BMC Nephrology

RESEARCH

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Hypertension after living kidney donation: incidence, predictors, and consequences

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Abstract

Living donor (LD) kidney transplantation is the optimal treatment for elective end-stage kidney disease (ESKD) patients. However, concerns about the long-term health risks for donors, including hypertension, remain incompletely defined. We aimed to determine the incidence and predictors of hypertension after kidney donation and to assess its association with estimated glomerular filtration rate (eGFR) trajectories over time. We conducted a retrospective cohort study of 300 LD who underwent nephrectomy between 1998 and 2020, after excluding those with pre-donation hypertension. Donors were followed for a mean of 6.4 ± 4.5 years The primary outcome was the development of de novo hypertension, defined by office blood pressure measurements exceeding 140/90 mmHg or initiation of antihypertensive therapy. Predictors of post-donation hypertension were evaluated using Cox regression analysis, and the association with longitudinal changes in eGFR was assessed using linear mixed-effects models. Hypertension developed in 30% of the cohort, with an incidence of 21% at 5 years and over 50% at 15 years post-donation. Higher body mass index (BMI) (HR 1.074 per Kg/m2, $P=0.039$), dyslipidemia (HR 1.959, $P=0.035$), and elevated systolic (HR 1.030 per mmHg, $P=0.009$) and diastolic blood pressure (HR 1.036 per mmHg, $P=0.039$) were independently associated with an increased risk of hypertension post-donation. The development of hypertension was associated with a modest but statistically significant decline in eGFR over time (-0.03 vs. +0.40 ml/min/year, $P=0.026$). No donor progressed to ESKD during follow-up and cardiovascular events were rare in the cohort. In conclusion, hypertension was a frequent complication post-donation in this Southern European cohort and was associated with modifiable risk factors, as higher BMI and dyslipidemia. While the associated decline in renal function over time is modest, these findings highlight the importance of long-term monitoring and proactive management of cardiovascular risk factors in living kidney donors. These results should inform pre-donation counseling, post-donation follow-up strategies, and further research into interventions that may mitigate hypertension risk while preserving donor health.

Keywords Living kidney donor, Hypertension, Renal function, eGFR, Nephrectomy, Cardiovascular risk, Longitudinal study

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Introduction

Living donor (LD) kidney transplantation (KT) remains the best treatment for eligible patients with end-stage kidney disease (ESKD) [1–3]. Although living kidney donation does not provide direct medical benefits to the donor, the perceived risks are generally low and considered ethically acceptable [3]. However, emerging evidence suggests a small but measurable increase in risks such as hypertension, cardiovascular disease, ESKD, and mortality among donors [4–6]. As the global organ shortage persists, transplant programs are increasingly accepting donors with controlled hypertension or obesity, reflecting broader population trends [7]. Nevertheless, long-term data on these changing risks remain limited.

Reduction of renal mass has been linked to increased blood pressure and the development of hypertension in both animal models and humans with reduced nephron mass [8, 9]. Although a connection between nephron endowment, hypertension, and renal function has been proposed, these pathways are not well-defined in living donors [10, 11]. Kidney donation may increase the risk of developing hypertension through mechanisms such as hyperfiltration, altered vascular tone, and activation of the renin-angiotensin-aldosterone system [12–21]. However, it remains uncertain whether kidney donation independently increases the risk of hypertension or whether higher detection rates simply reflect closer medical follow-up of donors, a phenomenon known as 'surveillance bias.' [12–19].

Hypertension is a major cause of cardiovascular and renal morbidity in the general population and is commonly reported as a cause of ESKD in living donors, particularly late after donation [22]. However, its underlying mechanisms, diagnosis, and management in donors are not well characterized [23]. Recognizing these uncertainties, the Kidney Disease Improving Global Kidney Outcomes (KDIGO) Clinical Practice guidelines highlight the need for robust studies to evaluate the incidence and consequences of hypertension in living donors [24].

In this context, our study investigates the incidence of hypertension after living kidney donation in a Portuguese cohort, identifies its risk factors, and examines its impact on renal function over time. To achieve this, we analyzed the incidence and predictors of hypertension following donation and evaluated its association with annual changes in estimated glomerular filtration rate (eGFR) in living donors.

Methods

We retrospectively reviewed the clinical data of all adult living kidney donors who underwent nephrectomy at our center between January 1998 and January 2020 ($n = 365$). Of these, 55 donors with pre-donation hypertension were excluded (detailed characteristics and comparisons with the study cohort are provided in Table 1). An additional ten donors were excluded due to missing data on pre- and post-donation blood pressure. The final study cohort thus comprised 300 living kidney donors.

