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Mortality in Living Kidney Donors With ESRD: A Propensity Score Analysis Using the United States Renal Data System

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Introduction: In recent years, data have emerged on the outcomes of living kidney donors who develop end-stage renal disease (ESRD). We aimed to evaluate mortality rates in kidney donors who had initiated dialysis compared with a propensity-matched cohort of dialysis patients without previous kidney donation.

Methods: We used the United States Renal Data System (USRDS) and abstracted 274 previous living kidney donors between 1995 and 2009. There were 609,398 individuals on dialysis without kidney donation. We used propensity score matching to identify 258 donors and 258 nondonors. The time-dependent Cox proportional hazards model was used to compare survival between the 2 matched cohorts.

Results: In the propensity score–matched cohort, mortality was lower in donors compared with nondonors (19% vs. 49%; $P < 0.0001$). The time-dependent Cox proportional hazards model demonstrated that donors had significantly lower mortality compared with nondonors 0 to 5 years since start of dialysis (hazard ratio [HR]: 0.17; 95% confidence interval [CI] 0.11–0.27; $P < 0.0001$) and with nondonors 5 to 10 years on dialysis (HR: 0.34; 95% CI: 0.19–0.63; $P < 0.001$). We were unable to estimate the difference between the 2 groups after 10 years on dialysis with any precision (HR: 0.51; 95% CI: 0.18–1.42; $P = 0.20$) due to the small sample size.

Conclusion: We observed a lower mortality rate in living kidney donors with ESRD compared with matched nondonors. This data should guide clinicians in the informed consent process with prospective donors.

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Candidates for living kidney donation in the United States are rigorously screened and have to meet strict medical and psychosocial eligibility criteria before organ donation. Partly because of the current screening evaluation, living kidney donation is generally considered safe. Survival among screened kidney donors appears to be similar or better than those in the general population.[1,2] Furthermore, Segev *et al.*[2] reported that kidney donors were not at higher risk for mortality compared with healthy matched nondonors, after a median of 6.3 years. Nonetheless, kidney donors have an increased relative risk of developing end-stage renal disease (ESRD) compared with healthy matched nondonors, although the magnitude of this risk remains small.[3,4]

Previous living donors who unfortunately progress to ESRD are listed as active status and receive priority on the transplantation waiting list in a timely manner.[5] However, one-half of previous living donors who did not receive preemptive transplantation were on dialysis for ≥332 days before being placed on the list. Potluri *et al.*[6] assessed kidney transplantation outcomes for previous living donors and found that they received higher quality allografts and experienced lower posttransplantation mortality than matched nondonors (HR: 0.19; $P < 0.001$). In an analysis of 99 donors with ESRD who were individually matched to 5 nondonors with ESRD based on demographic and clinical characteristics, Muzaale *et al.*[7] found that donors had lower mortality than matched nondonors (HR 0.7; $P < 0.05$).

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For the present study, we used a larger sample size based on data from the USRDS to further evaluate mortality among kidney donors who had initiated dialysis compared with a propensity-matched cohort of dialysis patients without previous kidney donation. We used the propensity score method over traditional multivariable regression to provide less biased estimates in a small cohort with fewer outcome events per adjustment covariate.[8,9]

MATERIALS AND METHODS

We used the USRDS to identify living kidney donors who progressed to ESRD from 1995 to 2009, based on the *International Classification of Diseases, Ninth Revision, Clinical Modification* diagnosis code V59.4. The study cohort was restricted to patients with Medicare as the primary payer upon dialysis initiation. This restriction was necessary to ensure accurate ascertainment of Medicare claims, which might not be reported in patients covered primarily by an insurer other than Medicare. We compared dialysis patients who were previous kidney donors with those who had no history of kidney donation. We excluded patients who were younger than 18 years of age and those on peritoneal dialysis.

