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Brief Communication

Patterns of End-Stage Renal Disease Caused by Diabetes, Hypertension, and Glomerulonephritis in Live Kidney Donors

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Inferences about late risk of end-stage renal disease (ESRD) in live kidney donors have been extrapolated from studies averaging <10 years of follow-up. Because early (<10 years) and late (≥10 years) postdonation ESRD may differ by causal mechanism, it is possible that extrapolations are misleading. To better understand postdonation ESRD, we studied patterns of common etiologies including diabetes, hypertension and glomerulonephritis (GN; as reported by providers) using donor registry data linked to ESRD registry data. Overall, 125 427 donors were observed for a median of 11.0 years (interquartile range 5.3–15.7 years; maximum 25 years). The cumulative incidence of ESRD increased from 10 events per 10 000 at 10 years after donation to 85 events per 10 000 at 25 years after donation (late vs. early ESRD, adjusted for age, race and sex: incidence rate ratio [IRR] $_{1.3}1.7_{2.3}$ [subscripts are 95% confidence intervals]). Early postdonation ESRD was predominantly reported as GN-ESRD; however, late postdonation ESRD was more frequently reported as diabetic ESRD and hypertensive ESRD (IRR $_{2.3}7.7_{25.2}$ and $_{1.4}2.6_{4.6}$, respectively). These time-dependent patterns were not seen with GN-ESRD (IRR $_{0.4}0.7_{1.2}$). Because ESRD in live kidney donors has traditionally been reported in studies averaging <10 years of follow-up, our findings suggest caution in extrapolating such results over much longer intervals.

Abbreviations: CKD, chronic kidney disease; CMS, Centers for Medicare and Medicaid Services; eGFR, estimated GFR; ESRD, end-stage renal disease; GN, glomerulonephritis; IQR, interquartile range; IRR, incidence rate ratio; OPTN, Organ Procurement and Transplantation Network; SRTR, Scientific Registry of Transplant Recipients

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Introduction

Recent reports suggest an increase in the risk of end-stage renal disease (ESRD) following live kidney donation (1–3). Estimates of this risk correlate with the duration of donor follow-up: 1.34 cases of donor ESRD per 10 000 person-years in a U.S. national study with a mean follow-up of 9.8 years (4) versus 3.02 cases per 10 000 person-years in a Norwegian national study with a median follow-up of 15.1 years (1). Ideally, donors seek information about lifetime risk of ESRD; meanwhile, observational data averaging <10 years of follow-up inform our understanding of early postdonation ESRD, and extrapolations of these data have attempted to make inferences about late postdonation ESRD (2,5). Because the risk of ESRD may increase over time, it is plausible that extrapolations based on the first decade of follow-up substantially underestimate the proportion of donors who develop ESRD in subsequent decades (6,7).

ESRD in the early postdonation period is unlikely to result from diabetes and hypertension because these conditions are absolute and relative contraindications to live kidney donation (8–10); however, glomerulonephritis (GN) may cause early ESRD (1,4). GN may be associated with poorly characterized molecular risks in those with normal renal function (11,12) and may also be associated with poorly characterized genetic risks in donors who are biologically related to live donor kidney transplant recipients with GN-ESRD (13,14), making predonation screening challenging. Moreover, if proteinuria and persistent hematuria develop after donation as manifestations of GN, they may in some instances progress rapidly to ESRD even after interventions are implemented (13). By contrast, ESRD in the late postdonation period may result from *de novo* diabetes (15) and what providers commonly report as hypertensive nephrosclerosis (16),

both systemic conditions associated with aging and arteriosclerosis.

To better understand the patterns of postdonation ESRD without extrapolation, we studied cause-specific cumulative incidence, exploring diabetes, hypertension and GN as commonly reported etiologies with potentially different patterns of development. To inform extrapolations and risk prediction, we estimated the hazard rate of ESRD caused by these commonly reported diseases for each successive postdonation year.

Methods

Data sources

This study used data from the Scientific Registry of Transplant Recipients (SRTR). The SRTR data system includes data on all donors, waitlisted candidates and transplant recipients in the United States, submitted by the members of the Organ Procurement and Transplantation Network (OPTN) and has been described elsewhere (17). The Health Resources and Services Administration, U.S. Department of Health and Human Services, oversees the activities of the OPTN and SRTR contractors.