Medical records of the LDs were reviewed to extract relevant data, including baseline demographic, anthropometric, analytical, and clinical information. Hypertension was defined as an office blood pressure measurement exceeding 140/90 mmHg, a previous diagnosis of hypertension, or the use of antihypertensive medication. Ambulatory blood pressure monitoring (ABPM) was used to confirm or rule out hypertension in patients with high office blood pressure when diagnostic uncertainty existed, and in treated patients when there was doubt about blood pressure control. ABPM diagnostic thresholds followed ESC/ESH guidelines [25]: 135/85 mmHg for daytime average, 130/80 mmHg for 24-hour average, and 120/70 mmHg for nighttime average.

Dyslipidemia was defined as a total cholesterol ≥ 200 mg/dL, LDL cholesterol > 130 mg/dL, triglycerides > 150 mg/dL, HDL cholesterol < 40 mg/dL, or the use of lipid-lowering therapy. Obesity was defined as a Body Mass Index (BMI) ≥ 30 Kg/m^2, while overweight was defined as a BMI between 25 and 29.9 Kg/m^2. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-Epidemiology Collaboration (CKD-EPI) creatinine-based equation, ignoring race. The date of nephrectomy was considered the start of follow-up and all donors are offered lifetime annual follow-up appointments at our center.

The primary outcome of this study was the development of hypertension after donation. Donors were followed from the date of nephrectomy until one of the following occurred: death, ESKD, attaining 15-year follow-up, or end-of-study period (December 31, 2022).

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Hospital de Santo António. Informed consent was obtained from all donors prior to donation, and data confidentiality was strictly maintained throughout the study.

Statistical analysis

Continuous data were described using mean and standard deviation (SD) or median (interquartile range [IQR]), and categorical data were expressed as numbers (and percentages) as appropriate. Categorical data including were compared using Pearson chi-square test or Fisher exact test, and continuous variables were compared with Student's t-test or Mann–Whitney U-test, as appropriate. The primary outcome was the incidence of de novo hypertension, analyzed as a time-to-event approach and visualized as a Kaplan-Meier curve. Predictors of this outcome were explored through Cox regression models. Risk factors associated with pre-donation

Table 1 Baseline characteristics for the study cohort

	Total ***N*** **= 300**	**No HT** ***N*** **= 211 (70%)**	**HT** ***N*** **= 89 (30%)**	***P***	**Univariate HR (95% CI)**	***P***
Age, mean ± SD	45.6 ± 10.5	45.2 ± 10.7	46.6 ± 10.5	0.287	1.019 (0.999–1.039)	0.064
Age, n (%)				0.787		
< 40	89 (30)	65 (31)	24 (27)		Ref	
40–55	148 (49)	103 (49)	45 (51)		1.224 (0.746–2.011)	0.424
>= 55	63 (21)	43 (20)	20(22)		1.476 (0.813–2.679)	0.200
Sex F, n (%)	213 (71)	152 (72)	61 (69)	0.542	0.864 (0.552–1.352)	0.521
BMI Kg/m^2, mean ± SD	24.9 ± 3.2	24.5 ± 3.2	26.0 ± 3.0	**< 0.001**	1.132 (1.061–1.208)	**< 0.001**
BMI, n (%)				**0.001**		
<25	162 (54)	127 (60)	35 (39)		Ref	
25–30	117 (39)	68 (32)	49 (55)		1.997 (1.293–3.083)	**0.002**
>=30	21 (7)	16 (8)	5 (6)		1.216 (0.476–3.107)	0.682
Smoking habits, n (%)	52 (17)	44 (21)	8 (9)	**0.013**	0.461 (0.223–0.955)	**0.037**
Dyslipidemia, n (%)	30 (10)	17 (8)	13 (15)	0.084	1.997 (1.105–3.608)	**0.022**
ProtU 0.15–0.5 g/g, n (%)	83 (28)	64 (30)	19 (21)	0.112	0.831 (0.499–1.383)	0.476
Serum creatinine, mean ± SD	0.75 ± 0.16	0.74 ± 0.16	0.75 ± 0.16	0.805	1.338 (0.357–5.022)	0.666
Pre- donation eGFR ml/min/1.73m^2, mean ± SD	101.8 ± 14.7	102.1 ± 15.0	101.1 ± 14.0	0.570	0.993 (0.979–1.006)	0.294
Pre- donation eGFR ml/min/1.73m^2, n (%)				0.858		
<80	25 (8)	18 (9)	7 (8)		1.033 (0.474–2.250)	0.936
80–90	39 (13)	26 (12)	13 (15)		1.337 (0.738–2.420)	0.338
>=90	236 (79)	167 (79)	69 (78)		Ref	
Related donor, n (%)	200 (67)	132 (63)	68 (76)	**0.020**	1.434 (0.876–2.348)	0.151
Family history HT, n (%)	105 (35)	53 (25)	52 (58)	**< 0.001**	1.850 (1.198–2.856)	**0.006**
SBP, mean ± SD	120.4 ± 11.2	118.3 ± 11.0	125.4 ± 10.2	**< 0.001**	1.048 (1.029–1.067)	**< 0.001**
DBP, mean ± SD	72.7 ± 8.5	71.2 ± 8.4	76.3 ± 7.6	**< 0.001**	1.057 (1.030–1.085)	**< 0.001**
Total cholesterol, mean ± SD	193.6 ± 36.2	191.5 ± 35.0	198.6 ± 38.6	0.119	1.003 (0.998–1.009)	0.257