We used a propensity score–matched cohort of donors and nondonors to compare all-cause mortality between the 2 groups. We favored a propensity score matching approach more than a regression model adjusting for covariates because of its advantages in bias reduction when large differences in observed covariates exist between groups, without modeling the association between the outcome and the confounders.[8]

Propensity scores were calculated from a logistic regression model, using the following variables: age, sex, race, Hispanic ethnicity, primary cause of ESRD, estimated glomerular filtration rate (eGFR), time when dialysis was initiated since 1995 (start of inception cohort), and comorbid conditions, including hypertension, peripheral vascular disease, diabetes mellitus, chronic obstructive pulmonary disease (COPD), and ischemic heart disease. As indicated by Rosenbaum *et al.*, matching on propensity scores can achieve a balance on the covariates used for creating the scores.[10] Unlike randomization, it does not achieve balance on the covariates not used in the propensity matching, except for the extent that they are correlated with the ones used in the matching. There were several other variables related to mortality that we intended to use, but these were limited by a large number of missing observations. Two such variables were serum albumin and body mass index (BMI).

Donors were matched to nondonors with a 1:1 matching in propensity scores without replacement. As suggested by Rosenbaum *et al.*[10] matching was performed after a transformation of the estimated propensity scores (with the function log [(1 − x)/x]), which resulted in an approximately normal distribution of the transformed scores. The matching was performed with the nearest neighbor method without replacement using a SAS (SAS Institute, Cary, NC) macro developed by Coca-Perraillon.[11]

Survival after dialysis initiation for both donors and nondonors was investigated in the propensity score–matched cohort. The survival curve was estimated using the Kaplan-Meier method, and the log-rank test was used to compare survival between the 2 groups. Proportional hazards assumptions were examined by graphing log (−log [survival function]) versus log (time) for the 2 groups. There were some indications that the assumptions of proportional hazards were not satisfied. Thus, we used a time-dependent Cox proportional hazards model to compare the survival between donors and nondonors. To obtain HRs for each of the 3 time intervals after dialysis initiation (0–5, 5–10, and >10 years), we used time-dependent indicator variables for being a donor, 1 for each time interval. To account for the matching, we used a Cox model with a random effect for the matched pairs (shared frailty model, using a gamma distribution). Baseline variables were compared between donors and nondonors using 2-sample *t* and chi-square tests, as appropriate. As previously described,[8,12] 2-sample *t*-statistic and the standardized percentage difference were used to determine variables with noticeable differences between the groups initially, and to asses if balance was achieved after the matching. The Fine and Gray[13] model was used to compare the cumulative incidence of transplantation in the donors and nondonors groups. This model allowed us to calculate a subdistribution HR for the difference between the groups, considering death as a competing risk of transplantation. Analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC). Statistical significance was defined as a $P < 0.05$.

RESULTS

We identified 274 donors and 609,398 nondonors in our study cohort. Table 1 shows the baseline characteristics of the 2 groups. Nondonors were significantly older at start of dialysis compared with donors (70.5 years vs. 43.9 years; $P < 0.0001$) and were more likely to be female (47.3% vs. 31.8%; $P < 0.0001$). There were significant differences between the 2 groups in terms of the cause of ESRD ($P < 0.0001$). Patients in the nondonor group were more likely to have diabetes mellitus as the primary cause of ESRD compared with donors (45.7% vs. 25.9%). Hypertension as the primary cause

Table 1. Baseline characteristics of end-stage renal disease patients who were previous kidney donors versus nondonors