Study population

All live kidney donors who underwent donor nephrectomy in the United States between October 1, 1987, and July 31, 2014, were included in this study. Those who donated after April 1, 1994 (the first available outcomes linkage), entered the analysis on the date of donation, whereas those who donated before April 1, 1994, entered on this date as late entries (i.e. data from the date of donation through April 1, 1994, were left censored).

Outcome ascertainment

Incident ESRD was defined as the initiation of maintenance dialysis or receipt of a living or deceased donor kidney transplant, whichever was identified first (2). For donors with incident ESRD, the cause of ESRD as reported by providers was ascertained from the Centers for Medicare and Medicaid Services (CMS) medical evidence form 2728 (a validated tool) (18,19) and, because of small or no case counts for some categories (not considered for this study), reclassified into three broad categories: diabetes, hypertension or large vessel disease, and GN. Diabetes includes type II (adult-onset type or unspecified type) and type I (juvenile type, ketosis prone diabetes). Hypertension includes renal disease caused by hypertension (no primary renal disease), renal artery stenosis, renal artery occlusion, and cholesterol and renal emboli. GN includes GN (histology not examined), focal segmental GN, membranous nephropathy, membranoproliferative GN, dense deposit disease, IgA nephropathy, IgM nephropathy (proven by immunofluorescence), rapidly progressive GN, Goodpasture's syndrome, postinfectious GN and other proliferative GN.

Cumulative incidence of cause-specific ESRD

We used the Kaplan–Meier method to generate three unadjusted cumulative incidence estimates, one each for diabetic ESRD, hypertensive ESRD and GN-ESRD. Donors contributed follow-up time from the day they entered the study until they developed ESRD, died or reached the end of study follow-up (July 31, 2014).

Incidence and hazard rate of cause-specific ESRD in the early versus late postdonation period

We used Poisson regression to generate three adjusted incidence rate ratios (IRRs; adjusted for age, sex, and race), one each for diabetic ESRD, hypertensive ESRD and GN-ESRD. Each adjusted IRR compared the incidence of cause-specific ESRD in the late postdonation period (10–25 years after donation) with the early postdonation period (<10 years after donation). For each cause-specific model, we divided the follow-up time into 10–25 years versus <10 years after donation and used these time periods as the primary exposures under investigation; however, we also reported the associations between risk of ESRD and age, race and sex. We subsequently used a three-parameter generalized gamma regression (β, σ, λ) to describe the hazard rate of ESRD for these common etiologies for every successive year after donation (20).

Statistical analysis

Two types of absolute risk were estimated: cumulative incidence (using Kaplan–Meier and generalized gamma models) and hazard rate (using generalized gamma models). We also estimated one type of relative risk: the incidence rate ratio (IRR; using multivariable Poisson models) to compare follow-up beyond 10 years with the first 10 years of follow-up. Confidence intervals are reported as per the method of Louis and Zeger (21). All analyses were performed using Stata 14.0/MP for Linux (Stata Corp, College Station, TX). All hypothesis tests were two-sided ($\alpha = 0.05$).

Results

Study population

Among 125 427 live kidney donors, the median age at donation was 40 years, 58.9% were female, 74.8% were of white or other race, 37.1% were college graduates and 31.3% were unrelated to their recipient. The median BMI (kg/m^2) was 27, median serum creatinine was 0.8 mg/dL and median estimated GFR (eGFR) was 98 mL/min per 1.73 m^2. Overall, 59% of the donors in this study underwent nephrectomy between 1987 and 2005; the rest underwent nephrectomy between 2006 and 2014 (Table 1). There were no substantive differences in demographic and health characteristics between the pre- and post-2005 donor populations.

Cumulative incidence of cause-specific ESRD

Donors were followed for a median of 11.0 years (interquartile range 5.3–15.7 years, maximum 25 years). Over 1 329 964 person-years, 257 donors developed ESRD; 158 (61%) of these were reported as diabetic ESRD (n = 33), hypertensive ESRD (n = 70) or GN-ESRD (n = 55). Of these commonly reported etiologies, ESRD in the early postdonation period was predominantly GN-ESRD. By contrast, ESRD in the late postdonation period was reported most frequently as diabetic and hypertensive ESRD (Table 2).

