff P, Baumgarten A, Bia MJ. Microalbuminuria and hypertension in long-term renal donors. *Transplantation* 1988;45:59-65.
 11. Williams SL, Oler J, Jorkasky DK. Long-term renal function in kidney donors: A comparison of donors and their siblings. *Ann Intern Med* 1986;105:1-8.
 12. Eberhard OK, Kliem V, Offner G, Oldhafer K, Fangmann J, Pichlmay R, *et al.* Assessment of long-term risks for living related kidney donors by 24-hour blood pressure monitoring and testing for microalbuminuria. *Clin Transplant* 1997;11:415-9.
 13. Kiproff DD, Colvin RB, McCluskey RT. Focal and segmental glomerulosclerosis and proteinuria associated with unilateral renal agenesis. *Lab Invest* 1982;46:275-81.
 14. Hostetter TH, Olson JL, Rennke HG, Venkatachalam MA, Brenner BM. Hyperfiltration in remnant nephrons: A potentially adverse response to renal ablation. *Am J Physiol* 1981;241:F85-93.
 15. Brenner BM, Cooper ME, de Zeeuw D, Keane WF, Mitch WE, Parving HH, *et al.* Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. *N Engl J Med* 2001;345:861-9.
 16. Nathan DM, Davidson MB, DeFronzo RA, Heine RJ, Henry RR, Pratley R, *et al.* Impaired fasting glucose and impaired glucose tolerance: Implications for care. *Diabetes Care* 2007;30:753-9.
 17. Aroda VR, Ratner R. Approach to the patient with pre-diabetes. *J Clin Endocrinol Metab* 2008;93:3259-65.
 18. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2008;31:S55-60.
 19. Chen J, Muntner P, Hamm LL, Fonseca V, Batuman V, Whelton PK, *et al.* Insulin resistance and risk of chronic kidney disease in nondiabetic US adults. *J Am Soc Nephrol* 2003;14:469-77.
 20. Nerpin E, Risérus U, Ingelsson E, Sundström J, Jobs M, Larsson A, *et al.* Insulin sensitivity measured with euglycemic clamp is independently associated with glomerular filtration rate in a community-based cohort. *Diabetes Care* 2008;31:1550-5.
 21. Fox CS, Larson MG, Leip EP, Meigs JB, Wilson PW, Levy D. Glycemic status and development of kidney disease: The Framingham Heart Study. *Diabetes Care* 2005;28:2436-40.
 22. Dengel DR, Goldberg AP, Mayuga RS, Kairis GM, Weir MR. Insulin resistance, elevated glomerular filtration fraction, and renal injury. *Hypertension* 1996;28:127-32.
 23. Perlstein TS, Gerhard-Herman M, Hollenberg NK, Williams GH, Thomas A. Insulin induces renal vasodilation, increases plasma renin activity, and sensitizes the renal vasculature to angiotensin receptor blockade in healthy subjects. *J Am Soc Nephrol* 2007;18:944-51.
 24. Mac-Moune Lai F, Szeto CC, Choi PC, Ho KK, Tang NL, Chow KM, *et al.* Isolate diffuse thickening of glomerular capillary basement membrane: A renal lesion in pre-diabetes? *Mod Pathol* 2004;17:1506-12.
 25. Pabico RC, McKenna BA, Freeman RB. Renal function before and after unilateral nephrectomy in renal donors. *Kidney Int* 1975;8:166-75.
 26. Chandran S, Masharani U, Webber AB, Wojciechowski DM. Pre-diabetic living kidney donors have preserved kidney function at 10 years after donation. *Transplantation* 2014;97:748-54.
 27. Tarabeih M, Na'ammih W. Comparison between female kidney donors with pre-diabetes and without diabetes in blood pressure measurements, kidney and diabetes biomarkers: A prospective cohort study. *J Nephrol* 2025;38:1201-8.

How to cite this article: Jadaon AS, Champawat C. Long-term follow-up data of living kidney donors with pre-existing pre-diabetes. *World Adv Renal Med.* 2026;2:23-7. doi: 10.25259/WARM_30_2025