Variable	Controls (n = 609,398)	Donors (n = 274)	P value[a]
Age at dialysis initiation (yr)	70.5 ± 11.7	43.9 ± 15.1	<0.0001
eGFR at dialysis initiation	10.1 ± 4.7	7.1 ± 3.5	<0.0001
Time at which dialysis was initiated (yr) since 1995	7.4 ± 4.1	5.6 ± 4	<0.0001
Female sex	47.3	31.75	<0.0001
Black race	25.3	20.3	0.0563
Hispanic ethnicity	33.2	33.9	0.928
Comorbid conditions			
Diabetes mellitus	12.19	6.93	0.0079
Hypertension	77.95	72.6	0.0335
Chronic obstructive pulmonary disease	10.77	1.09	<0.0001
Ischemic heart disease	13.95	1.82	<0.0001
Peripheral vascular disease	18.38	4.38	<0.0001
Cause of ESRD			<0.0001
Diabetes mellitus	45.7	25.9	
Hypertension	31.3	21.9	
Glomerulonephritis	5.79	25.5	
Cystic kidney disease	1.15	4.01	
Other urologic	2.58	2.92	
Other cause	9.35	16.06	
Unknown cause	4.07	3.65	

eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease.
Data are mean ± SD or %.
[a]P values are from the 2-sample t-test or χ^2 test, as appropriate.

of ESRD was also more common in nondonors versus donors (31.3% vs. 21.9%). In contrast, ESRD was more likely to be caused by glomerulonephritis in donors compared with nondonors (25.5% vs. 5.8%). Ischemic heart disease, peripheral vascular disease, and COPD were more prevalent in nondonors compared with donors.

Table 2 presents the comparisons between the 2 groups before the propensity score matching. Results of the standardized percentage difference and the 2-sample t-statistic demonstrated significant differences in clinical characteristics between the 2 groups. In addition to the differences mentioned previously, eGFR at start of dialysis and time of dialysis initiation since 1995 were 2 covariates with large initial differences.

Table 3 presents the comparisons between the 2 groups after matching. The groups were balanced, with the standardized percentage difference not exceeding 10% in absolute value, which is often used as a benchmark.[5] Further, P values based on 2-sample t-tests showed nonsignificant differences in the means of the 2 groups. We inspected the histograms for the distribution of propensity scores for both groups after matching (Figure 1); we were satisfied of the overlap in their distributions, which is an important assumption to ensure valid causal inference.[19]

Survival after dialysis initiation for donors and nondonors was investigated in the propensity score–matched cohort. There were 49 deaths in the 258 donors, and there were 127 deaths in the 258 control subjects. As shown in Figure 2, the donor group had significantly better survival than the

Table 2. Comparisons between donors and nondonors before propensity score matching

Variable	Controls (n = 609,398) Mean ± SD	Donors (n = 274) Mean ± SD	Comparisons: Two-sample t-statistic	Comparisons: Standardized difference (%)[a]
Age at dialysis initiation (yr)	70.55 ± 11.69	43.89 ± 15.12	29.18[b]	−197.257
eGFR at dialysis initiation	10.13 ± 4.74	7.07 ± 3.49	14.19[b]	−73.471
Time at which dialysis was initiated (yr) since 1995	7.38 ± 4.09	5.59 ± 4.03	7.34[b]	−44.01
Female sex	0.47 ± 0.5	0.32 ± 0.47	5.52[b]	−32.914
Black race	0.25 ± 0.43	0.2 ± 0.4	2.0[c]	−12.029
Hispanic ethnicity	0.33 ± 0.47	0.33 ± 0.47	−0.09	0.545
Comorbid conditions				
Diabetes mellitus	0.12 ± 0.33	0.07 ± 0.25	3.41[b]	−17.919
Hypertension	0.78 ± 0.41	0.73 ± 0.45	1.97[c]	−12.358
Chronic obstructive pulmonary disease	0.11 ± 0.31	0.01 ± 0.1	15.33[b]	−41.827
Ischemic heart disease	0.14 ± 0.35	0.02 ± 0.13	14.9[b]	−46.06
Peripheral vascular disease	0.18 ± 0.39	0.04 ± 0.21	11.29[b]	−45.169
Cause of ESRD				
Diabetes milletus	0.46 ± 0.5	0.26 ± 0.44	7.48[b]	−42.271
Hypertension	0.31 ± 0.46	0.22 ± 0.41	3.76[b]	−21.385
Glomerulonephritis	0.06 ± 0.23	0.26 ± 0.44	−7.48[b]	56.39
Cystic kidney disease	0.01 ± 0.11	0.04 ± 0.2	−2.41[c]	18.086
Other urologic	0.03 ± 0.16	0.03 ± 0.17	−0.33	2.069
Other cause	0.09 ± 0.29	0.16 ± 0.37	−3.02[c]	20.234
Unknown cause	0.04 ± 0.2	0.04 ± 0.19	0.37	−2.155

eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease.
[a]Standardized difference in percent is the mean difference as a percentage of the average SD $100(\bar{x}_d - \bar{x}_c)/\sqrt{\{(s_d^2 + s_c^2)/2\}}$, where $\bar{x}_d$, $\bar{x}_c$ are the sample means in the donor and control groups and the s_d^2, s_c^2 are the corresponding variances.
[b]$P < 0.01$.
[c]$0.05 > P > 0.01$.

Table 3. Comparisons between donors and nondonors after propensity score matching

Variable	Nondonor (n = 258) Mean ± SD	Donors (n = 258) Mean ± SD	Comparisons: Two sample t-statistic[a]	Comparisons: Standardized difference (%)
Age at dialysis initiation (yr)	44.81 ± 16.61	44.24 ± 15.08	0.41	−3.591
eGFR at dialysis initiation	6.73 ± 2.89	7.02 ± 3.43	−1.07	9.413
Time at which dialysis was initiated (yr) since 1995	6.02 ± 5.51	5.75 ± 3.98	0.63	−5.566
Female sex	0.33 ± 0.47	0.32 ± 0.47	0.28	−2.473
Black race	0.21 ± 0.41	0.19 ± 0.39	0.55	−4.841
Hispanic ethnicity	0.35 ± 0.48	0.34 ± 0.48	0.09	−0.813
Comorbid conditions				
Diabetes mellitus	0.08 ± 0.27	0.07 ± 0.26	0.17	−1.464
Hypertension	0.77 ± 0.42	0.74 ± 0.44	0.71	−6.29
Chronic obstructive pulmonary disease	0 ± 0.06	0.01 ± 0.11	−1	8.83
Ischemic heart disease	0.01 ± 0.11	0.02 ± 0.14	−0.71	6.265
Peripheral vascular disease	0.05 ± 0.22	0.05 ± 0.21	0.2	−1.802
Cause of ESRD				
Diabetes mellitus	0.26 ± 0.44	0.27 ± 0.44	−0.2	1.756
Hypertension	0.22 ± 0.41	0.21 ± 0.41	0.11	−0.941
Glomerulonephritis	0.28 ± 0.45	0.25 ± 0.43	0.79	−6.996
Cystic kidney disease	0.03 ± 0.18	0.04 ± 0.19	−0.23	2.054
Other urologic	0.02 ± 0.14	0.03 ± 0.17	−0.84	7.411
Other cause	0.14 ± 0.35	0.16 ± 0.37	−0.61	5.374
Unknown cause	0.04 ± 0.2	0.03 ± 0.18	0.46	−4.009

eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease.
[a]P values for all t-tests >0.05.

matched nondonor group ($P < 0.0001$). We noted that only nondonors who were matched to donors were used in creating the survival curve. Therefore, the resulting survival curve did not represent the mortality in the nondonor general population. Survival between the donors and nondonors was based on a time-dependent Cox proportional hazards model (with shared frailty to account for the matching). Because the 2 groups were balanced on the variables used for creating the propensity scores, not adjusting for these variables in the Cox model would not lead to bias. Donors had significantly lower mortality 0 to 5 years (HR: 0.17; 95% CI: 0.11−0.27; $P < 0.0001$) and 5 to 10 years after start of dialysis (HR: 0.34: 95% CI: 0.19−0.63; $P < 0.001$). We were unable to estimate the difference between the 2 groups after 10 years on dialysis with any precision (HR: 0.51; 95% CI: 0.18−1.42; $P = 0.20$) due to the small sample size (i.e., 71 donors and 48 nondonors at 10 years; 3 donors and 8 nondonors at 20 years).