Incidence rate of cause-specific ESRD in the late versus early postdonation periods

The cumulative incidence of ESRD increased from 10 ESRD cases per 10 000 at 10 years after donation to 85 ESRD cases per 10 000 at 25 years after donation (late vs. early ESRD, adjusted for age, race and sex: IRR $_{1.3}1.7_{2.3}$). More specifically, the incidence of diabetic ESRD was higher in the late postdonation period compared with the early postdonation period (IRR $_{2.3}7.7_{25.2}$),

Table 1: Characteristics of live kidney donors at the time of donation, United States, October 1, 1987, to July 31, 2014[1]

Characteristic	n = 125 427
Age, years, median (IQR)[2]	40 (31–48)
18–39	49
40–49	29
50–59	17
≥60	4
Female (%)[2]	59
Race or ethnicity (%)[2]	
White or other	75
Black	13
Hispanic	12
Education (%)[3]	
High school or less	35
Attended college	28
Graduate or more	37
Relationship (%)[2]	
Parent/child	30
Sibling/other related	39
Unrelated	31
BMI, kg/m^2, median (IQR)[4]	27 (24–30)
<25	32
25–29	41
30–34	21
≥35	5
Creatinine, mg/dL, median (IQR)[5]	0.8 (0.7–1.0)
eGFR, mL/min/1.73 m^2, median (IQR)[5,6]	98 (84–110)
<80	18
80–99	35
≥100	47
Blood pressure, mmHg[7]	
SBP, median (IQR)	120 (111–130)
<120	45
120–139	47
≥140	8
DBP, median (IQR)	74 (68–80)
<80	69
80–89	26
≥90	5
Year of transplant (%)[2]	
1987–1993	11
1994–1997	11
1998–2001	16
2002–2005	21
2006–2009	20
2010–2014	21

eGFR, estimated GFR; IQR, interquartile range.
[1]Data before April 1, 1994, were left censored.
[2]Data on age, sex, race or ethnicity, donor/recipient relationship, and year of transplant were available throughout the study.
[3]Not available before 1999, 50% with missing values in 2000, 30% with missing values in 2005 and 10% with missing values in 2009.
[4]Not available before 1998, 57% with missing values in 1999, 20% with missing values in 2000 and 10% with missing values in 2005.
[5]Not available before 1999, 49% with missing values in 1999, 11% with missing values in 2000 and 6% with missing values in 2001.
[6]Estimated using the Chronic Kidney Disease Epidemiology Collaboration equation (41).
[7]Not available before 1999, 43% with missing values in 1999, 20–30% with missing values from 2000 to 2003, 9–16% with missing values from 2004 to 2007.

Table 2: Cumulative incidence of ESRD following live kidney donation, United States, October 1, 1987, to July 31, 2014

	Cumulative incidence of ESRD per 10 000 donors By cause of ESRD[1]		
Year	Diabetes (n = 33)[2]	Hypertension (n = 70)[3]	GN (n = 55)[4]
5	$_{0.00}0.10_{0.70}$	$_{0.10}0.30_{0.90}$	$_{0.30}0.70_{1.50}$
10	$_{0.10}0.40_{1.20}$	$_{1.20}2.00_{3.20}$	$_{2.29}3.40_{4.90}$
15	$_{1.10}2.00_{3.59}$	$_{5.40}7.40_{10.1}$	$_{4.59}6.30_{8.60}$
20	$_{5.80}8.90_{13.6}$	$_{11.5}15.3_{20.7}$	$_{8.10}11.1_{15.3}$
25	$_{9.69}14.8_{22.7}$	$_{20.2}28.2_{39.2}$	$_{9.19}12.8_{17.8}$

ESRD, end-stage renal disease; GN, glomerulonephritis.
[1]Cause of ESRD as per the Centers for Medicare and Medicaid Services form 2728 narrative (18).
[2]Type II: adult-onset type or unspecified type diabetes; type I: juvenile type, ketosis prone diabetes.
[3]Renal disease due to hypertension (no primary renal disease), renal artery stenosis, renal artery occlusion, and cholesterol and renal emboli.
[4]GN includes GN (histology not examined), focal segmental GN, membranous nephropathy, membranoproliferative GN, dense deposit disease, IgA nephropathy, IgM nephropathy (proven by immunofluorescence), rapidly progressive GN, Goodpasture's syndrome, postinfectious GN and other proliferative GN.