n - number; HT – hypertension; HR – hazard ratio; SD - standard deviation; F - female: BMI - Body Mass Index; ProtU - protein/creatinine ratio in the urine; eGFR - estimated glomerular filtration rate, SBP- systolic blood pressure; DBP- diastolic blood pressure

hypertension were analyzed by logistic regression models. Donor eGFR annual change between one-year to 15-years post-donation was assessed by univariate linear mixed regression model, that imputed subject-specific random effects (intercept and slope defined as eGFR at one-year and time in years, respectively) on an unstructured covariance matrix. The dependent variable was all eGFR measurements and the independent variable (de novo hypertension as a time-varying variable) was entered as 2-way interaction terms between them and the time (in years) variable and imputed as time-varying variable. A similar approach was used to analyze the annualized change in SBP and DBP.

To compare the incidence of hypertension in living kidney donors with that in the general population from Northern Portugal, we calculated standardized event rates (SERs) using the indirect standardization method, adjusted for age and sex. Specifically, the observed number of hypertension cases in the donor cohort was compared with the expected number of cases, calculated by applying the age- and sex-specific incidence rates from the Portuguese population cohort to the age and sex distribution of the living kidney donor cohort.

Statistical calculations were performed using STATA/MP, version 17.1 (Stata Corp, College Station, TX, USA). A 2-sided P-value < 0.05 was considered as statistically significant.

Results

A total of 300 living kidney donors were included, with a mean follow-up of 6.4 ± 4.5 years. The baseline characteristics of our study cohort are summarized in Table 1.

The mean age of the population at the time of donation was 45.6 ± 10.5 years, and more than 20% of the individuals were 55 years or older. Most donors were female (71%), and all donors but one were Caucasian. Overweight and obesity were present in 39% and 7% of donors, respectively, and 17% reported smoking habits. Two-thirds were related to the recipient, and 35% had a family history of hypertension.

During the follow-up, 89 patients (30%) developed hypertension. Donors who developed hypertension were comparable in age, sex, and pre-donation kidney function (creatinine and eGFR) to those who remained normotensive but had higher pre-donation BMI and systolic blood

Table 2 Cumulative incidence of hypertension post-donation

Timepoint	Incidence % (95% CI)
1 year	11.1 (8.0-15.3)
5 years	21.1 (16.6–26.6)
10 years	41.1 (33.8–49.5)
15 years	52.2 (41.9–63.4)

CI - confidence interval

pressure (SBP), were more likely to have dyslipidemia, to smoke, and to have donated to a first-degree relative.

The incidence of hypertension increased progressively, reaching 11% at 1 year, 21% at 5 years, 41% at 10 years, and over 50% at 15 years post-donation (Table 2). The median time to onset of hypertension was 14.6 years. The Kaplan-Meier curve (Fig. 1) illustrates the cumulative incidence of hypertension over time.

In the multivariable cox-regression analysis (Table 3), independent pre-donation predictors of de novo hypertension included higher BMI (HR 1.074 per 1 Kg/m2 increase, 95% CI 1.004–1.149, $P=0.039$), dyslipidemia (HR 1.959, 95% CI 1.050–3.656, $P=0.035$), and higher baseline SBP (HR 1.030 per 1 mmHg increase, 95% CI 1.007–1.053, $P=0.009$), and diastolic blood pressure (DBP) (HR 1.036 per 1 mmHg increase, 95% CI 1.002–1.067, $P=0.039$). This indicates a 3% increase in risk for each one mmHg increase in pre-donation SBP and a 3,6% increase in risk for each one mmHg increase in pre-donation DBP.

