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A STUDY OF RENAL OUTCOMES IN AFRICAN AMERICAN LIVING KIDNEY DONORS

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Abstract

Background—Very 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—39 subjects (36.4%) completed evaluation at a mean of 7.1 $\pm$ 1.6 (range 3.9–10.2) years post-donation. The mean estimated glomerular filtration rate using the abbreviated MDRD equation (eGFR(MDRD)) at followup was 72.1 $\pm$ 16.3 (range 42–106) ml/min per 1.73m², and 18% of subjects had an eGFR(MDRD) of 30–59. The mean absolute and relative decrease in eGFR(MDRD) from the time of donation to followup were 30.5 $\pm$ 16.4 ml/min per 1.73m² and 28.8%, respectively. Subjects whose BMI was $\geq$ 35 kg/m² (n=8) were found to have a greater decrement in eGFR(MDRD) than those with BMI < 35 kg/m² (40.1 $\pm$ 7.3 and 28.3 $\pm$ 17.1 ml/min per 1.73m², respectively; p = 0.009). Sixteen (41%) were hypertensive at followup, as defined as treatment with antihypertensive medications (n=8) or average BP of $\geq$ 140 systolic or 90 mmHg diastolic (n=10, of whom 2 were on antihypertensive medications). One subject had macroalbuminuria (> 300 mcg/mg creatinine), and 6 (15.4%) had microalbuminuria (30–300 mcg/mg creatinine).

Conclusions—AALKDs experience a substantial incidence of hypertension and a modest drop in eGFR(MDRD) post-donation, 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

Background

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, long-term safety has not been demonstrated with live kidney donation from individuals with a more-than-minimal risk for development of chronic kidney disease (CKD), and there are many reasons

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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 (ESRD) (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).

Prior 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. assessed outcomes after approximately six years in a small series of AALKDs (n=24). Compared to the same number of white donors, AALKDs had higher mean arterial pressure but similar changes in serum creatinine (17). 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 who donated at University of Maryland Medical Center between 3/1/96 and 2/28/02, 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 (using a standardized blood pressure measurement technique(18)), determination of the urine albumin/microalbumin ratio, urine creatinine, and serum creatinine. A letter of introduction was mailed to the potential subjects with a pre-addressed, postage-paid reply form that allowed them to decline participation. If we did not receive the reply form within two weeks, a study coordinator then contacted the subjects by phone to invite donors to participate in the study. Pre-donation serum creatinine and early post-donation serum creatinine values were obtained from a computerized database.

We estimated renal function using five different equations:

1. The abbreviated 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 (MC-eGFR)

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

If SCr was <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 (actualCG-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 body surface area (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 (3-variable) glomerular filtration rate (AASK-eGFR)(21): $329 \times (\text{SCr})^{-1.096} \times (\text{age})^{-0.294} \times (0.736 \text{ for women})$.

Daily albumin excretion rate was estimated using the urine albumin-to-creatinine ratio (UACR), defined as the ratio of urine albumin concentration to creatinine concentration expressed as mcg of albumin/mg of creatinine. Microalbuminuria was defined as UACR of 30–300 mcg/mg creatinine and macroalbuminuria as >300 mcg/mg creatinine. Hypertension was defined as treatment with hypertensive drugs for an indication of hypertension, the average systolic BP at the GCRC of ≥140 mmHg, or the average diastolic BP at the GCRC of ≥90 mmHg. Median household family annual income (in 1999 dollars) and percent of families below poverty level for a donor's zip code at the time of donation per Census 2000 data was 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 and/or percentages and will be compared using 2×2 contingency tables and chi-squared tests. Correlation between two continuous values was estimated with Pearson correlation coefficient (r). p values <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) as compared to 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 < 60 ml/min vary dramatically depending on the estimation equation utilized. In order to assist in the interpretation of the results, we included in this table data from the study by Ibrahim et al. 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(9). We also included data from an earlier report on a series of 112 University of Minnesota donors that calculated renal clearances using various estimation equations(6).

Among those subjects who had an eGFR(MDRD) < 60 ml/min per 1.73m² at the time of follow-up in the GCRC, the individual 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) < 60 ml/min per 1.73m² was higher than for those with eGFR(MDRD) ≥ 60 ml/min per 1.73m² (35.0 ± 4.5 versus 27.5 ± 5.18 kg/m², respectively; p-value = 0.001). The mean eGFR(MDRD) was lower for those with BMI ≥ 35 kg/m² as compared to those with BMI < 35 kg/m² (61.4 ± 16.3 versus 74.9 ± 15.4 ml/min per 1.73m², respectively; p-value = 0.034). Four of 8 subjects (50%) with BMI ≥ 35 kg/m² and 3 of 31 subjects (9.7%) with a BMI < 35 kg/m² had an eGFR(MDRD) < 60 ml/min per 1.73m² (p-value = 0.008).

