Risk of Hyperkalemia in Nondiabetic Patients With Chronic Kidney Disease Receiving Antihypertensive Therapy

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Background: The incidence and factors associated with hyperkalemia in patients with chronic kidney disease (CKD) treated with angiotensin converting enzyme inhibitors (ACEIs) and other antihypertensive drugs was investigated using the African American Study of Kidney Disease and Hypertension (AASK) database.

Methods: A total of 1094 nondiabetic adults with hypertensive CKD (glomerular filtration rate [GFR], 20-65 mL/min/1.73 m²) were followed for 3.0 to 6.4 years in the AASK trial. Participants were randomly assigned to ACEI, β-blocker (BB), or dihydropyridine calcium channel blocker (CCB). The outcome variables for this analysis were a serum potassium level higher than 5.5 mEq/L (to convert to millimoles per liter, multiply by 1.0), or a clinical center initiated hyperkalemia stop point.

Results: A total of 6497 potassium measurements were obtained, and 80 events in 51 subjects were identified (76 events driven by a central laboratory result and 4 driven by a clinical center–initiated hyperkalemia stop point). Compared with a GFR higher than 50 mL/min/1.73 m², after multivariable adjustment, the hazard ratio (HR) for hyperkalemia in patients with a GFR between 31 and 40 mL/min/1.73 m² and a GFR lower than 30 mL/min/1.73 m² was 3.61 (95% confidence interval [CI], 1.42-9.18 [P=.007]) and 6.81 (95% CI, 2.67-17.35 [P<.001]), respectively; there was no increased risk of hyperkalemia if GFR was 41 to 50 mL/min/1.73 m². Use of ACEIs was associated with more episodes of hyperkalemia compared with CCB use (HR, 7.00; 95% CI, 2.29-21.39 [P<.001]) and BB group (HR, 2.85; 95% CI, 1.50-5.42 [P=.001]). Diuretic use was associated with a 59% decreased risk of hyperkalemia.

Conclusions: In nondiabetic patients with hypertensive CKD treated with ACEIs, the risk of hyperkalemia is small, particularly if baseline and follow-up GFR is higher than 40 mL/min/1.73 m². Including a diuretic in the regimen may markedly reduce risk of hyperkalemia.

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Several studies have demonstrated that angiotensin-converting enzyme inhibitors (ACEIs) blunt progression of renal disease in nondiabetic patients with chronic kidney disease (CKD).1-4 However, ACEIs can cause hyperkalemia by impairing renal potassium excretion through interference with production and/or secretion of aldosterone.5 Hyperkalemia from ACEI use has been frequently described,6 and ACEIs are often underprescribed in patients with CKD because of concerns of hyperkalemia.6,9 β-Blocker (BB) use has also been associated with hyperkalemia, most likely through redistribution of potassium from intracellular to extracellular compartments as a result of blockade of β₂-adrenoreceptor–mediated cellular potassium uptake.10,11 The African American Study of Kidney Disease and Hypertension (AASK) was a randomized clinical trial in nondiabetic African Americans with hypertensive CKD. One primary goal was to determine the effects of 3 different classes of antihypertensive agents on progression of renal disease: a dihydropyridine calcium channel blocker (CCB), a BB, and an ACEI. The most beneficial drug therapy was with ACEIs.12 The other primary goal was to determine the effects of 2 different BP goals on progression of renal disease; the trial demonstrated that a target mean arterial BP (MAP) of 102 to 107 mm Hg was as effective as stricter BP goal of a MAP lower than 92 mm Hg.13 Participants had a glomerular filtration rate (GFR) between 20 and 65 mL/min/1.73 m² and no identified causes of renal insufficiency other than hypertension. After the close of the trial phase of the AASK, the investigators were directed by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) appointed Data

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Group Information: Members of the African American Study of Hypertension and Kidney Disease Collaborative Research Group are listed on page 1593.
Safety Monitoring Board to use the AASK database to explore factors associated with development of hyperkalemia. In this report, we describe the incidence of hyperkalemia by class of antihypertensive drug in the AASK and report the independent associations of other clinically measured factors.