De novo hypertension was associated with a modest yet significantly greater annual decline in eGFR compared to normotensive donors (-0.03 vs. +0.40 ml/min/1.73 m^2/year; difference −0.43 ml/min/1.73 m^2/year, $P=0.026$) (Table 4). No donor progressed to ESKD or CKD stage 5 during follow-up. At the last available assessment, two donors had reached CKD stage 4, four donors had CKD stage 3B, and 51 donors were classified as having CKD stage 3a, according to KDIGO CKD classification.

Donors with hypertension had a higher annual increase in systolic blood pressure (+0.65 vs. +0.18 mmHg/year; $P=0.024$), while diastolic blood pressure changes did not differ significantly. The mean (±SD) SBP was 117.8 ± 0.3 mmHg in the group without hypertension and 132.7 ± 2.5 in the group with de novo hypertension.

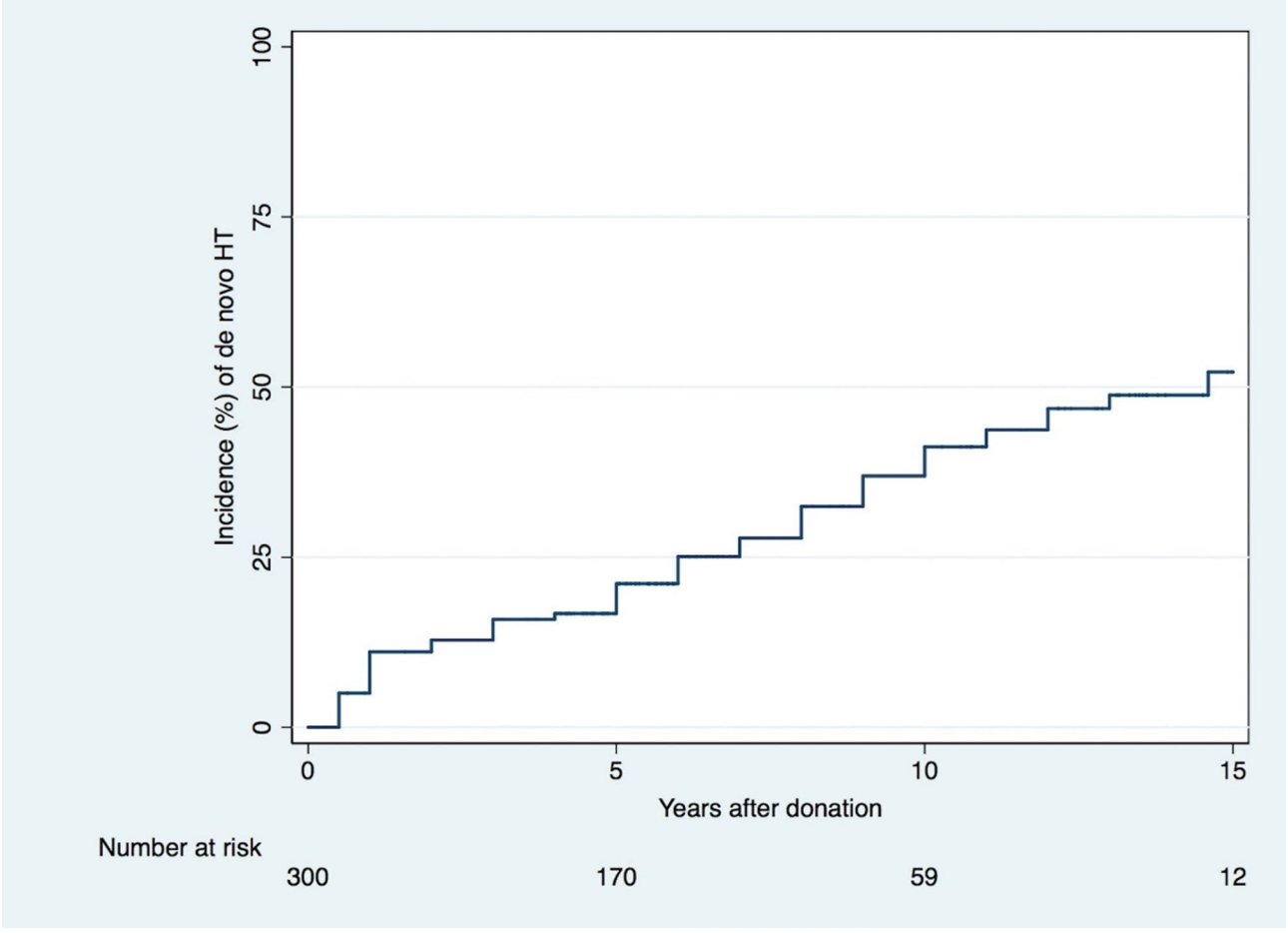

Fig. 1 Kaplan-Meier curve depicting the cumulative incidence of de novo hypertension in the living kidney donor cohort during follow-up