higher in older compared with younger donors (IRR $_{1.0}1.3_{1.8}$ per 10-year increase in age), higher in black compared with white donors (IRR $_{1.9}4.0_{8.5}$) and higher in male compared with female donors (IRR $_{2.5}5.0_{10.0}$). Similarly, the incidence of what was reported as hypertensive ESRD was higher in the late postdonation period compared with the early postdonation period (IRR $_{2.3}2.6_{4.6}$), higher in older compared with younger donors (IRR $_{0.9}1.1_{1.3}$ per 10-year increase in age), higher in black compared with white donors (IRR $_{2.3}3.9_{6.7}$) and higher in male compared with female donors (IRR $_{1.3}2.0_{3.3}$). By marked contrast, the incidence of GN-ESRD was no higher in the late postdonation period compared with the early postdonation period (IRR $_{0.4}0.7_{1.2}$), no higher in older compared with younger donors (IRR $_{0.6}0.8_{1.1}$ per 10-year increase in age), almost an order of magnitude higher in black compared with white donors (IRR $_{4.1}7.3_{12.8}$) and only marginally higher in male compared with female donors (IRR $_{0.9}1.7_{2.5}$) (Table 3).

Hazard rate of cause-specific ESRD

The hazard rate of diabetic ESRD increased exponentially from $_{0.1}0.2_{0.3}$ ESRD cases per 10 000 donors per year at

Table 3: Incidence of late postdonation ESRD 10–25 years following live kidney donation compared with early postdonation ESRD 0–9 years following live kidney donation, United States, October 1, 1987, to July 31, 2014

	IRRs from cause-specific ESRD models[1]		
	Diabetes	Hypertension	Glomerulonephritis
Timing of postdonation ESRD			
<10 years (early)	Reference	Reference	Reference
10–25 years (late)	$_{2.3}7.7_{25.2}$	$_{1.5}2.6_{4.6}$	$_{0.4}0.7_{1.2}$
Age, per 10-year increase[2]	$_{1.0}1.3_{1.8}$	$_{0.9}1.1_{1.3}$	$_{0.6}0.8_{1.1}$
Race or ethnicity			
White or other	Reference	Reference	Reference
Black	$_{1.9}4.0_{8.5}$	$_{2.3}3.9_{6.7}$	$_{4.1}7.3_{12.8}$
Hispanic	$_{0.2}0.8_{3.4}$	$_{1.0}2.1_{4.1}$	$_{0.2}0.8_{2.6}$
Sex			
Female	Reference	Reference	Reference
Male	$_{2.5}5.0_{10.0}$	$_{1.3}2.0_{3.3}$	$_{0.9}1.7_{2.5}$

ESRD, end-stage renal disease; IRR, incidence rate ratio.
[1]IRRs from Poisson parametric models 1–3. The parametric assumption is that incidence rate was constant <10 years after donation and may change to another constant rate 10–25 after donation, IRR≠1.
[2]Incidence rate ratios were adjusted for age, sex, and race or ethnicity. Inferences were the same regardless of how age was modeled (lagged or not lagged by 10 years in the postdonation period) and how variance was quantified (clustered or not on the individual donor; a donor might have contributed person-time to the early and late postdonation periods).

10 years after donation to $_{1.0}2.5_{3.8}$ ESRD cases per 10 000 donors per year at 25 years after donation. Similarly, the hazard rate of what providers reported as hypertensive ESRD increased from $_{0.5}0.6_{0.8}$ ESRD cases per 10 000 donors per year at 10 years after donation to $_{1.7}2.9_{4.1}$ ESRD cases per 10 000 donors per year at 25 years after donation. By contrast, the hazard rate of GN-ESRD did not change significantly, from $_{0.5}0.61_{0.8}$ ESRD cases per 10 000 donors per year at 10 years after donation to $_{0.3}0.67_{1.3}$ ESRD cases per 10 000 donors per year at 25 years after donation (Table 4 and Figure 1).

