

A Study of Renal Outcomes in African American Living Kidney Donors

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Background. Little is known about the long-term outcomes of African American living kidney donors (AALKDs). We undertook this study to describe renal outcomes of AALKDs several years after donation.

Methods. We invited 107 AALKDs to come for follow-up health evaluation.

Results. Thirty-nine subjects (36.4%) completed evaluation at a mean of 7.1 ± 1.6 (range, 3.9–10.2) years postdonation. The mean estimated glomerular filtration rate using the abbreviated Modification of Diet in Renal Disease equation [eGFR(MDRD)] at follow-up was 72.1 ± 16.3 (range, 42–106) mL/min/1.73 m², and 18% of subjects had an eGFR(MDRD) of 30 to 59. The mean absolute and relative decrease in eGFR(MDRD) from the time of donation to follow-up was 30.5 ± 16.4 mL/min/1.73 m² and 28.8%, respectively. Subjects whose body mass index was more than or equal to 35 kg/m² (n=8) were found to have a greater decrement in eGFR(MDRD) than those with body mass index less than 35 kg/m² (40.1 ± 7.3 and 28.3 ± 17.1 mL/min/1.73 m², respectively; $P=0.009$). Sixteen (41%) were hypertensive at follow-up, as defined as treatment with antihypertensive medications (n=8) or average blood pressure of more than or equal to 140 systolic or 90 mm Hg diastolic (n=10, of whom two were on antihypertensive medications). One subject had macroalbuminuria (>300 μg/mg creatinine), and six (15.4%) had microalbuminuria (30–300 μg/mg creatinine).

Conclusions. AALKDs experience a substantial incidence of hypertension and a modest drop in eGFR(MDRD) postdonation, and obesity may increase the magnitude of renal decline. Further study is urgently needed to determine the long-term risks of AALKDs.

Keywords: Living donor, Kidney transplantation, African Americans.

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Living donor kidney transplantation has been encouraged as a source of kidneys for transplantation. A fairly strong body of literature has failed to show long-term adverse health effects of unilateral nephrectomy performed on a healthy individual (1–9). However, the long-term safety has not been demonstrated with live kidney donation from individuals with more than minimal risk for development of chronic kidney disease (CKD), and there are many reasons to believe that

African American living kidney donors (AALKDs) may be at higher risk for adverse long-term renal complications from unilateral nephrectomy. Numerous studies have demonstrated higher risks of hypertension (HTN) (10), CKD (11, 12), and end-stage renal disease (12–15) in African Americans. Furthermore, a recent study indicated that African Americans made up a disproportionately high percentage of live kidney donors who were later listed for kidney transplantation (16).

Previous studies that support the long-term safety of unilateral nephrectomy do not provide sufficient data to assure safety in AALKDs. In fact, three of the most important donor follow-up studies did not specify racial makeup of the cohort and were conducted in areas with relatively low proportions of blacks in the general population—Germany (2), Sweden (3), and Minnesota (4). Laskow et al. (17) assessed outcomes after approximately 6 years in a small series of AALKDs (n=24). Compared with the same number of white donors, AALKDs had higher mean arterial pressure but similar changes in serum creatinine (SCr). Clearly, studies that examine the safety of live kidney donation from African Americans are needed. We undertook this study to describe the renal outcomes in a cohort of AALKDs.

METHODS

After obtaining approval from the University of Maryland Institutional Review Board, locally residing AALKDs

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who donated at the University of Maryland Medical Center between March 1, 1996, and February 28, 2002, inclusive were invited to come to the University of Maryland General Clinical Research Center (GCRC) for a health questionnaire and measurement of height, weight, three blood pressures (BPs) (using a standardized BP measurement technique [18]), urine albumin, urine creatinine, and SCr. A letter of introduction was mailed to the potential subjects with a preaddressed, postage-paid reply form that allowed them to decline participation. If we did not receive the reply form within 2 weeks, a study coordinator then contacted the subjects by phone to invite donors to participate in the study. Predonation SCr and early postdonation SCr values were obtained from a computerized database.

We estimated renal function using five different equations:

1. The abbreviated Modification of Diet in Renal Disease (MDRD) equation estimated glomerular filtration rate [eGFR(MDRD)].

$$\text{eGFR(MDRD)} = 186 \times \text{SCr}^{-1.154} \times \text{age}^{0.203} \times 0.742 \text{ (if female)} \\ \times 1.210 \text{ (if black) (19).}$$

2. Mayo Clinic quadratic formula as described by Rule et al. (20) to estimate GFR (eGFR).

$$\text{GFR} = \exp \left(1.911 + \frac{5.249}{\text{SCr}} - \frac{2.114}{\text{SCr}^2} - 0.00686 \times \text{age} \right. \\ \left. - 0.205 \text{ (if female)} \right).$$

If SCr was less than 0.8 mg/dL, then a value of 0.8 was used.