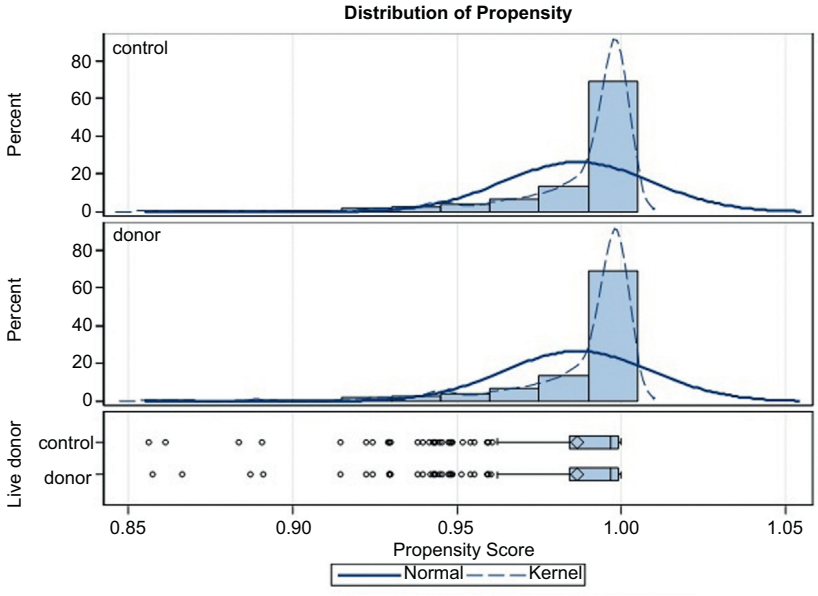

Figure 1. Propensity scores after matching.

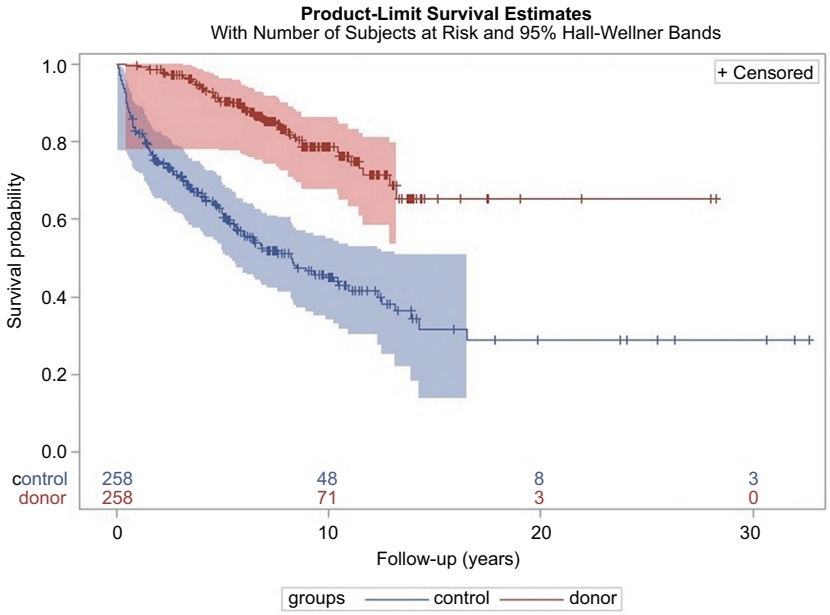

Figure 2. Product limit survival estimates with number of subjects at risk and 95% confidence bands.