Discussion

In this national study of cause-specific ESRD in kidney donors, there was a 7.7-fold higher risk of late (10–25 years) versus early (<10 years) postdonation diabetic ESRD. Similarly, there was a 2.6-fold higher risk of late versus early postdonation events reported by providers as hypertensive ESRD. By contrast, there was no significant change over time in event rate for GN-ESRD. An emerging belief, supported by the relatively high incidence of GN-ESRD in the first 10 postdonation years in this study, is that kidney diseases that progress quickly enough to be enumerated within 10 years of nephrectomy are predominately glomerulonephritides (22,23). As such, our findings support the view that extrapolations of postdonation risk of ESRD based on the first decade of follow-up substantially underestimate the proportion of donors that develop ESRD in subsequent decades (15), especially for ESRD cases with a long prodromal course. Methods that account for the development of diabetes, hypertension and other risk factors are necessary for improved prediction of late postdonation risk of ESRD, particularly in selected cohorts such as kidney donors.

Unlike prior reports that have treated ESRD in donors as an all-encompassing clinical outcome (1–4,16,24), this study viewed ESRD in donors as an end point preceded by biologically distinct pathways that providers reported as diabetic ESRD, hypertensive ESRD and GN-ESRD. In showing that the three most commonly reported causes of ESRD in donors mirror those in the general population (although not necessarily in the same order of relative frequently) (25), our findings suggest that similar biological pathways lead to ESRD in these two populations. Unlike the general population, donors were screened for

Table 4: Parametric models for risk of cause-specific ESRD following live kidney donation, United States, October 1, 1987, to July 31, 2014

	Generalized gamma parameters					
ESRD model[1]	β	p	σ	p	λ	p
Diabetes	$_{2.75}5.11_{7.46}$	<0.01	$_{0.02}0.41_{9.35}$	0.6	$_{-1.60}0.63_{2.88}$	0.6
Hypertension	$_{2.67}5.42_{8.16}$	<0.01	$_{0.00}0.40_{71.8}$	0.7	$_{-3.80}0.91_{5.70}$	0.8
Glomerulonephritis	$_{5.91}11.2_{16.6}$	<0.01	$_{4.95}15.8_{50.9}$	<0.01	$_{-31.0}-9.50_{12.2}$	0.4

[1]Parametric models 1–3: generalized gamma. For interpretation of these parameters, see Discussion.

