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Association between Living Kidney Donor Post-Donation Hypertension and Recipient Graft Failure

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Abstract

Background: Recipients of kidneys from living donors who subsequently develop ESRD also have higher graft failure, suggesting the two donor kidneys share risk factors that could inform recipient outcomes. Given that donor ESRD is rare, an earlier and more common post-donation outcome could serve as a surrogate to individualize counseling and management for recipients. Hypertension is a frequent event prior to donor ESRD; thus early post-donation hypertension might indicate higher risk of graft failure.

Methods: We studied SRTR data to quantify the association between early post-donation hypertension and recipient graft failure using propensity score-weighted Cox proportional hazards regression. We also examined the association between post-donation systolic blood pressure and graft failure.

Results: Of 37,901 recipients, 2.4% had a donor who developed hypertension within 2 years post-donation. Controlling for donor and recipient characteristics, recipients whose donors developed hypertension had no higher risk for graft failure (aHR 1.03, 95% CI 0.85-1.25, p=0.72). This was consistent among subgroups of recipients at higher risk for adverse outcomes due to hyperfiltration: African American recipients (aHR 1.10, 95% CI 0.70-1.73, p=0.68) and those with ESRD caused by hypertension (aHR 1.10, 95% CI 0.65-1.85, p=0.73) or diabetes (aHR 0.80, 95% CI 0.56-1.13, p=0.20). However, graft failure was associated with post-donation systolic blood pressure (per 10 mmHg, aHR 1.05, 95% CI 1.03-1.08, p<0.001).

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CMH, TI, CEH, KRJ, JMGW, FAA, DLS, and ABM participated in research design. CMH, TI, YY, and ABM participated in analysis. CMH, TI, CMH, TI, CEH, KRJ, JMGW, FAA, DLS, and ABM participated in writing of the paper. All authors participated in critical revision and approve of the final version.

Disclosure:

The authors declare no conflicts of interest.

Conclusion: Although post-donation systolic blood pressure is associated with graft failure, the reported diagnosis of hypertension as determined by the requirement for blood pressure treatment early post-donation did not portend a higher risk of recipient graft failure in the same way as eventual post-donation ESRD.

INTRODUCTION

Risk prediction for recipient outcomes following living donor kidney transplantation (LDKT) remains a challenge despite detailed pre-donation information available from living kidney donors. Interestingly, recipients of kidneys from living donors who subsequently develop end-stage renal disease (ESRD) also have higher graft failure,[1] suggesting the two donor kidneys share risk factors that could inform recipient outcomes. Given that donor ESRD is rare,[2,3] an earlier and more common post-donation outcome such as hypertension[4] could serve as a surrogate to improve and better individualize counseling and management for recipients.

Hypertension is one of the most frequent proximal causes of donor ESRD;[5,6] early post-donation hypertension might indicate pre-existing donor subclinical renal disease[7] which could increase the risk of graft failure for LDKT recipients. For example, the current literature on renal hyperfiltration in patients with ESRD suggests that hyperfiltration is associated with progressive kidney disease, particularly in those with ESRD caused by hypertension[8] or diabetes.[9] Recipients with hypertension- or diabetes-caused ESRD might therefore be at higher risk for graft failure associated with underlying subclinical renal disease in the donor kidney. Similarly, because there is an association between hypertension and glomerular hyperfiltration in African Americans,[10] African American LDKT recipients whose donors developed hypertension might be at higher risk for graft failure.

With robust early donor follow-up data becoming available through OPTN mandates,[11,12] we now have the opportunity to examine how early post-donation incident hypertension is associated with LDKT recipient graft failure. To do this, we used national registry data to compare LDKT outcomes in recipients whose donors did or did not develop early post-donation hypertension. We further examined how this association might be different in subgroups that might be more sensitive to donor kidney hyperfiltration, namely African American LDKT recipients and those with ESRD caused by hypertension or diabetes.

MATERIALS AND METHODS

Data source

This study used data from the Scientific Registry of Transplant Recipients (SRTR) standard analysis files, available as of 9/1/2018. The SRTR data system includes data on all donor, wait-listed candidates, and transplant recipients in the US, submitted by the members of the Organ Procurement and Transplantation Network (OPTN), and has been described elsewhere.[13] The Health Resources and Services Administration (HRSA), U.S. Department of Health and Human Services provides oversight to the activities of the OPTN and SRTR contractors.