### METHODS

#### TRIAL DESIGN

The design of the AASK study, including complete eligibility and exclusion criteria, has been described elsewhere. The AASK study was a 21-center, NIDDK-sponsored study that randomized 1094 patients. In a 3 × 2 factorial design, patients were randomized to initial treatment with either a β-blocker (metoprolol succinate extended release, 50-200 mg/d) or a CCB (amlodipine besylate, 5-10 mg/d), or a CCB (amlodipine besylate, 5-10 mg/d) and to 1 of 2 nonsteroidal anti-inflammatory agents (NSAIDs) more than 15 d/mo, except for aspirin, or inability to discontinue NSAIDs or aspirin for 3 days prior to GFR measurement; and (9) locally measured potassium level higher than 5.5 mEq/L during screening.

Each individual institutional review board of the participating institutions approved the study protocol, and written informed consent was obtained for all subjects before enrollment in the trial.

#### VARIABLE DEFINITIONS

Hyperkalemia was defined as the occurrence of a centrally measured (Cleveland Clinic Foundation, Cleveland, Ohio) potassium concentration higher than 5.5 mEq/L at one of the follow-up visits (at months 3, 6, and 12 and at 6-month intervals thereafter throughout the follow-up period until the patient's final serum potassium measurement prior to death) or a clinical center–initiated, hyperkalemia-related stop point. Patients were followed up until the occurrence of end-stage renal disease (ESRD) or, if taking a CCB, until September 22, 2000, when the CCB arm was terminated during trial. Baseline factors evaluated for association with the first episode of hyperkalemia included age, sex, weight, body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared), systolic BP, diastolic BP, MAP, creatinine level, UP/Cr, randomization drug, baseline NSAID use, baseline serum glucose level, and baseline potassium level. Body mass index values were grouped into the following categories: 25 or lower, higher than 25 to 30 or lower, and higher than 30. Because earlier studies reported increased incidence of elevated potassium level, and baseline potassium level. Body mass index values were grouped into the following categories: 25 or lower, higher than 25 to 30 or lower, and higher than 30. Because earlier studies reported increased incidence of elevated potassium level.
STATISTICAL ANALYSES

Baseline characteristics were summarized by standard descriptive statistics (means and standard deviations or frequencies and percentages, as appropriate). Event rates for hyperkalemia events, defined as the first occurrence of a follow-up serum potassium measurement higher than 5.5 mEq/L or a clinical center–initiated, hyperkalemia-related stop point, were computed as the ratio of the number of events to the total patient-years of follow-up and expressed as the number of events per 100 patient-years. Exact 95% confidence intervals (CIs) for event rates were calculated based on the Poisson distribution. The association between hyperkalemia and randomized treatment group was assessed by using a discrete-time proportional hazards regression model with a complementary log-log link function to relate the probability of first occurrence of hyperkalemia to a patient’s most recent potassium measurement to the randomized treatment assignment.\textsuperscript{17} Discrete-time proportional hazards regression is analogous to proportional hazards Cox regression in continuous time but accounts for the fact that hyperkalemia could only be observed at visits with a serum potassium measurement. Additional discrete-time proportional hazards models were used to relate the hazard of hyperkalemia to the individual baseline risk factors designated in the previous subsection, controlling only for randomized treatment assignment, and to jointly relate the hazard for hyperkalemia to each of the baseline risk factors and randomized treatment assignment in a multivariable analysis. To determine if the effects of the randomized treatment assignments differed by baseline GFR or BMI levels, interaction tests were performed between randomized groups and baseline GFR and BMI, respectively, treating both baseline factors as continuous variables. Finally, a time-dependent discrete time proportional hazards regression was performed to jointly relate the probability of hyperkalemia to a patient’s most recent potassium measurement, diuretic use, GFR, and UP/Cr, controlling for randomization to low vs usual BP goal (Table 3). The HR for the BB vs CCB comparison was 2.45 (95% CI, 0.79–7.65) after adjustment for the baseline covariates but did not attain statistical significance (P = .12). There were no significant differences in the rate of hyperkalemia between dose levels of ACEI, although the power to detect a difference, if it existed, was low because subjects randomized to ACEI were taking 10 mg/d on at least 68.8% (range, 68.8%–76.3%) of their visits during the trial. There was no significant difference in rate of hyperkalemia according to randomization to low vs usual BP goal (Table 3).