3. The body surface area (BSA)-adjusted Cockcroft-Gault formula for creatinine clearance using actual body weight (actual CG-Cl_{cr}): [(140 - age) × actual body weight / (72 × SCr)] × (0.85 if female). We adjusted the creatinine clearance estimate for by multiplying by (1.73 m²/BSA in m²).
4. The BSA-adjusted Cockcroft-Gault formula for creatinine clearance using calculated lean body weight rather than actual body weight (leanCG-Cl_{cr}).
5. The African American Study of Hypertension and Kidney Disease simplified (three variable) eGFR (21): 329 × (SCr)^{-1.096} × (age)^{-0.294} × (0.736 for women).

Daily albumin excretion rate was estimated using the urine albumin-to-creatinine ratio, defined as the ratio of urine albumin concentration to creatinine concentration expressed as microgram of albumin per milligram of creatinine. Microalbuminuria was defined as urine albumin-to-creatinine ratio of 30 to 300 μg/mg creatinine and macroalbuminuria as more than 300 μg/mg creatinine. HTN was defined as treatment with hypertensive drugs for an indication of HTN, the average systolic BP at the GCRC of more than or equal to 140 mm Hg, or the average diastolic BP at the GCRC of more than or equal to 90 mm Hg. Median household family annual income (in 1999 dollars) and percentage of families below poverty level for donor's zip code at the time of donation per Census 2000 data were obtained from the following website:

http://factfinder.census.gov/home/saff/main.html?_lang=en. A high poverty zip code was defined as poverty level greater than 150% of the U.S. national average poverty level of 9.2% for the same year. Donor survival status was obtained by entering the social security into the following website, which searched the U.S. Social Security Death Index: <http://ssdi.rootsweb.ancestry.com>.

Statistical Methods

Continuous variables were reported as mean ± standard deviation and were compared using Student's *t* tests. Categorical variables were reported as absolute numbers or percentages and will be compared using 2 × 2 contingency tables and chi-squared tests. Correlation between two continuous values was estimated with Pearson's correlation coefficient (*r*). *P* values less than 0.05 were considered statistically significant.

RESULTS

Table 1 summarizes the results of recruitment, which took place between December 2004 and December 2007. Table 2 provides details of various baseline parameters that were available in our database for the study group (group 1) when compared with the group of AALKDs who were invited to participate but did not accept (group 2) and to the group of AALKDs who were not invited because they did not reside locally (group 3). As can be seen, the demographic, survival, and baseline clinical parameters at the time of donation were similar in the study group and the other AALKD groups. There were no early or late donor deaths.

Table 3 details the demographics and outcomes of interest in the group of subjects who completed the evaluation in the GCRC. It shows that the estimations of renal function and therefore the proportion of subjects with eGFR or creatinine clearance less than 60 mL/min vary dramatically depending on the estimation equation used. To assist in the interpretation of the results, data from the study by Ibrahim et al. (9) on similar outcomes in a mostly (99.2%) white cohort of 255 donors with long-term follow-up (mean follow-up of 12.2 ± 9.2 years) at the University of Minnesota GCRC were included in Table 3. We also included

TABLE 1. Recruitment results

African American live kidney donors who donated during study period	192
Locally residing African American live kidney donors to whom letters were sent = invited subjects	107
Replies obtained	88
Refused to participate	19 (17.8% of those recruited)
Agreed to participate	69 (64.5% of those recruited)
Consented	48
Evaluated in GCRC	39 (36.4% of invited subjects, 20.3% of all AALKDs)

GCRC, General Clinical Research Center; AALKD, African American living kidney donors.