Table 3 Independent predictors of de novo hypertension-multivariable analysis*

	Multivariable HR (95% CI)	*P*
Age (years)	-	ns
< 40	-	ns
40–55		
>= 55		
Sex F	-	ns
BMI Kg/m^2	1.074 (1.004–1.149)	**0.039**
BMI	-	ns
<25		
25–30		
>=30		
Smoking habits	-	ns
Dyslipidemia	1.959 (1.050–3.656)	**0.035**
ProtU 0.15–0.5 g/g	-	ns
Serum creatinine	-	ns
Pre- donation eGFR ml/min/1.73m^2	-	ns
Pre- donation eGFR ml/min/1.73m^2	-	ns
<80		
80–90		
>=90		
Related donor	-	ns
Family history HTA	-	ns
SBP	1.030 (1.007–1.053)	**0.009**
DBP	1.036 (1.002–1.067)	**0.039**
Total cholesterol	-	ns

HR – hazard ratio; ns - non-significant; F - female: BMI - Body Mass Index; ProtU - protein/creatinine ratio in the urine; eGFR - estimated glomerular filtration rate, SBP- systolic blood pressure; DBP- diastolic blood pressure

*All variables in Table 3, including age and sex, were included in the multivariable model, regardless of univariate significance (Table 1). We did not report the non-significant ($P > 0.05$) HR (95% CI)

Table 4 Annual change in eGFR according to de novo hypertension status (time-varying) (data available in 264 donors)

De novo HT	Yearly change in eGFR, ml/min	*P*
No	+ 0.40 (-0.03; +0.84)	
Yes	-0.03 (-0.55; +0.50)	
Difference	-0.43 (-0.81; -0.05)	**0.026**

HT – hypertension; eGFR - estimated glomerular filtration rate

Overweight and obesity were significantly more prevalent among donors who developed hypertension throughout follow-up. Additionally, at 10 years post-donation, dyslipidemia was significantly more common among donors with hypertension compared to those without hypertension (53% vs. 24%, $P = 0.002$). In contrast, the prevalence of proteinuria and smoking did not differ significantly between the groups during the follow-up period.

Beyond these metabolic risk factors, we also evaluated post-donation cardiovascular events within the cohort. The very low number of events precluded meaningful statistical analysis. Within the main cohort, one donor without post-donation hypertension experienced an acute coronary syndrome with unremarkable coronary angiography. Another donor who developed hypertension post-donation suffered sudden death without a confirmatory autopsy.

To contextualize the incidence of hypertension in our donor cohort, we compared our findings with those of a contemporaneous, unselected cohort from northern Portugal [26] (Table 5). After adjusting for age and sex, the incidence of hypertension in our living donor cohort was markedly higher than expected based on population rates (Table 5). During the first five years post-donation, donors experienced a nearly fourfold higher incidence of hypertension (SER 3.75, 95% CI 2.94–4.86). Between 5 and 10 years post-donation, the incidence remained more than threefold higher (SER 3.25, 95% CI 2.12–4.76). At 10–15 years, the incidence was still approximately 2.3 times higher, although this difference did not reach statistical significance, likely due to the smaller number of donors at risk during this interval (SER 2.28, 95% CI 0.84–4.97).

Finally, as a sensitivity analysis, we characterized donors with pre-donation hypertension, who were excluded from the main analysis, and compared them with donors without hypertension (Supplemental Table S1). Moreover, we searched for pre-donation hypertension risk factors. Donors with pre-donation hypertension were generally older, had a higher BMI, and were more likely to be smokers. Additionally, they had lower eGFR

Table 5 Incidence of hypertension in the cohort of LKD and in a cohort of the Portuguese population from the North of Portugal