Figure 1 displays the rates and 95% CIs of first occurrences of hyperkalemic events by assigned randomized drug plotted against varying levels of GFR. The figure demonstrates that there was a negligible risk for a hyperkalemic event in each drug group if the baseline GFR was higher than 40 mL/min/1.73 m\(^2\).

Body mass index was independently associated with hyperkalemia. A total of 9.8% of patients whose BMI at baseline was 25 or lower experienced hyperkalemic events compared with 3.6% of patients with a baseline BMI higher than 25. The lower BMI category was associated with an increased hazard for hyperkalemia compared with 25 to 30 BMI group in both univariate and multivariable analyses (Table 3). Figure 2 displays the rates of hyperkalemic events by randomized drug assignment plotted against varying levels of BMI. Especially notable is the relatively greater rate of hyperkalemia in subjects with low BMI who were assigned to either the ACEI or BB groups. Figure 3 presents the rates of hyperkalemic events by BMI category plotted against GFR category. As shown, subjects in the lowest BMI and GFR categories had the greatest risk for a hyperkalemic event.

In univariate analysis, higher baseline levels of the UP/Cr were associated with increased risk of hyperkalemic events throughout the range of this variable (Table 3). However, in multivariable analysis, only those subjects with the greatest amount of protein excretion exhibited a significantly elevated risk of hyperkalemia. Baseline NSAID use was not found to be a significant pre-
dictor for hyperkalemia, but only 10.9% of the patients were taking NSAIDs at baseline (Table 1).

Table 4 presents results jointly relating the probability of hyperkalemia to the most recent recorded GFR, UP/Cr, diuretic use, and potassium level from the previous visit. Similar to the result observed for baseline GFR and potassium level, the most recent GFR, at both 30 mL/min/1.73 m² or lower and between 30 and 40 mL/min/1.73 m², was a significant predictor of a hyperkalemia event compared with the reference category of a GFR higher than 50 mL/min/1.73 m². For most recent potassium measurement, the categories of higher than 5 mEq/L and between 4 and 5 mEq/L were a significant predictor of a hyperkalemia event compared with lower than 4 mEq/L. In contrast to analyses of baseline covariates, the most recent UP/Cr was not a significant predictor of hypokalemia after controlling for the most recent GFR. During the trial, diuretics were used for an average of 75% of follow-up visits. After controlling for the most recent GFR, use of diuretics was associated with a reduction in the probability of hyperkalemia by 59% ($P = .006$).

Figure 4 presents the distribution of levels of serum potassium at the time of the first occurrence of the potassium level–defined hyperkalemic events. Most hyperkalemic events fell into the category of 5.6 to 5.8 mEq/L for all of the 3 drug classes. No potassium level higher than 5.8 mEq/L was observed in the CCB group. Only 3 of the hyperkalemic events in the ACEI group were associated with a potassium level higher than 6.2 mEq/L, and only 4 of the hyperkalemic events in the BB group were associated with a potassium level higher than 6.2 mEq/L. Table 5 presents the number of patients with at least 1 serum potassium measurement higher than 5.5 or 6.0 mEq/L, as well as the time from randomization until the first visit at which a serum potassium level was higher than 6.0 mEq/L, stratified by GFR, BMI, and ACEI therapy. Of note, a serum potassium level higher than 6.0 mEq/L did not occur until more than 14 months of ACEI treatment in participants with a GFR higher than 40 mL/min/1.73 m².