Among the 37 subjects who had pre-donation renal function data available in our database, the follow-up eGFR(MDRD) was 30.5 ± 16.4 ml/min per 1.73m² less than the pre-donation value (p-value < 0.001 on paired t-test), which represents a 28.8% drop in the serial eGFR(MDRD)'s. Subjects whose BMI was ≥ 35 kg/m² were found to have a greater absolute decrement in eGFR(MDRD) than those with BMI < 35 kg/m² (40.1 ± 7.3 and 28.3 ± 17.1 ml/min per 1.73m², 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 as compared to female donors (27.8 ± 17.3 and 32.2 ± 15.9 ml/min per 1.73 m², respectively; p = NS), in donors younger than 30 years old as compared those 30 years old or older (30.9 ± 19.8 and 29.6 ± 14.9 ml/min per 1.73 m², respectively; p = NS), or in hypertensive as compared to non-hypertensive subjects (30.2 ± 15.4 and 30.7 ± 17.3 ml/min per 1.73 m², respectively; p = NS). Likewise, we did not find an association with hypertension and BMI ≥ 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 beta is 3.51 ± 1.6 (95% confidence interval 0.22–6.8) ml/min/1.73m² 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 compare outcomes to those reported in the literature on the general live kidney donor population (most of whom were likely not African American). Prior 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% Caucasian) were used to compare GFR changes post donation (6,9). As can be seen in Table 2, the proportion of subjects with eGFR or creatinine clearance < 60 ml/min varied widely 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 <

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 illustrates the limitations of estimating renal function using serum creatinine in this population. The relative loss of GFR from pre-donation to follow-up several years later of 28% in our group of AALKD's does not appear to be excessive when considering that reports from the literature indicate that the GFR following uni-nephrectomy should settle at approximately 75% of the two-kidney baseline(2,3,⁵,6,8).

In order to assess the risk of donation from African Americans, our AALKD cohort should be compared to 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 to our group. Nevertheless, the microalbuminuria prevalence of 15.4% rate in AALKDs appears reassuringly similar to the NHANES III prevalence of 11% in the general population(22). Although the prevalence of eGFR or creatinine clearance < 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 subjects with eGFR(MDRD) < 60 ml/min per 1.73m² was found to have microalbuminuria, and none had macroalbuminuria. Additionally, 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 and/or due to inaccuracies in using serum creatinine to identify individuals with CKD, rather than indicating threatening renal pathology.

It is important to note that the risk of having eGFR(MDRD) < 60 ml/min per 1.73m² and the magnitude of absolute and relative loss of eGFR(MDRD) were significantly higher in obese (BMI ≥ 35 kg/m²) AALKD's than in non-obese AALKD's. 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 hypertension within the follow-up period. In order to understand the significance of this incidence of hypertension in our cohort of AALKD's, we can compare it to 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 post donation, they found that 24.7% were on antihypertensive medications and that 7.5% had newly diagnosed hypertension. In their study, the mean BP was 122/73, as compared to 121/80 mmHg in our group. Additionally, among the 48 studies included in a recent meta-analysis by Boudville et al.(23) that addressed the risk for hypertension in the general population of living kidney donors, they found a 5 mmHg increase in blood pressure over that anticipated with normal aging and that the incidence of hypertension varied markedly among the studies – from 0 to 62%, with a crude mean of 19%.

More important than whether AALKD's have a higher risk for hypertension than non-African American donors is what the effect of donating will have on blood pressure in AALKD's. This issue is addressed by comparing the rate of hypertension in AALKD's to African Americans who have not donated. Given that no donors had a diagnosis of hypertension 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. Utilizing the

NHANES data (24) the prevalence of hypertension for non-Hispanic black men is approximately 20% in the 30–39 year old age group and 36% in the 40–49 year old age group. The prevalence of hypertension for non-Hispanic black women is approximately 10% in the 30–39 year old age group and 28% in the 40–49 year old age group. Given that the mean age of the donors in our cohort at the time of donation (when none had a hypertension) was 37 years old and the mean age at follow up was 44 years old, 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 hypertension in non-donating African Americans. An incidence rate of 41% over 7.1 years of follow up is clearly higher than the incidence rate that would be expected in the general African American population.

On the other hand, it is certainly plausible that the proportion of study subjects with hypertension may be higher than the proportion in the whole AALKD population, as suggested by findings in the meta-analyses by Boudville et al. that showed that the higher the proportion of donors lost to follow-up in a study, the higher the reported increase in blood pressure(23). Although the true significance of the 41% incidence of hypertension is debatable, this high proportion of AALKD's with hypertension in our cohort is concerning given that hypertension may be more nephrotoxic in African Americans and given that individuals with one kidney have less renal reserve. Of further concern is that half of these individuals did not know that their blood pressure was high. On the other hand, 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 was limited by our reliance on serum creatinine, and inaccuracies of using the commonly used serum creatinine-based estimation equations in individuals without known CKD has been well described(6,20). Clearly all of the renal function estimation techniques that we utilized have important limitations in this population. Still, it should be noted that a recent study by Ibrahim et al. 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(6). 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. that achieved a 14.3% participation in follow-up iohexol GFR measurements(9). 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 pathology 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 hypertension within several years of donation.
2. AALKDs lose a significant proportion of their eGFR(MDRD) after donating a kidney. However, the magnitude of change appears 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 AALKD's. Our findings also indicate that close monitoring of AALKDs for HTN and CKD is clearly warranted.