Time after donation	LKD cohort (*n*)	Observed cases (*n*)	Crude incidence (%)	Expected cases (*n*)*	Standardized event ratio (SER) (95% CI)	Excess cases (Observed – Expected)
0–5 years	300	57	19%	15.2	3.75 (2.94–4.86)	+ 41.8
5–10 years	161	26	16%	8.0	3.25 (2.12–4.76)	+ 18.0
10–15 years	55	6	11%	2.6	2.28 (0.84–4.97)	+ 3.4

*Expected cases were calculated by applying age- and sex-specific incidence rates from a Northern Portuguese population cohort (Pereira et al., 2012 [26]) to the age/sex distribution of the donor cohort

Abbreviations: LKD = living kidney donor; SER = standardized event ratio (observed/expected cases, adjusted for age and sex); CI = confidence interval

at the time of donation. In the multivariable analysis, age, BMI and SBP emerged as significant risk factors of pre-donation hypertension.

Discussion

In this Southern European cohort of 300 living kidney donors with a mean follow-up of over six years, we found that hypertension was common, affecting 30% of donors, with an incidence reaching 21.1% at five years, and over 50% at 15 years. Key pre-donation predictors of de novo hypertension included higher BMI, dyslipidemia, and elevated SBP, and DBP underscoring the importance of modifiable cardiovascular risk factors in this setting. Additionally, donors who developed hypertension experienced a modest but statistically significant decline in eGFR over time compared to those who remained normotensive. However, the clinical relevance of this decline is likely limited.

Prior studies have reported inconsistent findings regarding hypertension risk after donation, often due to variations in definitions, follow-up durations, and comparator groups. Studies such those from Holscher et al. [12], Haugen et al. [27] and the seminal metanalysis by Boudeville et al. [19] demonstrated a higher incidence of hypertension in donors compared to matched non-donors. In contrast, the meta-analysis by O´Keeffe [28], the work by Sanchez et al. [23], and two recent large prospective, matched control study by Garg et al. [29] and Kasiske [30] found no clear evidence of an increased risk for hypertension or cardiovascular disease in living kidney donors. Our findings differ from these latter studies. Whereas these North American cohorts were predominantly composed of standard-risk White donors and employed matched controls with a median follow-up of 7–9 years, our study excluded donors with pre-existing hypertension but no other comorbidities, extended follow-up to 15 years, and used regional population data for comparison [26]. Under these conditions, we observed a three- to fourfold higher incidence of hypertension among Portuguese donors. Moreover, Portuguese donors may carry a higher baseline cardiometabolic burden, which could amplify long-term hypertension risk. Taken together, these differences suggest that living donor outcomes are not uniform across populations, and that region-specific data are crucial to inform donor counseling and surveillance strategies. The observation of a higher incidence despite this selection highlights a potential role of kidney donation itself in developing hypertension over time.

We identified modifiable metabolic factors, including higher BMI and dyslipidemia, as significant predictors of post-donation hypertension, underscoring the importance of these parameters in donor selection and follow-up. Additionally, higher pre-donation systolic and diastolic blood pressures were independent predictors of post-donation hypertension, with each 1 mmHg increase associated with a 3% and 3.6% higher risk, respectively. While these increments are modest per unit, they are clinically meaningful when evaluating donors with higher baseline blood pressures, highlighting the importance of thorough blood pressure assessment during donor evaluation. In contrast, although age was associated with pre-donation hypertension, it was not an independent predictor of post-donation hypertension in our cohort, emphasizing the prominent role of modifiable risk factors in the development of hypertension after nephrectomy. Post-donation hypertension also remained significantly associated with overweight and obesity throughout follow-up. Finally, we were unable to evaluate the relationship between post-donation hypertension and cardiovascular events due to the very low number of cardiovascular events observed in our cohort.

Regarding kidney function, donors who developed hypertension experienced a small but statistically significant decline in eGFR after diagnosing hypertension compared to those who remained normotensive. Although the clinical relevance of this decline is likely limited, the finding supports the need for continued monitoring of renal function in donors, particularly those who develop hypertension. Importantly, no donor progressed to end-stage kidney disease during follow-up, and only a small proportion (n = 2, 0.6%) developed stage 4 chronic kidney disease, consistent with literature indicating a low risk of severe kidney function decline in appropriately selected donors [31, 32].