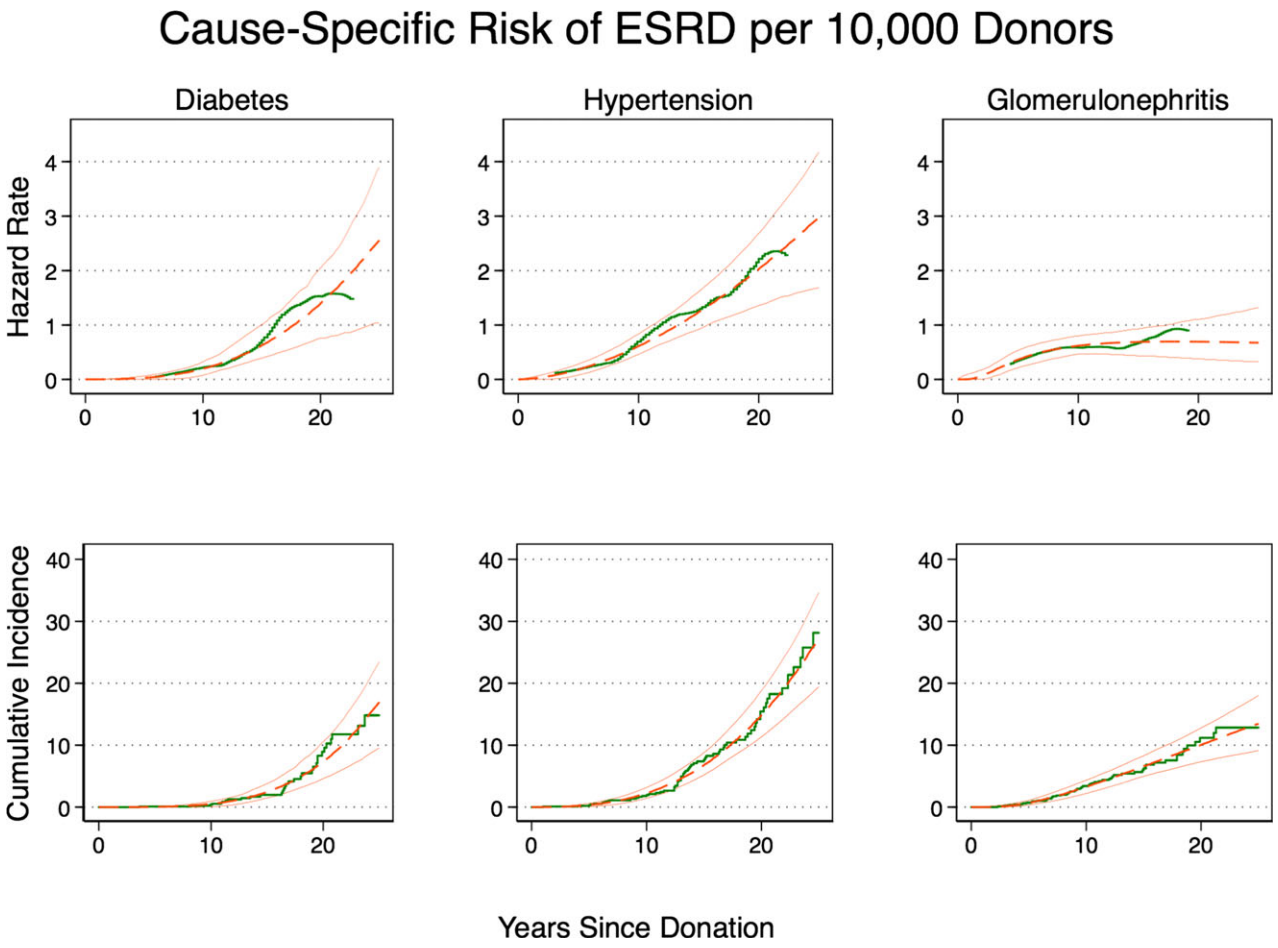

Figure 1: Hazard rates and cumulative incidence of cause-specific end-stage renal disease (ESRD) in live kidney donors, United States, October 1, 1987, to July 31, 2014. Kaplan–Meier curves (green, solid line) and generalized gamma models (orange, dashed line; 95% confidence interval, solid line) illustrate the following results for diabetes: a low unadjusted hazard rate of ESRD <10 years after donation ($_{0.1}0.2_{0.3}$ case per 10 000 donors per year at 10 years) and a substantial increase in unadjusted hazard rate of ESRD 10–25 years after donation ($_{1.0}2.5_{3.8}$ cases per 10 000 donors per year at 25 years). Results for hypertension show a low unadjusted hazard rate of ESRD at <10 years after donation ($_{0.5}0.6_{0.8}$ case per 10 000 donors per year) and a substantial increase in the unadjusted hazard rate of ESRD at 10–25 years after donation ($_{1.7}2.9_{4.1}$ cases per 10 000 donors per year). Results for glomerulonephritis (GN) show a relatively constant unadjusted hazard rate of ESRD at 0–25 years after donation ($_{0.5}0.61_{0.8}$ case per 10 000 donors per year at 10 years vs. $_{0.3}0.67_{1.3}$ case per 10 000 donors per year at 25 years). As per generalized gamma regression, the cumulative incidence of cause-specific ESRD at 25 years was $_{9.3}16.6_{22.7}$, $_{19.0}26.8_{33.7}$ and $_{9,1}13.4_{17.7}$ per 10 000 donors for diabetes, hypertension and GN (very closely approximating the Kaplan–Meier estimates).

diabetes (via blood tests for hyperglycemia), hypertension (via blood pressure measurements) and GN (via urine tests for hematuria and proteinuria), with confirmation of normal test results prior to donation (8–10,26). This explains why donors had very low risk of ESRD in the early postdonation period but an exponential increase in risk in the late postdonation period. Donor nephrectomy represents a 50% reduction in nephron number and a 25–40% reduction in GFR (27,28), and for these reasons, *de novo* renal disease may reach ESRD sooner in donors compared with their healthy nondonor counterparts (1–3). Our study, however, shows that the absolute risk of what providers reported as diabetic ESRD, hypertensive ESRD and GN-ESRD was very low over a 25-year period. As such, our findings reaffirm the effectiveness of the current practices of donor evaluation (8–10).

Given that some primary glomerular disease remains undiagnosed and may be reported as hypertensive ESRD (29–32), a very important limitation of our study is that clinical data including serology and renal biopsy results were not available to us to accurately define the cause of ESRD. And like other large population-based studies of ESRD in the general population (33–36), our kidney donor study was limited by the absence of baseline and

longitudinal assessments of renal function and blood pressure. As such, like other studies, we were unable to evaluate the incidence of ESRD attributable to hypertension in the specific subgroup of participants with benign essential hypertension, normal serum creatinine and no albuminuria (i.e. the profile of a patient with hypertension who is cleared for donor nephrectomy). In other words, our data cannot quantify the proportion of cases that providers reported as hypertensive ESRD that, in fact, resulted from benign essential hypertension.