TABLE 2. Baseline characteristics and early renal function in study group, recruited AALKDs who did not participate, and nonrecruited AALKDs (nonlocally residing)

Characteristics	Group 1: study participants	Group 2: invited AALKDs who did not participate	Group 3: noninvited AALKDs (nonlocally residing)
Number subjects	39	68	92
Age (yr) at donation	37.4±11.0	33.7±9.9	35.8±10.0
Male (%)	34.2	34.8	40.2
BMI	28.9±5.9	29.3±6.1	28.2±5.5
Predonation serum creatinine	0.94±0.26	0.92±0.22	1.04±0.97
Predonation eGFR(MDRD)	102.5±20.3	102.8±21.2	102.5±18.1
Unrelated to recipient (%)	23.7	12.1	21.7
Donor deaths	0	0	0
Donor residence in high-poverty zip code at donation (%)	38.5	28.6	28.8
Median family annual income in donor zip code of residence at time of donation (in thousands of dollars)	\$46.1±20.4	\$42.7±14.4	42.9±14.8

Continuous values presented as mean±standard deviation. There were no statistically significant differences in these parameters in group 2 vs. group 1 and in group 3 vs. group 1.

AALKD, African American living kidney donors; eGFR(MDRD), estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation; BMI, body mass index.

data from an earlier report on a series of 112 University of Minnesota donors, which calculated renal clearances using various estimation equations (6).

Among those subjects who had an eGFR(MDRD) less than 60 mL/min/1.73 m² at the time of follow-up in the GCRC, the individual body mass indexes (BMIs) at time of donation were 28.2, 31.0, 33.0, 36.2, 36.4, 38, and 41.2 kg/m². The BMI of the individual who developed macroalbuminuria was 21.3 kg/m². The mean BMI for those with eGFR(MDRD) less than 60 mL/min/1.73 m² was higher than for those with eGFR(MDRD) more than or equal to 60 mL/min/1.73 m² (35.0±4.5 vs. 27.5±5.18 kg/m², respectively; *P*=0.001). The mean eGFR(MDRD) was lower for those with BMI more than or equal to 35 kg/m² when compared with those with BMI less than 35 kg/m² (61.4±16.3 vs. 74.9±15.4 mL/min/1.73 m², respectively; *P*=0.034). Four of the eight subjects (50%) with BMI more than or equal to 35 kg/m² and 3 of 31 subjects (9.7%) with a BMI less than 35 kg/m² had an eGFR(MDRD) less than 60 mL/min/1.73 m² (*P*=0.008).

Among the 37 subjects who had predonation renal function data available in our database, the follow-up eGFR(MDRD) was 30.5±16.4 mL/min/1.73 m² less than the predonation value (*P*<0.001 on paired *t* test), which represents a 28.8% drop in the serial eGFR(MDRD)s. Subjects whose BMI was more than or equal to 35 kg/m² were found to have a greater absolute decrement in eGFR(MDRD) than those with BMI less than 35 kg/m² (40.1±7.3 and 28.3±17.1 mL/min/1.73 m², respectively; *P*=0.009) as well as a greater relative decrement (39.8% and 26.2%, respectively; *P*=0.024). Figure 1 graphically depicts the eGFR(MDRD)s of the subjects, with obese (BMI ≥30 kg/m²) subjects identified by dashed lines.

We found no difference in absolute or relative decrement in eGFR(MDRD) in male donors when compared with female donors (27.8±17.3 and 32.2±15.9 mL/min/1.73 m², respectively; *P*=nonsignificant [NS]), in donors younger

than 30 years when compared to those 30 years or older (30.9±19.8 and 29.6±14.9 mL/min/1.73 m², respectively; *P*=NS), or in hypertensive when compared with nonhypertensive subjects (30.2±15.4 and 30.7±17.3 mL/min/1.73 m², respectively; *P*=NS). Likewise, we did not find an association with HTN and BMI more than or equal to 35 kg/m² (4 of 8 [50%] with BMI ≥35 kg/m² and 12 of 31 [38.7%] with BMI <35 kg/m², *P*=NS).

Figure 2 plots the duration of time from pre- to post-donation measurement of eGFR(MDRD)s and the absolute change in eGFR(MDRD)s for the subjects. It shows that the longer intervals between measurements were not associated with greater absolute loss of eGFR(MDRD). Rather, longer intervals were associated with lesser loss of eGFR(MDRD). Using linear regression, the slope estimate for β is 3.51±1.6 (95% confidence interval: 0.22–6.8) mL/min/1.73 m² per year between donation and follow-up (*P*=0.037), with correlation coefficient (*r*)=0.34.

DISCUSSION

This is the largest published series describing renal outcomes in a group of African American live kidney donors. Despite the limitations of this study, the data suggest that African American living donors should be carefully selected and advised to have regular medical follow-up.