As a sensitivity analysis, we repeated the analysis, limiting it to patients who were on dialysis for at least 1 year because of the high death rate in the nondonor group early after dialysis initiation. The estimated HRs were similar, with significantly lower mortality for donors at 1 to 5 years (HR: 0.27; 95% CI: 0.16−0.46; $P < 0.0001$) and 5 to 10 years (HR: 0.35; 95% CI: 0.19−0.64; $P < 0.001$). The difference between the 2 groups after 10 years remained nonsignificant (HR: 0.50; 95% CI: 0.18−1.42; $P = 0.19$). In another sensitivity analysis, we added serum albumin and BMI in the Cox model. Although the study sample size was reduced from 516 (258 per group × 2) to 389 due to missing values, the estimated HRs for the survival of donors versus nondonors were very similar (HR: 0.18; 95% CI: 0.10−0.31; $P < 0.0001$; HR: 0.46; 95% CI: 0.23−0.97; $P = 0.04$; and HR: 0.79; 95% CI: 0.27−2.36; $P = 0.68$ for the time periods of <5, 5−10, and >10 years since the start of dialysis, respectively).

There was a difference in the rates in which patients underwent transplantation, with 252 (98%) donors and only 72 (28%) nondonors who underwent the transplant procedure. The median (25th−75th percentile) times from start of ESRD to transplantation (in years) were 1.3 (0.8−2.3) and 1.7 (0.7−2.7) for the donor and nondonor groups, respectively. We used the competing risk model of Fine and Gray[13] to compare the cumulative incidence of transplantation in the donor and nondonor groups. The rate of transplantation was significantly higher in donors compared with nondonors (subdistribution HR: 8.3; 95% CI: 6.3−10.9; $P < 0.0001$). Most of the transplantations were performed in the first 3 years, with 213 in donors and 57 in nondonors; both curves did not change after this time.

DISCUSSION

Although the risk of ESRD in kidney donors has been assessed in multiple studies,[4,7,14,15] there have only been a few recent studies that have examined the outcomes of living kidney donors who develop ESRD requiring renal replacement therapy.[5–7] We found in a national multiyear study of the US incident hemodialysis population that patients who were previous kidney donors had a significantly lower risk of death compared with a propensity-matched cohort that did not have a history of kidney donation. The groups were matched on demographic factors, primary cause of ESRD, eGFR at dialysis initiation, comorbid conditions, and the time at which dialysis was initiated during the 14-year study period, represented as the years elapsed since 1995. The latter variable was included in the matching algorithm to account for any potential period effects that might have arisen from new therapeutic interventions.

Our study was unique in that we conducted propensity score analyses because of an imbalance of baseline characteristics in the donor and nondonor groups that could lead to biased results caused by confounding by indication. For instance, the nondonor patients were significantly older at dialysis initiation versus their donor counterparts (mean 70.5 years vs. 43.9 years, respectively). Using the Organ Procurement and Transplantation Network (OPTN) and the Center for Medicare and Medicaid Services

databases, Cherikh *et al.*[16] analyzed 56,458 living kidney donors who donated from 1987 to 2003. The median age at donation was 35.7 years for kidney donors who developed ESRD. Forty-four percent of donors with ESRD developed postdonation renal failure between 35 and 49 years old, with an overall median age of 46.8 years for developing ESRD. In another OPTN analysis of all living kidney donors from 1994 to 2011, Muzaale *et al.*[3] found that ESRD developed after a mean of 8.6 $\pm$ 3.6 years after kidney donation. The findings of these studies are consistent with the age of ESRD onset of kidney donors in our study cohort. In addition to being younger, kidney donors in our study cohort were less likely to have diabetes or hypertension as the cause of ESRD and were less likely to have comorbid conditions, such as peripheral vascular disease, COPD, and ischemic heart disease. In contrast, kidney donor patients were more likely to be male and were more likely to have cystic kidney disease and glomerulonephritis as the causes of ESRD.