Study population

We studied 45,441 adult LDKT recipients between 1/1/2008-3/1/2016 with 2-year follow up information for their living donors. We excluded recipients whose donors were missing follow-up on hypertension at all mandated follow-up reports (n=2346, 5.2%) and recipients whose donors had baseline hypertension (n=1358, 3.0%) or were missing data regarding baseline hypertension at the time of donation (n=143, 0.3%) (Figure 1). We also excluded recipients missing baseline data on BMI or cause of ESRD (n=3693, 8.8%); there were no other baseline characteristics with missing data.

Early post-donation incident hypertension

We defined early post-donation incident hypertension as a donor reporting hypertension at one or more follow-up reports mandated at 6-months, 1-year, and 2-years post-donation in response to the form question “Donor developed hypertension requiring medication.” Characteristics of recipients whose donors did not develop hypertension were compared to those whose donors developed early post-donation incident hypertension using chi-square tests and Wilcoxon rank-sum tests, as appropriate.

Propensity score weighting

Because we were interested in isolating the effect of early post-donation hypertension on graft outcomes, and because the donors who developed early post-donation incident hypertension and their recipients were quite different from recipients and donors who did not develop hypertension, we used inverse probability of treatment weighting using a propensity score, a method that seeks unbiased estimates of average treatment effects with observational data.[14]

We created a propensity score quantifying the probability of the LDKT recipient having a donor who developed early post-donation incident hypertension, using logistic regression. This model included recipient age as a linear spline with a knot at 40 years, sex, race, body mass index (BMI), cause of ESRD, eGFR at hospital discharge after LDKT; and donor age, sex, race, BMI, smoking status, ABO incompatibility, and relationship with recipient. These factors were selected because they were associated with early incident post-donation hypertension and one or both of the graft failure outcomes described below.

To avoid bias due to extreme values, we used stabilized weights.[15] To see how well the population was balanced by weighting, we calculated standardized differences between LDKT recipients whose donors developed early post-donation incident hypertension and those whose donors did not, both before and after weighting. Standardized differences of <0.1 after weighting demonstrate balance between groups.[14]

Recipient graft failure

To examine how LDKT recipient all-cause graft failure (ACGF) was associated with early post-donation incident hypertension, we estimated incidence of graft failure at 1, 5, and 10 years after transplantation using Kaplan-Meier methods with log-rank tests. We then used Cox proportional hazards regression models to quantify the adjusted hazard ratio (aHR) of early post-donation incident hypertension in the weighted population. We also created

standardized survival curves adjusted for donor and recipient characteristics, by using the weighted population. As a sensitivity analysis, we repeated this for recipient death-censored graft failure (DCGF).

Subgroup analyses

To determine whether ACGF differed for recipients with hypertension-caused or diabetes-caused ESRD and for African American recipients, we examined these subgroups separately, and repeated the analysis using inverse probability of treatment weighting and Cox proportional hazards regression as described above.

Sensitivity analysis

We examined the association between ACGF and the median systolic blood pressure reported for each donor across all follow-up reports as a sensitivity analysis, scaled per 10 mmHg. We used Cox proportional hazards regression adjusting for recipient age as a linear spline with a knot at 40 years, sex, race, BMI, cause of ESRD, education, and peak panel-reactive antibody (PRA); and donor age, sex, race, BMI, smoking status, ABO incompatibility, human leukocyte antigen (HLA) mismatches, and relationship with recipient. Because there were 5,211 donors (13.7%) who were missing systolic blood pressure at all follow-up reports, we used multiple imputation by chained equations to impute missing values using predictors of recipient age, sex, race, cause of ESRD, BMI, education, insurance, peak PRA, and ACGF; and donor age, sex, race, BMI, smoking status, ABO incompatibility, HLA mismatches, relationship with recipient, preoperative eGFR, preoperative systolic blood pressure, preoperative diastolic blood pressure, postoperative systolic blood pressure, postoperative diastolic blood pressure, and baseline hazard of early hypertension requiring antihypertensive medication.