To address the risk of adverse renal outcomes in AALKDs relative to non-African American donors, we compared outcomes to those reported in the literature on the general live kidney donor population (most of whom were likely not African American). Previous studies by Ibrahim et al. (6, 9) provided the most useful information to help us interpret the GFR changes. These demographically different study cohorts (98%–99% white) were used to compare GFR changes postdonation (6, 9). As can be seen in Table 2, the

TABLE 3. Demographics and outcomes of interest at the time of follow-up evaluation in GCRC

	AALKDs in our current study	University of Minnesota cohort of 255 mostly white live kidney donors(9)
Length of follow-up (from donation to GCRC visit)	7.1±1.6	12.2±9.2
Age at time of follow-up (yr)	46.2±1.6	52.9±9.9
Mean BMI (kg/m ²) at time of follow-up visit	31.1±6.3 (21.5–46.4)	
BMI >30 kg/m ² at time of follow-up visit	18 (46.2%)	29.3%
Average weight gain from donation to GCRC visit (kg/m ²)	2.2±3.7	
Average SBP during GCRC visit (mm Hg)	120.8±14.5 (92–144)	121.8±14.6
Average DBP during GCRC visit (mm Hg)	79.7±9.3 (62–99)	73±8.9
SBP ≥140 or DBP ≥90 at GCRC visit	10 (25.6%)	14.4%
Diagnosed with hypertension (using antihypertensive drugs) before GCRC visit	8 (20.5%)	24.7%
Current diagnosis of hypertension (defined as use of antihypertensive medications or BP >140/90 at time of follow-up visit)	16 (41.0%)	
Undiagnosed hypertension	8 (20.5%)	
Microalbuminuria	6 (15.4%)	
Macroalbuminuria	1 (2.6%)	
Serum creatinine (mg/dL)	1.22±0.25 (0.8–1.7)	1.1±0.2
eGFR(MDRD) <60 mL/min/1.73 m ² or macroalbuminuria	8 (20.5%)	
eGFR(MDRD) <60 mL/min/1.73m ² and macroalbuminuria	0 (0%)	
Diagnosed with diabetes mellitus	1 (2.6%)	3.1%
eGFR(MDRD) (mL/min/1.73 m ²)	72.1±16.4, (42–106), 7 (17.9%) <60	63.7±11.3
		University of Minnesota cohort of 112 mostly Caucasian live kidney donors(6)
eGFR(MDRD) (mL/min/1.73 m ²)	72.1±16.4 (42–106), 7 (17.9%) <60	65±13, 39.3% <60
MC-eGFR (mL/min/1.73 m ²)	78.8±19.4 (42–111), 17.9% (n=7) <60	86±17, 6.2% <60
Actual CG-Cl _{Cr} (mL/min/1.73 m ²)	75.1±19.5 (45–129), 23.1% (n=9) <60	75±17, 22.3% <60
Lean CG-Cl _{Cr} (mL/min/1.73 m ²)	52.7±14.1 (25–80), 69.2% (n=27) <60, 10.3% (n=4) <30	
AASK-eGFR (mL/min/1.73 m ²)	74.4±16.1 (43–107), 6 (15.4%) <60	

Continuous values presented as mean±standard deviation (range from lowest to highest). Proportions presented as total number (percent of total number of subjects evaluated in GCRC).

For comparison, we included similar outcomes at approximately 12-yr postdonation in two series of mostly white donors performed at the University of Minnesota and reported by Ibrahim et al.(6, 9).

AALKD, African American living kidney donors; GCRC, General Clinical Research Center; BMI, body mass index; eGFR(MDRD), estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation; CG-Cl_{Cr}, Cockcroft-Gault creatinine clearance; AASK-eGFR, The African American Study of Hypertension and Kidney Disease simplified (three variable) glomerular filtration rate; BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; MC-eGFR, estimated glomerular filtration rate calculated using the Mayo Clinic quadratic formula.

proportion of subjects with eGFR or creatinine clearance less than 60 mL/min varied in both study groups (from 10.3% to 23.1% in AALKDs and from 6.2% to 39.3% in the Minnesota series [6]); and therefore conclusions about relative risk in the groups varied dramatically depending on which equation was used to calculate glomerular filtration rate or creatinine clearance. However, overall, the risk eGFR less than 60 mL/min does not appear to be greater in African Americans. We suspect that these differences in renal clearance estimates are due to racial effect on the various eGFR equations. The wide fluctuations in the rate of this outcome and the differing conclusions that would be made about the relative risk in these groups further illustrate the limitations of estimating renal function using SCr in this population. The relative loss of

GFR from predonation to follow-up several years later of 28% in our group of AALKDs does not appear to be excessive when considering that reports from the literature indicate that the GFR after uninephrectomy should settle at approximately 75% of the two-kidney baseline (2, 3, 5, 6, 8).