Based on graphic assessment, there were indications of nonproportionality of hazards between the 2 groups. We therefore conducted time-segmented Cox regression analyses using follow-up time intervals of 0 to 5 years, 5 to 10 years, and >10 years after initiation of dialysis. The survival advantage among the kidney donor group attenuated over time and became nonsignificant after 10 years. However, caution is warranted in the interpretation of the 10+ year data because of the small sample size of patients at risk.

There are several potential explanations for the survival advantage among ESRD patients who were previous kidney donors. Although we matched donors to their nondonor counterparts on key demographic and clinical characteristics, there were likely residual confounders that could account for the lower mortality in the donor population. Candidates for kidney donation undergo comprehensive medical and psychosocial evaluation, and are thus inherently healthier than nondonor patients. Donors may have greater motivation to maintain healthy habits and adhere to treatment regimen. They may also have better social support and resources within their families and communities. Furthermore, they may be in a better socioeconomic status and have greater access to health care. In a retrospective cohort study that used Scientific Registry of Transplant Recipients data, a higher percentage of previous kidney donors had a college education (46% vs. 39%) and private insurance (56% vs. 44%) compared with all listed candidates for kidney transplantation.[7] These differences were likely greater when comparing ESRD patients who were previous kidney donors with all other nondonor ESRD patients, which included those who had never undergone a transplantation evaluation. Importantly, we found that most of the donors (98%) were subsequently transplanted compared with only 28% of nondonors. Donors were also transplanted earlier than their nondonor counterparts. Previous organ donors have been given priority by the OPTN since 1996 in the allocation of deceased donor kidneys, and their access to rapid transplantation has been maintained under the new US kidney allocation system.[17–19] These differential transplantation rates are likely one of the key drivers of the survival advantage among ESRD patients who were previous kidney donors compared with nondonors.

Our study had several limitations. We used the USRDS database, which has advantages given its size, and almost complete inclusion of the ESRD population in the United States. However, inherent limitations, such as completeness of data in the Medical Evidence Report at initiation of renal replacement therapy, have been well described.[20] In our study, the cause of ESRD in donors was more likely to be glomerulonephritis compared with nondonors, which suggests that it had a relatively early onset after donation. These results differed from those of Matas *et al.*[21] We acknowledge that our results could not be generalized for donors who developed ESRD $\geq$30 years after donation. We could not make conclusions about causality because of the retrospective nature of our study. Because the identification of kidney donors was based on claims data, our study cohort was limited to those with Medicare as primary insurance. Although there is high specificity for comorbid conditions obtained from the Medical Evidence Form 2728, its low sensitivity might have led to differential bias when comparing 2 groups.[22] Due to substantial missing data, we did not include serum albumin, BMI, predialysis nephrology care, and vascular access as covariates in the Cox regression model as a primary analysis. However, in sensitivity analyses that accounted for available data on serum albumin and BMI, the estimated HRs for death for both groups were similar to the primary results. Propensity score techniques can only balance known confounders and do not account for unmeasured covariates that may not be readily available in a large registry database such as the USRDS.

In conclusion, we observed a lower mortality rate in living kidney donors with ESRD compared with propensity-matched nondonors, particularly in the first 10 years after starting dialysis. We validated and expanded on the existing literature by using the propensity score method over traditional multivariable regression. We anticipate that this additional data would guide the clinician in obtaining informed consent and facilitate communication with prospective donors during the donor evaluation process. We believe that it would be important for potential donors to incorporate these study findings in their decision-making process to pursue kidney donation.

DISCLOSURE

All the authors declared no competing interests.

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AUTHOR CONTRIBUTIONS

AB, DGS, RMJ, MOS, MJ, BC, and RN participated in research design; AB, DGS, RMJ, MOS, MJ, BC, and RN participated in the writing of the paper; AB, DGS, RMJ, MOS, MJ, BC, and RN participated in the performance of the research; AB, DGS, MJ, RMJ, MOS, and RN contributed new reagents or analytic tools; and AB, DGS, MJ, RMJ, MOS, and RN participated in data analysis.

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