Statistical analysis

Confidence intervals are reported as per the method of Louis and Zeger.[16] An α of 0.05 was considered statistically significant. All analyses were performed using Stata 14.2/MP for Linux (College Station, Texas).

RESULTS

Study population

Of 37,901 living kidney donors, 923 (2.4%) developed early post-donation incident hypertension. Donors who developed early post-donation incident hypertension were older (median 49 vs. 42 years, $p<0.001$), more frequently male (47.7% vs. 37.3%, $p<0.001$), less frequently Hispanic (9.2% vs. 14.2%, $p<0.001$), and more frequently overweight (46.2% vs. 42.0%) or obese (30.0% vs. 22.3%, $p<0.001$) compared to donors who did not develop hypertension. Donors who developed hypertension were more frequently a first-degree relative (44.2% vs. 42.4%) or spouse or partner (18.4% vs. 13.3%, $p<0.001$) to their recipient compared to donors who did not develop hypertension (Table 1).

Of donors who developed post-donation hypertension, the median systolic blood pressure reported across all follow-up reports was 131 mmHg (interquartile ratio [IQR] 120.5–141,

with 11.3% missing) and the median diastolic blood pressure reported across all follow-up reports was 81 mmHg (IQR 75–88.5, with 11.4% missing) (Figure 2). Of donors who did not develop post-donation hypertension, the median systolic blood pressure reported across all follow-up reports was 119 mmHg (IQR 111.5–126, with 14.0% missing) and the median diastolic blood pressure reported across all follow-up reports was 74 mmHg (IQR 69.5–80, with 14.0% missing) (Figure 2).

LDKT recipients whose donors developed hypertension were older (median 53 vs. 50 years, p<0.001), less frequently Hispanic (10.6% vs. 14.7%, p=0.002), and had a lower eGFR at discharge after transplantation (median 53 vs. 56 mL/min, p<0.001) (Table 1). After applying inverse probability of treatment weights to the study population, recipient and donor characteristics between those who developed post-donation hypertension and those who did not were balanced as demonstrated by all standardized differences <0.1 (Table 2). In other words, estimates of the association between post-donation incident hypertension and recipient outcomes are unbiased by baseline characteristics because balance in baseline characteristics has been achieved between groups through weighting.[14]

All-cause graft failure

At 1, 5, and 10 years after transplantation, 2.9%, 17.0%, and 38.6% of LDKT recipients whose donors developed early post-donation incident hypertension had ACGF compared to 2.8%, 12.8%, and 30.5% of those whose donors did not develop hypertension (p=0.02). LDKT recipients whose donors developed early post-donation incident hypertension were at 19% higher risk for ACGF (HR $_{1.03}1.19_{1.38}$, p=0.02). However, after weighting to control for donor and recipient characteristics, LDKT recipients whose donors developed early post-donation incident hypertension were not at higher risk for ACGF (aHR $_{0.85}1.03_{1.25}$, p=0.72) (Figure 3).

Death-censored graft failure

Our sensitivity analysis examining DCGF confirmed inferences. At 1, 5, and 10 years after transplantation, 1.8%, 8.7%, and 25.4% of LDKT recipients whose donors developed early post-donation incident hypertension had DCGF compared to 1.7%, 7.2%, and 15.5% of those whose donors did not develop hypertension (p=0.14). LDKT recipients whose donors developed early post-donation incident hypertension were not at higher risk for DCGF (HR $_{0.94}1.16_{1.44}$, p=0.1). After weighting, LDKT recipients whose donors developed early post-donation incident hypertension were not at higher risk for DCGF (aHR $_{0.81}1.05_{1.37}$, p=0.66) (Figure 4).

Subgroup analyses

The association between early post-donation incident hypertension and recipient ACGF did not vary among LDKT recipients with ESRD from hypertension (n=6251), those with ESRD from diabetes (n=8563), or among African American recipients (n=4967). After weighting to control for donor and recipient characteristics, there was no statistically significant association between donor incident hypertension and recipient ACGF in any subgroup analysis (recipients with hypertension-caused ESRD: aHR $_{0.65}1.10_{1.85}$, p=0.73; recipients

with diabetes-caused ESRD: aHR $_{0.56}0.80_{1.13}$, p=0.20; African American recipients: aHR $_{0.70}1.10_{1.73}$, p=0.68).