To assess the risk of donation from African Americans, our AALKD cohort should be compared with a group of African American individuals with similar baseline characteristics who did not donate a kidney. Unfortunately, we could not find data in the literature that were sufficiently specific to make valid comparisons with our group. Nevertheless, the microalbuminuria prevalence of 15.4% rate in AALKDs seems reassuringly similar to the National Health and Nutrition Examination Survey III (NHANES III) prevalence of 11% in the general popula-

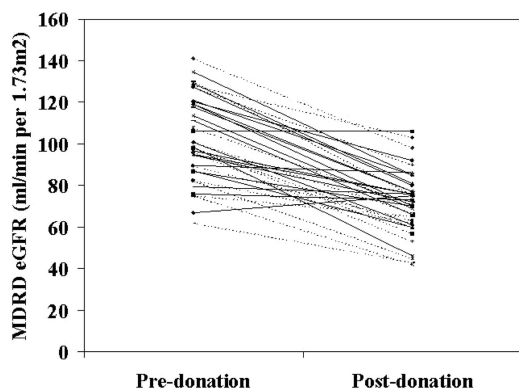


FIGURE 1. eGFR(MDRD)s of the individual subjects predonation and postdonation (at the time of follow-up in the General Clinical Research Center [GCRC]), with obese (body mass index ≥ 30 kg/m²) subjects identified by dashed lines and nonobese individuals identified by solid line. eGFR(MDRD), estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation.

tion (22). Although the prevalence of eGFR or creatinine clearance less than 60 mL/min in our cohort [17.9% using eGFR(MDRD)] is several times the 4.3% prevalence in the general population (22), the significance of this finding is uncertain. Were the solitary kidney state to produce progressive CKD in this population, one might expect it to be due to hyperfiltration injury and would thus expect it to be accompanied by albuminuria. Our data are not consistent with this scenario, as only one of the subject with eGFR(MDRD) less than 60 mL/min/1.73 m² was found to have microalbuminuria, and none had macroalbuminuria. In addition, the finding that longer intervals between measurements were not associated with greater absolute loss of eGFR(MDRD) also argues against progressive CKD in this cohort. These findings may suggest that these relatively low eGFRs may simply be due to reduced renal mass or due to inaccuracies in using SCr to identify individuals with CKD, rather than indicating threatening renal pathologic condition.

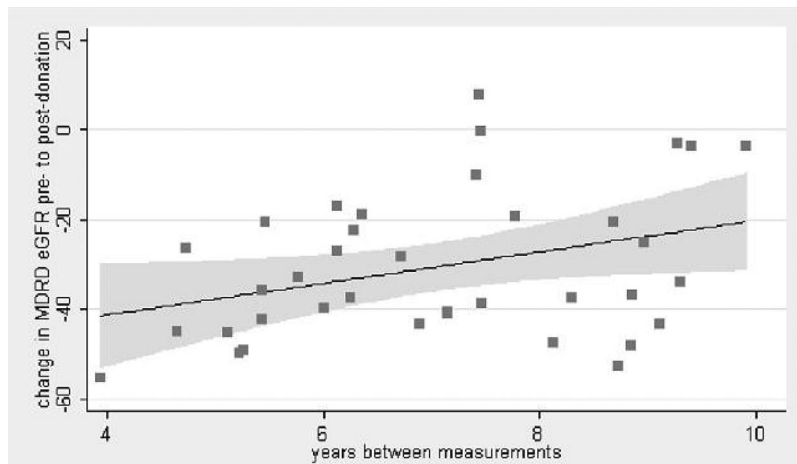
It is important to note that the risk of having eGFR(MDRD) less than 60 mL/min/1.73 m² and the magnitude of absolute and relative loss of eGFR(MDRD) were significantly higher in obese (BMI ≥ 35 kg/m²) AALKDs than in nonobese AALKDs.

This suggests that the combination of these two risk factors for CKD may be especially problematic in live kidney donors.