While our study did not assess the pathophysiological mechanisms underlying the development of hypertension after kidney donation, our findings are consistent with proposed processes involving hyperfiltration, nephron hypertrophy, and neurohormonal activation [33, 34]. Although these compensatory adaptations are initially beneficial, they may contribute over time to detrimental effects, including disruption of the renin-angiotensin-aldosterone system, sodium balance, and progressive glomerulosclerosis [33, 34]. Notably, a long-term study of glomerular hemodynamics following kidney donation reported that glomerular hypertension did not emerge as a significant contributor [35]. Age-related physiological changes and the accumulation of additional cardiovascular risk factors may further amplify these effects, underscoring the importance of the adequacy of blood pressure monitoring and control in donors who develop hypertension. The association we observed between post-donation hypertension and modest eGFR decline warrants further investigation to clarify whether hypertension contributes to, or results from, reduced renal function. Although hypertension was linked to a decline in eGFR over time

in our cohort, causality cannot be established from these findings.

Several limitations should be acknowledged in our study. The retrospective design and the definition of hypertension based on either medication use or in-office blood pressure measurement may have introduced misclassification bias. The relatively modest sample size and the predominantly Caucasian population in our cohort may limit the generalizability of the findings to more diverse populations and the ability to detect associations with less frequent outcomes, such as major cardiovascular events. Furthermore, the analysis did not account for the severity of hypertension, the specific anti-hypertensive regimens used, or the adequacy of blood pressure control over time, factors that could potentially influence renal outcomes and should be assessed in future studies.

Despite these limitations, our study adds important insights from a Southern European perspective, reinforcing the need for long-term, structured follow-up of living kidney donors with a focus on cardiovascular risk management. Future research should explore interventions aimed at mitigating post-donation hypertension and preserving donor kidney function, including the potential role of nephroprotective therapies, such as renin-angiotensin system inhibitors and SGLT2 inhibitors, in this population.

In conclusion, we found that hypertension is common after kidney donation in our cohort, with modifiable risk factors such as elevated BMI and dyslipidemia playing a significant role in its development. Post-donation hypertension is associated with a modest but measurable decline in kidney function over time, highlighting the importance of long-term monitoring and proactive management of metabolic risk factors in living kidney donors. These findings should guide pre-donation counseling and post-donation monitoring strategies to support donor health and inform shared decision-making in living kidney donation.

Abbreviations

ABPM	Ambulatory Blood Pressure Monitoring
BMI	Body Mass Index
CKD	Chronic Kidney Disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
CI	Confidence Interval
CV	Cardiovascular
DBP	Diastolic Blood Pressure
ESKD	End-Stage Kidney Disease
eGFR	Estimated Glomerular Filtration Rate
HR	Hazard Ratio
HT	Hypertension
IQR	Interquartile Range
KT	Kidney transplantation
LD	Living Donor
ProtU	Proteinuria
SBP	Systolic Blood Pressure
SD	Standard Deviation

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-04513-5.

Supplementary Material 1

Author contributions

LM, SP, and JS participated in data collection. JM contributed to data collection, article design, and statistical analysis. JF and MA contributed to data collection, article design, and writing.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Specific consent to participate was not obtained as it was not required under national regulations either for consent to participate or for data publication, but at time of transplant all patients consent data their clinical data mighth be collected, analyzed and published in a anonymously manner. Additionally, ethics approval was deemed unnecessary in accordance with Portuguese legislation.

Consent for publication

Specific consent for publication was not obtained as it was not required under national regulations either for consent to participate or for data publication, but at time of transplant all patients consent data their clinical data mighth be collected, analyzed and published in a anonymously manner.

Competing interests

The authors declare no competing interests.

Received: 24 December 2024 / Accepted: 22 September 2025
Published online: 11 November 2025

References

1. Gruessner RWG, Gruessner AC. Solid-organ transplants from living donors: cumulative united States experience on 140,156 living donor transplants over 28 years. Transpl Proc. 2018;50(10):3025–35.
2. Park YH, et al. Comparison of survival probabilities for living-unrelated versus cadaveric renal transplant recipients. Transpl Proc. 2004;36(7):2020–2.
3. Reese PP, Boudville N, Garg AX. Living kidney donation: outcomes, ethics, and uncertainty. Lancet. 2015;385(9981):2003–13.
4. Muzaale AD, et al. Risk of end-stage renal disease following live kidney donation. JAMA. 2014;311(6):579–86.
5. Mjoen G, et al. Long-term risks for kidney donors. Kidney Int. 2014;86(1):162–7.
6. Garg AX, et al. Gestational hypertension and preeclampsia in living kidney donors. N Engl J Med. 2015;372(15):1469–70.
7. Ahmadi AR, et al. Shifting paradigms in eligibility criteria for live kidney donation: a systematic review. Kidney Int. 2015;87(1):31–45.
8. Bhathena DB, et al. Focal sclerosis of hypertrophied glomeruli in solitary functioning kidneys of humans. Am J Kidney Dis. 1985;5(5):226–32.
9. Haylor J, et al. Renal function and morphometry in the Dwarf rat following a reduction in renal mass. Nephrol Dial Transpl. 1996;11(4):643–50.
10. Novick AC, et al. Long-term follow-up after partial removal of a solitary kidney. N Engl J Med. 1991;325(15):1058–62.
11. Al Ammary F, et al. Risk of ESKD in older live kidney donors with hypertension. Clin J Am Soc Nephrol. 2019;14(7):1048–55.