Sensitivity analysis

Higher post-donation systolic blood pressure was associated with higher risk of ACGF. For each increment of 10 mmHg of post-donation systolic blood pressure, there was a 5% higher risk of ACGF (aHR $_{1.03}1.05_{1.08}$, p<0.001).

DISCUSSION

In this national study of 37,901 living donor kidney transplant recipients, we used propensity score weighting to isolate the association between early post-donation incident hypertension in donors and the graft outcomes of their recipients. In adjusted models, we found no association between early post-donation incident hypertension and recipient all-cause graft failure (aHR 1.03, p=0.72) or death-censored graft failure (aHR 1.05, p=0.66). We then studied subgroups of recipients who might be at highest risk for graft failure—African American recipients and those with ESRD caused by hypertension and diabetes—and also found no higher risk of graft failure associated with early post-donation incident hypertension in these subgroups.

Our finding of no association between early incident post-donation hypertension and recipient graft failure is surprising, given the known link between donor hypertension and ESRD[5,6] and the association of recipient graft failure with donor ESRD.[1] However, we did find an association between donor post-donation systolic blood pressure and graft failure. It may be that kidneys from donors whose post-donation hypertension was more easily controlled by antihypertensive medication were not at higher risk for graft failure, while those from donors whose hypertension was more difficult to control did carry a higher risk of graft failure. It also might be that the reporting of diagnosed hypertension in the registry is less reliable than the reporting of measured blood pressure. Regardless, our findings suggest that there are differences in renal physiology between donors and the recipients of their kidneys. Prior literature does suggest differences in renal physiology in donors versus those with kidney disease. Although Lenihan et al. found that donor hyperfiltration was a benign adaptive process not associated with albuminuria in their study of 21 living kidney donors at median 6 years post-donation,[17,18] Steiner cites evidence that hyperfiltration causes mechanical injury and progressive loss of GFR in rat studies and suggests that long-term outcomes deserve further consideration.[19] Our results contrast with the study from Miller et al. of 115,124 kidney transplant recipients which found that recipients were at higher risk of graft failure due to “nephron underdosing” when their donors were ≥10 kg lighter than they were.[20] While our epidemiologic study found no association between the diagnosis of post-donation incident hypertension and recipient graft loss, we found that the reported post-donation systolic blood pressure may be more indicative of a linked physiology between the donor’s remaining kidney and transplanted kidney. Further work should continue to clarify the physiologic impact of subclinical kidney disease and glomerular hyperfiltration on both kidney donors and transplant recipients.

Our study has several limitations inherent to its use of national registry data. We are limited by missing data and by the clinical granularity of the available data. We do not have information regarding donor albuminuria, detailed family history, or presence of apolipoprotein L1 high risk variants,[21,22] all of which impact donor risk for later ESRD.[23] Although we use a propensity score method for balancing baseline characteristics between recipients whose donors developed early post-donation incident hypertension and those whose donors did not, we cannot account for potential unmeasured confounders that differ between groups.

In conclusion, we found that early post-donation hypertension did not portend a higher risk of recipient graft failure in the same way that eventual post-donation ESRD does. These findings held when limiting to recipient subsets at highest risk for progressive kidney disease due to graft hyperfiltration: African American recipients and those with ESRD caused by hypertension and diabetes. While these findings do not improve risk prediction beyond current models, they suggest some insights into the physiology of LDKT grafts in the milieu of the recipient with ESRD.

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Abbreviations:

ACGF	all-cause graft failure
aHR	adjusted hazard ratio
ATT	average treatment effect on the treated
BMI	body mass index
DCGF	death-censored graft failure
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
HRSA	Health Resources and Services Administration
HTN	hypertension
LDKT	living donor kidney transplantation

OPTN	Organ Procurement and Transplantation Network
SRTR	Scientific Registry of Transplant Recipients