That said, in a somewhat recent study of 316 675 participants with eGFR >60 mL/min per 1.73 m^2 and negative dipstick urinalysis for proteinuria or hematuria, a strong, direct and graded association was observed between systolic blood pressure and the risk of ESRD over 8 210 431 person-years of follow-up; this association was observed throughout the distribution of blood pressure readings (37). For these reasons, there might be an important and potentially preemptive role for the monitoring and control of blood pressure throughout the postdonation period regardless of hypertension status at the time of donation. Consequently, it is important to discuss the second limitation: Although we studied one of the most important physiological consequences of donor nephrectomy, ESRD represents a subset of stage 5 of chronic kidney disease (CKD) (38)—the tip of the iceberg, as it were (39)—and our study was not able to quantify the interim incidence and hazard rates for CKD or to describe the postdonation patterns of risk factors including hyperglycemia, hypertension and proteinuria. Because the earlier stages of CKD are more likely to lead to heart disease than to ESRD (40), our analysis was limited in not being able to characterize these very important nonrenal outcomes of CKD.

Granted, our study has several strengths, and these mainly lie in the generalizability of its inferences to all donors in the United States; the large sample size, permitting the study of rare outcomes; and the robustness of ESRD ascertainment. We identified all donors who developed ESRD by either initiation of maintenance dialysis treatment or receipt of a deceased or a living donor transplant. Using CMS and OPTN data, we ascertained the cause of ESRD as reported by care providers to the CMS (18). Our study is the first, to the best of our knowledge, to provide empirical support of the view that diabetic ESRD (15) and what is commonly reported as hypertensive ESRD may be among the leading determinants of risk of ESRD over the lifetime of kidney donors, despite being initially rare. Using parametric survival methods (20), our study characterized the risk of ESRD by reported underlying cause in each successive postdonation year. Because we also used nonparametric Kaplan–Meier methods to report our risk estimates, we were able to affirm that our parametric models fit that data well.

Our parametric models, fit using three-parameter generalized gamma regression (β, σ, λ), described the cause-specific hazard rate of ESRD for every successive postdonation year. Because β is directly correlated with the median time to ESRD for fixed values of σ and λ, it is difficult to interpret because σ and λ varied by reported cause of ESRD in this study; however, the hazard rate of ESRD increased with time if σ had values >0 and <1, as observed for what providers reported as diabetic and hypertensive ESRD. This reflects the biological characteristic of these diseases: They evolved only after cumulative, chronic exposure to putative risk factors including high blood glucose and high blood pressure. By contrast, the hazard rate was relatively constant if σ had values >1, as observed for GN-ESRD. This reflects the biological characteristic of glomerulonephritides: In some instances, they may evolve relatively rapidly in susceptible persons regardless of the number of years postdonation but perhaps might be dependent on susceptibility factors that are poorly understood (11–14). Finally, the percentiles of the parameters β and σ are described by λ, which describes how clustered ESRD events were in the postdonation years. For what providers reported as diabetic and hypertensive ESRD, ESRD events were clustered much later in time than in the early years following donor nephrectomy. By marked contrast, GN-ESRD events were spread somewhat uniformly over time.

In conclusion, because ESRD in live kidney donors has traditionally been reported in studies averaging <10 years of follow-up, our findings suggest caution in extrapolating such results over much longer intervals. In addition, because the risk of what providers reported as diabetic and hypertensive ESRD increases exponentially over time, these findings emphasize the importance of follow-up and surveillance of kidney donors for hyperglycemia, blood pressure elevation and renal function for many decades following nephrectomy.

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Disclaimer

The analyses described here are the responsibility of the authors alone and do not necessarily reflect the views or policies of the Department of Health and Human Services, nor does mention of trade names, commercial products or organizations imply endorsement by the U.S.

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Recipients (SRTR). The interpretation and reporting of these data are the responsibility of the authors and in no way should be seen as an official policy of or interpretation by the SRTR or the U.S. Government.

Disclosure

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