12. Holscher CM, et al. Self-Reported incident hypertension and Long-Term kidney function in living kidney donors compared with healthy nondonors. Clin J Am Soc Nephrol. 2019;14(10):1493–9.
13. Lentine KL, et al. Understanding antihypertensive medication use after living kidney donation through linked National registry and pharmacy claims data. Am J Nephrol. 2014;40(2):174–83.
14. Lentine KL, Segev DL. Understanding and communicating medical risks for living kidney donors: A matter of perspective. J Am Soc Nephrol. 2017;28(1):12–24.
15. Lentine KL, et al. Consistency of Racial variation in medical outcomes among publicly and privately insured living kidney donors. Transplantation. 2014;97(3):316–24.
16. Lentine KL, et al. Racial variation in medical outcomes among living kidney donors. N Engl J Med. 2010;363(8):724–32.
17. Lentine KL, et al. Postdonation eGFR and New-Onset antihypertensive medication use after living kidney donation. Transpl Direct. 2019;5(8):e474.
18. Garg AX, et al. Cardiovascular disease and hypertension risk in living kidney donors: an analysis of health administrative data in Ontario. Can Transplantation. 2008;86(3):399–406.
19. Boudville N, et al. Meta-analysis: risk for hypertension in living kidney donors. Ann Intern Med. 2006;145(3):185–96.
20. Brenner BM, Garcia DL, Anderson S. Glomeruli and blood pressure. Less of one, more the other? Am J Hypertens. 1988;1(4 Pt 1):335–47.
21. Torres VE, et al. Blood pressure determinants in living-related renal allograft donors and their recipients. Kidney Int. 1987;31(6):1383–90.
22. Anjum S, et al. Patterns of End-Stage renal disease caused by Diabetes, Hypertension, and glomerulonephritis in live kidney donors. Am J Transpl. 2016;16(12):3540–7.
23. Sanchez OA, et al. Hypertension after kidney donation: Incidence, predictors, and correlates. Am J Transpl. 2018;18(10):2534–43.
24. Lentine KL, et al. KDIGO clinical practice guideline on the evaluation and care of living kidney donors. Transplantation. 2017;101(8S Suppl 1):S1–109.
25. McEvoy JW, et al. 2024 ESC guidelines for the management of elevated blood pressure and hypertension. Eur Heart J. 2024;45(38):3912–4018.
26. Pereira M, et al. Incidence of hypertension in a prospective cohort study of adults from Porto, Portugal. BMC Cardiovasc Disord. 2012;12:114.
27. Haugen AJ, et al. Increased long-term risk for hypertension in kidney donors - a retrospective cohort study. Transpl Int. 2020;33(5):536–43.
28. O'Keeffe LM, et al. Mid- and Long-Term health risks in living kidney donors: A systematic review and Meta-analysis. Ann Intern Med. 2018;168(4):276–84.
29. Garg AX, et al. Hypertension and kidney function after living kidney donation. JAMA. 2024;332(4):287–99.
30. Kasiske BL, et al. A prospective controlled study of metabolic and physiologic effects of kidney donation suggests that donors retain stable kidney function over the first nine years. Kidney Int. 2020;98(1):168–75.
31. Almeida M, et al. Longitudinal trajectories of estimated glomerular filtration rate in a European population of living kidney donors. Transpl Int. 2024;37:13356.
32. Textor SC, et al. Blood pressure and renal function after kidney donation from hypertensive living donors. Transplantation. 2004;78(2):276–82.
33. Choi SY, et al. Adaptive functional change of the contralateral kidney after partial nephrectomy. Am J Physiol Ren Physiol. 2017;313(2):F192–8.
34. Srivastava T, et al. Hyperfiltration-mediated injury in the remaining kidney of a transplant donor. Transplantation. 2018;102(10):1624–35.
35. Lenihan CR, et al. Longitudinal study of living kidney donor glomerular dynamics after nephrectomy. J Clin Invest. 2015;125(3):1311–8.

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