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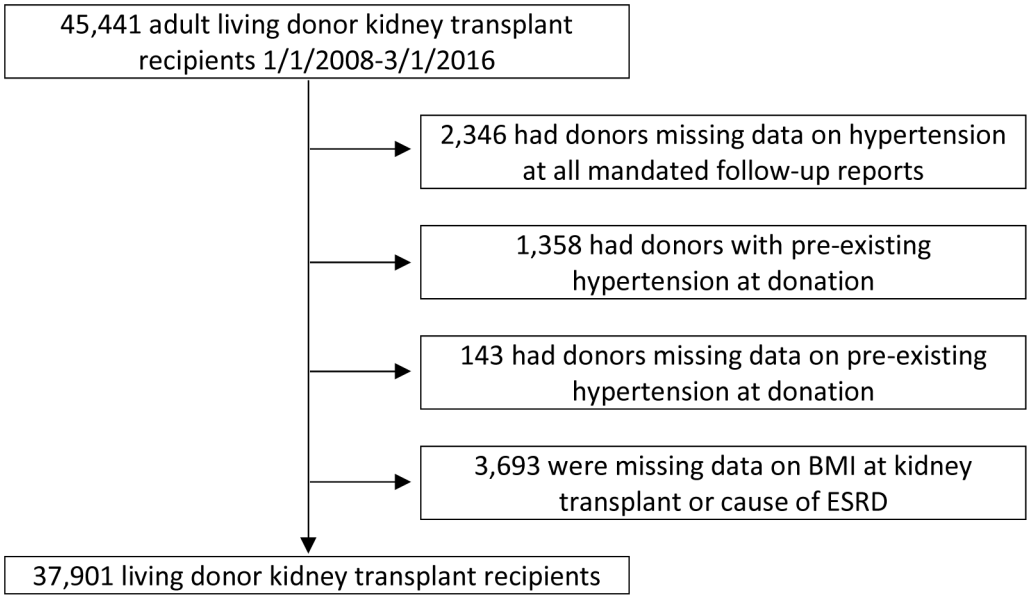

Figure 1. Study population with exclusion criteria.

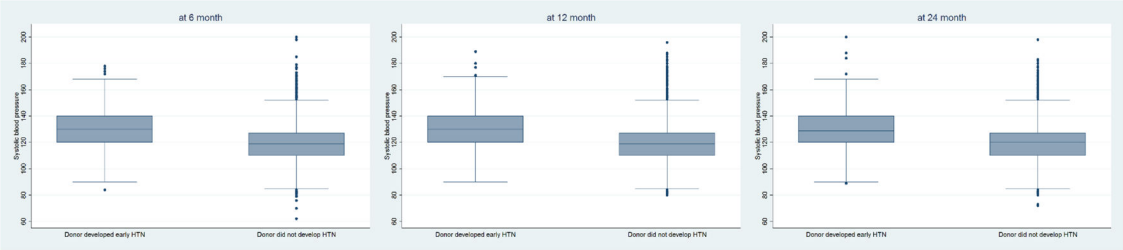

Figure 2. Donor systolic blood pressure reported at 6 month, 12 month, and 24 month follow-up after living kidney donation.