**COMMENT**

This study identifies readily measurable baseline and follow-up clinical variables that are associated with hyperkalemic events in nondiabetic African American patients with hypertensive CKD who are treated with commonly used classes of antihypertensive drug therapy. As expected, ACEI use was associated with significantly more episodes of hyperkalemia compared with CCBs and more events than BBs. Older age, baseline protein excretion, and both baseline and follow-up GFR and potassium levels were independent risk factors for develop-
The association of hyperkalemia with lower GFR and renal dysfunction is consistent with the literature. In a population that included patients with diabetes and in which hyperkalemia was defined as a serum potassium level higher than 5.1 mEq/L, risk factors for an event included a creatinine level higher than 1.5 mg/dL (to convert to micromoles per liter, multiply by 88.4). Also, during captopril treatment, transient elevations in potassium level higher than 6.0 mEq/L have been inversely related to GFR in markedly azotemic subjects. Our results extend these previous findings by demonstrating that this effect is independent of baseline randomized drug, age at randomization, sex, NSAID use, BMI, baseline U/P/Cr, and baseline potassium level. In the patient population we studied, there is a clear increase in events in those with a GFR between 20 and 30 mL/min/1.73 m² (ie, late stage 3 and stage 4 kidney disease). A limitation of the study is that the number of events in the group with a GFR lower than 20 mL/min/1.73 m² is most likely an underestimation, as laboratory results were no longer collected.