The most concerning finding in our study was that 41% of the subjects had developed HTN within the follow-up period. To understand the significance of this incidence of HTN in our cohort of AALKDs, we can compare it with the values reported in studies of the general population of live kidney donors (most of whom were not African American). In the study by Ibrahim et al. (9) that reported outcomes on 255 live kidney donors at a mean of 12.2 years postdonation, they found that 24.7% were on antihypertensive medications and that 7.5% had newly diagnosed HTN. In their study, the mean BP was 122/73, when compared with 121/80 mm Hg in our group. In addition, among the 48 studies included in a recent meta-analysis by Boudville et al. (23), which addressed the risk for HTN in the general population of living kidney donors, they found a 5 mm Hg increase in BP over that anticipated with normal aging and that the incidence of HTN varied markedly among the studies—from 0% to 62%, with a crude mean of 19%.

More important than whether AALKDs have a higher risk for HTN than non-African American donors is what the effect of donating will have on BP in AALKDs. This issue is addressed by comparing the rate of HTN in AALKDs to African Americans who have not donated. Given that no donors had a diagnosis of HTN at the time of donation, one should not apply prevalence data from the whole population of African Americans (many of whom would have already been hypertensive at the index time corresponding to donation). Rather, we should compare the incidence rates in the groups. Unfortunately, we could not find applicable incidence rates in the literature. Using the NHANES data (24), the prevalence of HTN for non-Hispanic black men is approximately 20% in the 30- to 39-year-old age group and 36% in the 40- to 49-year-old age group. The prevalence of HTN for non-Hispanic black women is approximately 10% in the 30- to 39-year-old age group and 28% in the 40- to 49-year-old age group. Given that the mean age of the donors in our cohort at the time of donation (when none had a HTN) was 37 years and the mean age at follow-up was 44 years, the “average” donor aged from the former to the latter age group during this study; and therefore these prevalence rates in these age groups could be cautiously used to estimate a control incidence of HTN in nondonating African Americans.

FIGURE 2. Change in eGFR(MDRD) pre- to postdonation plotted against years between measurements for individual study subjects (small solid boxes), with linear regression line (solid black line) and 95% confidence intervals (gray area). eGFR(MDRD), estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation.



An incidence rate of 41% during 7.1 years of follow-up is clearly higher than the incidence rate that would be expected in the general African American population.

Conversely, it is certainly plausible that the proportion of study subjects with HTN may be higher than the proportion in the whole AALKD population, as suggested by findings in the meta-analyses by Boudville et al. (23), which showed that the higher the proportion of donors lost to follow-up in a study, the higher the reported increase in BP. Although the true significance of the 41% incidence of HTN is debatable, this high proportion of AALKDs with HTN in our cohort is concerning given that HTN may be more nephrotoxic in African Americans, given that individuals with one kidney have less renal reserve. Of further concern is that one half of these individuals did not know that their BP was high. Conversely, we found it somewhat comforting that no subject had worse than stage 1 HTN at the time of evaluation and that the hypertensive group of donors had not experienced a greater loss of eGFR(MDRD).

It is important to appreciate the limitations of our study. First, our assessment of renal function and our ability to reliably identify individuals with CKD were limited by our reliance on SCr, and inaccuracies of using the commonly used SCr-based estimation equations in individuals without known CKD has been well described (6, 20). Clearly, all of the renal function estimation techniques that we used have important limitations in this population. Still, it should be noted that a recent study by Ibrahim et al. (6) indicated that the eGFR(MDRD) and actual CG-CICr provide estimates of GFR in former kidney donors that are within the clinically acceptable range of actual GFR. Second, with only 36% of invited subjects completing the evaluation in the GCRC, the broad applicability of our findings to the larger AALKD population is uncertain, even though our comparison of the baseline parameters of those AALKDs who participated versus those who did not participate did not reveal significant differences. It should be noted that this critical limitation has plagued nearly all long-term donor follow-up studies, including the recent landmark study by Ibrahim et al. (9), which achieved a 14.3% participation in follow-up iohexol GFR measurements. Those subjects who decided to participate may have done so because they had reason to be concerned about their health, such that the rates of pathologic condition may be overestimated relative to the whole AALKD population. Alternatively, one could hypothesize that the subjects who take the time to participate were more likely to be the type of person who takes good care of himself/herself.

In conclusion, this study demonstrates the following:

1. A substantial percentage of AALKDs will develop HTN within several years of donation.
2. AALKDs lose a significant proportion of their eGFR(MDRD) after donating a kidney. However, the magnitude of change seems to be similar to that observed in the general kidney donor population, and we found no evidence that the loss of GFR is progressive.
3. Obesity may be a risk factor for renal dysfunction after live kidney donation in AALKDs.

Our findings strongly argue for the need for further study to guide the selection and counseling of potential AALKDs. Our findings also indicate that close monitoring of AALKDs for HTN and CKD is clearly warranted.

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