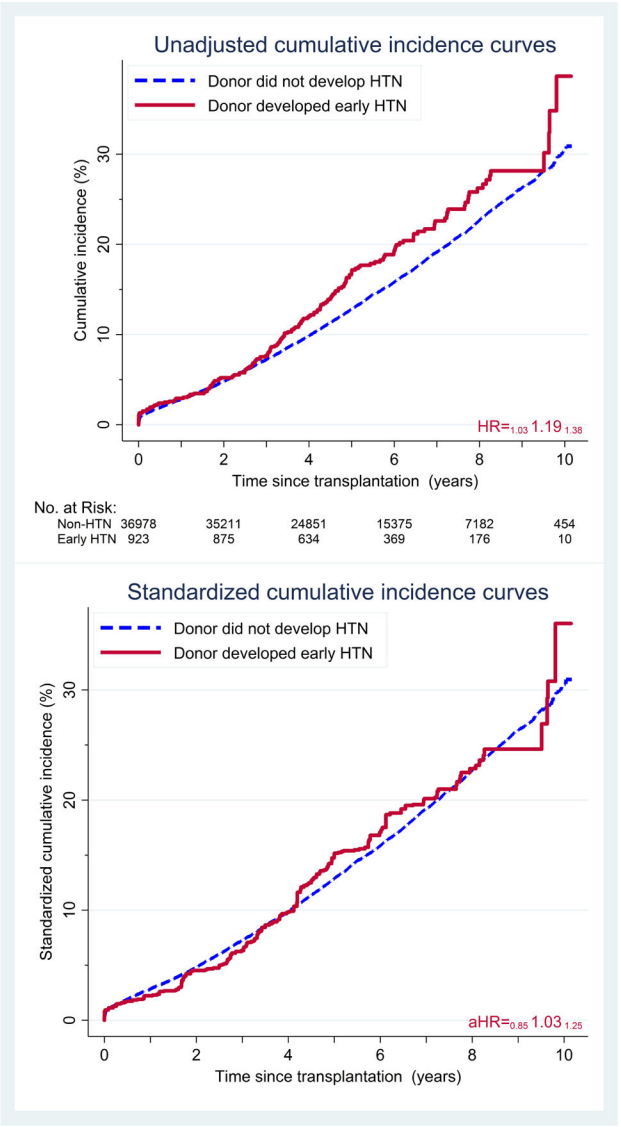

Figure 3. Unadjusted and standardized all-cause graft failure in LDKT recipients from donors who developed early post-donation incident hypertension (HTN) as compared to those whose donors did not develop early HTN.

Standardized curves are weighted by the propensity score method of inverse probability of the donor having developed hypertension, using both donor and recipient characteristics.

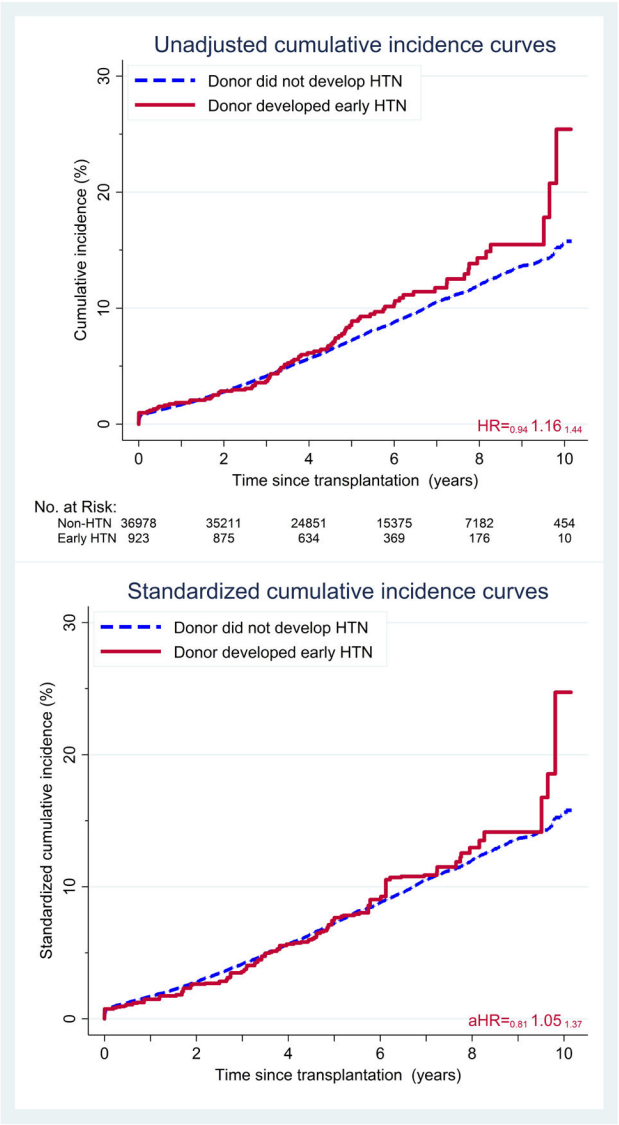

Figure 4. Unadjusted and standardized death-censored graft failure in LDKT recipients from donors who developed early post-donation incident hypertension (HTN) as compared to those whose donors did not develop early HTN.

Standardized curves are weighted by the propensity score method of inverse probability of the donor having developed hypertension, using both donor and recipient characteristics.

Table 1.

Characteristics of LDKT recipients and their donors by donor early incident post-donation hypertension (HTN).