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Figure 1. eGFR(MDRD)'s of the individual subjects pre-donation and post-donation (at the time of followup in the GCRC), with obese ($\text{BMI} \geq 30 \text{ kg/m}^2$) subjects identified by dashed lines and non-obese individuals identified by solid line. eGFR(MDRD) = estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation.

Figure 2.

Change in eGFR(MDRD) pre- to post-donation 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

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)

Table 2

Baseline characteristics and early renal function in study group, recruited AALKDs who did not participate, and non-recruited AALKDs (non-locally residing). Continuous values presented as mean $\pm$ standard deviation. There were no statistically significant differences in these parameters in Group 2 versus Group 1 and in Group 3 versus Group 1. BMI = body mass index, eGFR(MDRD) = estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation

Characteristic	Group 1: Study participants	Group 2: Invited AALKDs who did not participate	Group 3: Non-invited AALKDs (non-locally residing)
Number of subjects	39	68	92
Age (years) at donation	37.4 $\pm$ 11.0	33.7 $\pm$ 9.9	35.8 $\pm$ 10.0
Male	34.2%	34.8%	40.2%
BMI	28.9 $\pm$ 5.9	29.3 $\pm$ 6.1	28.2 $\pm$ 5.5
Pre-donation serum creatinine	0.94 $\pm$ 0.26	0.92 $\pm$ 0.22	1.04 $\pm$ 0.97
Pre-donation eGFR(MDRD)	102.5 $\pm$ 20.3	102.8 $\pm$ 21.2	102.5 $\pm$ 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 $\pm$ 20.4	\$42.7 $\pm$ 14.4	42.9 $\pm$ 14.8

Table 3

Demographics and outcomes of interest at the time of follow-up evaluation in GCRC. Continuous values presented as mean $\pm$ 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 years post-donation in two series of mostly Caucasian donors done at the University of Minnesota and reported by Ibrahim et al.(6,9). GCRC = General Clinical Research Center, BMI = body mass index, eGFR(MDRD) = estimated glomerular filtration rate calculated using Modification of Diet in Renal Disease equation, $CG-CI_{cr}$ = Cockcroft-Gault creatinine clearance. AASK-eGFR = The African-American Study of Hypertension and Kidney Disease simplified (3-variable) glomerular filtration rate.

	AALKD's in our current study	University of Minnesota cohort of 255 mostly Caucasian live kidney donors(9)
Length of follow up (from donation to GCRC visit)	7.1 $\pm$ 1.6	12.2 $\pm$ 9.2
Age at time of follow up (years)	46.2 $\pm$ 1.6	52.9 $\pm$ 9.9
Mean BMI (kg/m ²) at time of follow up visit	31.1 $\pm$ 6.3 range of 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 $\pm$ 3.7	
Average SBP during GCRC visit (mmHg)	120.8 $\pm$ 14.5 (92–144)	121.8 $\pm$ 14.6
Average DBP during GCRC visit (mmHg)	79.7 $\pm$ 9.3 (62–99)	73 $\pm$ 8.9
SBP $\geq$ 140 or DBP $\geq$ 90 at GCRC visit	10 (25.6%)	14.4%
Diagnosed with hypertension (using antihypertensive drugs) prior to 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 $\pm$ 0.25 (0.8–1.7)	1.1 $\pm$ 0.2
eGFR(MDRD) <60 ml/min per 1.73m ² or macroalbuminuria	8 (20.5%)	
eGFR(MDRD) <60 ml/min per 1.73m ² and macroalbuminuria	0 (0%)	
Diagnosed with diabetes mellitus	1 (2.6%)	3.1%
eGFR(MDRD) (ml/min per 1.73 m ²)	72.1 $\pm$ 16.4 range 42–106 7 (17.9%) < 60	63.7 $\pm$ 11.3

		University of Minnesota cohort of 112 mostly Caucasian live kidney donors(6)
eGFR(MDRD) (ml/min per 1.73 m ²)	72.1 $\pm$ 16.4 range 42–106 7 (17.9%) < 60	65 $\pm$ 13 39.3% < 60

		University of Minnesota cohort of 112 mostly Caucasian live kidney donors(6)
MC-eGFR (ml/min per 1.73 m ²)	78.8 +/- 19.4 range 42–111 17.9% (n=7) < 60	86 +/- 17 6.2% < 60
Actual CG-ClCr (ml/min per 1.73 m ²)	75.1 +/- 19.5 range 45–129 23.1% (n=9) <60	75 +/- 17 22.3% <60
Lean CG-ClCr (ml/min per 1.73 m ²)	52.7 +/- 14.1 range 25–80 69.2% (n=27) <60, 10.3% (n=4) <30	
AASK-eGFR (ml/min per 1.73 m ²)	74.4 +/- 16.1 range 43–107 6 (15.4%) < 60	