	Donor did not develop HTN N=36,978	Donor developed early HTN N=923	p-value
Recipient characteristics			
Age at transplant, median years (IQR)	50 (38, 59)	53 (40, 62)	<0.001
Male sex	61.9%	64.8%	0.08
Race			0.002
Caucasian/other	72.0%	75.8%	
African American	13.3%	13.5%	
Hispanic	14.7%	10.6%	
BMI at transplant, median (IQR)	27 (24, 31)	28 (24, 32)	0.03
BMI category			0.05
Normal/underweight	34.7%	30.9%	
Overweight	33.2%	35.0%	
Obese	32.1%	34.1%	
Cause of ESRD			0.18
Glomerulonephritis	31.0%	27.8%	
Diabetes	22.8%	24.5%	
Hypertension	16.7%	16.5%	
Other	29.5%	31.2%	
Peak PRA			0.85
0-9%	77.5%	77.4%	
10-79%	17.5%	17.8%	
80-98%	3.8%	3.5%	
99-100%	1.2%	1.4%	
College or higher education	61.6%	64.9%	0.04
Insurance			0.14
Public/Other	41.9%	44.3%	
Private	58.1%	55.7%	
Time on dialysis, median years (IQR)	0.6 (0, 1.8)	0.5 (0, 1.6)	0.11
eGFR at discharge after transplant, median mL/min/1.73m^2 (IQR)	56 (40, 74)	53 (35, 70)	<0.001
Transplant characteristics			
Zero HLA mismatches	7.3%	7.3%	0.92
ABO incompatible	1.6%	1.2%	0.34
Donor characteristics			
Age at donation, median years (IQR)	42 (33, 51)	49 (41, 56)	<0.001
Male sex	37.3%	47.7%	<0.001
Race			<0.001
Caucasian/other	74.5%	79.2%	
African American	11.2%	11.6%	

	Donor did not develop HTN N=36,978	Donor developed early HTN N=923	p-value
Hispanic	14.2%	9.2%	
Relationship with recipient			<0.001
Unrelated	36.3%	31.7%	
First degree	42.4%	44.2%	
Spouse/partner	13.3%	18.4%	
Other	8.1%	5.6%	
BMI at donation, median (IQR)	27 (24, 30)	28 (25, 31)	<0.001
BMI category			<0.001
Normal/underweight	35.7%	23.8%	
Overweight	42.0%	46.2%	
Obese	22.3%	30.0%	
History of cigarette smoking	25.5%	26.8%	0.39

Table 2.

Standardized differences between groups before and after weighting by inverse probability of treatment.

Estimates of the association between post-donation incident hypertension and recipient outcomes are unbiased by baseline characteristics, shown by balance in all baseline characteristics after weighting.[14]

	Before weighting	After weighting
Recipient characteristics		
Age	0.141	0.033
Male sex	0.060	−0.018
Race		
Caucasian/other	0.088	−0.018
African American	0.006	−0.001
Hispanic	−0.123	0.023
BMI category		
Normal/underweight	−0.082	0.030
Overweight	0.039	−0.031
Obese	0.043	0.001
Cause of ESRD		
Glomerulonephritis	−0.069	0.018
Diabetes	0.040	−0.007
Hypertension	−0.007	0.006
Other	0.037	−0.016
Peak PRA		
0-9%	−0.004	−0.046
10-79%	0.006	0.023
80-98%	−0.018	0.047
99-100%	0.023	0.010
College or higher education	0.068	−0.019
Insurance		
Public/Other	0.049	0.048
Private	−0.049	−0.048
eGFR at discharge after transplant		
ABO incompatible	−0.034	0.011
Donor characteristics		
Age at donation	0.536	0.026
Male sex	0.211	−0.013
Race		
Caucasian/other	0.111	−0.032
African American	0.012	0.018
Hispanic	−0.157	0.023
Relationship with recipient		
Unrelated	−0.096	−0.044

	Before weighting	After weighting
First degree	0.037	0.000
Spouse/partner	0.142	0.008
Other	−0.097	0.064
BMI category		
Normal/underweight	−0.261	−0.011
Overweight	0.084	0.002
Obese	0.176	0.010
History of cigarette smoking	0.028	−0.021