Table 3. Association of Risk of Hyperkalemia With Baseline Factors

<table>
<thead>
<tr>
<th>Variable</th>
<th>Randomized Group Comparisons</th>
<th>HR (95% CI)</th>
<th>P Value</th>
<th>Univariate Analysis</th>
<th>HR (95% CI)</th>
<th>P Value</th>
<th>Multivariable Analyses</th>
<th>HR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BB vs CCB</td>
<td>2.07 (0.70-6.16)</td>
<td>.19</td>
<td>NA</td>
<td>NA</td>
<td>2.45 (0.79-7.65)</td>
<td>.12</td>
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<tr>
<td>ACEI vs CCB</td>
<td>3.84 (1.35-10.89)</td>
<td>.01</td>
<td>NA</td>
<td>NA</td>
<td>7.00 (2.29-21.39)</td>
<td>&lt;.001</td>
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<tr>
<td>ACEI vs BB</td>
<td>1.85 (1.02-3.36)</td>
<td>.04</td>
<td>NA</td>
<td>NA</td>
<td>2.85 (1.50-5.42)</td>
<td>.001</td>
<td></td>
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<tr>
<td>Low vs usual BP</td>
<td>1.10 (0.64-1.91)</td>
<td>.72</td>
<td>NA</td>
<td>NA</td>
<td>1.28 (0.72-2.29)</td>
<td>.40</td>
<td></td>
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<tr>
<td>Age at randomization, 10 y</td>
<td>1.26 (0.95-1.67)</td>
<td>.10</td>
<td>1.40 (1.05-1.88)</td>
<td>.02</td>
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<tr>
<td>Female sex</td>
<td>0.85 (0.48-1.53)</td>
<td>.60</td>
<td>0.52 (0.28-0.98)</td>
<td>.04</td>
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<tr>
<td>Baseline NSAID use</td>
<td>1.02 (0.43-2.38)</td>
<td>.97</td>
<td>0.93 (0.39-2.32)</td>
<td>.88</td>
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<tr>
<td>Mean baseline GFR ≤30 vs &gt;50 mL/min/1.73 m²</td>
<td>13.09 (5.83-29.39)</td>
<td>.001</td>
<td>6.81 (2.67-17.35)</td>
<td>&lt;.001</td>
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<tr>
<td>Mean baseline GFR 31-40 vs &gt;50 mL/min/1.73 m²</td>
<td>5.44 (2.30-12.85)</td>
<td>&lt;.001</td>
<td>3.61 (1.42-9.18)</td>
<td>.007</td>
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<tr>
<td>Mean baseline GFR 41-50 vs &gt;50 mL/min/1.73 m²</td>
<td>0.85 (0.23-3.22)</td>
<td>.82</td>
<td>0.61 (0.16-2.35)</td>
<td>.47</td>
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<tr>
<td>BMI &gt;25 vs ≤25 kg/m²</td>
<td>2.68 (1.36-5.29)</td>
<td>.004</td>
<td>1.92 (0.95-3.89)</td>
<td>.07</td>
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<tr>
<td>BMI &gt;30 vs ≤25 kg/m²</td>
<td>0.77 (0.38-1.59)</td>
<td>.48</td>
<td>0.82 (0.39-1.74)</td>
<td>.61</td>
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<tr>
<td>Baseline U/P/Cr 0.08-0.22 vs ≥0.08</td>
<td>2.70 (1.23-5.93)</td>
<td>.01</td>
<td>2.27 (0.96-5.36)</td>
<td>.06</td>
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<tr>
<td>Baseline U/P/Cr 0.22-0.66 vs ≥0.08</td>
<td>1.83 (0.73-4.60)</td>
<td>.20</td>
<td>1.15 (0.42-3.14)</td>
<td>.78</td>
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<tr>
<td>Baseline U/P/Cr &gt;0.66 vs ≥0.08</td>
<td>5.86 (2.88-11.92)</td>
<td>&lt;.001</td>
<td>3.63 (1.58-8.34)</td>
<td>.002</td>
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<tr>
<td>Baseline glucose level 100-115 vs &lt;100 mg/dL</td>
<td>0.63 (0.30-1.35)</td>
<td>.24</td>
<td>1.06 (0.50-2.26)</td>
<td>.84</td>
<td></td>
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<tr>
<td>Baseline glucose level &gt;115 vs &lt;100 mg/dL</td>
<td>1.19 (0.52-2.68)</td>
<td>.66</td>
<td>2.04 (0.91-4.58)</td>
<td>.10</td>
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<tr>
<td>Baseline potassium level 4-5 vs &lt;4 mEq/L</td>
<td>18.48 (2.53-135.10)</td>
<td>.004</td>
<td>14.81 (2.01-109.10)</td>
<td>.008</td>
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</tr>
<tr>
<td>Baseline potassium level &gt;5 vs ≤4 mEq/L</td>
<td>85.54 (11.36-644.20)</td>
<td>&lt;.001</td>
<td>53.72 (6.97-414.20)</td>
<td>&lt;.001</td>
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</tbody>
</table>

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; BB, β-blocker; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); BP, blood pressure; CCB, calcium channel blocker; CI, confidence interval; GFR, glomerular filtration rate; HR, hazard ratio; NA, not applicable; NSAID, nonsteroidal anti-inflammatory drug; U/P/Cr, urinary protein to creatinine ratio.

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perkalemia, we considered the possibility that investiga-
tors used less diuretics in those with a lower GFR; we did not find evidence for this behavior. Consistent with previous
reports in the literature, age was also an independent
risk factor for hyperkalemia. The potential of ACEIs
to increase occurrence of hyperkalemia in elderly patients
was described over a decade ago. Use of NSAIDs has been
associated with hyperkalemia, but we did not observe this
association. However, only a small subset of patients were
using NSAIDs because prior to randomization we elimi-
nated patients with an inability to discontinue or a re-
ported excessive NSAID use.

Several factors not measured in our study have been shown
to increase the risk of hyperkalemia in various
populations treated with ACEIs. Howes et al suggest that
other predisposing factors include autonomic neuropathy
and adrenal insufficiency. When captopril was admin-
istered to 23 patients by Atlas et al, those with high
plasma renin activity experienced the greatest effects on
aldosterone secretion and potassium balance, as well as
the greatest reductions in BP.

We recognize several limitations of our study. Keilani
et al showed that in patients with mild CKD, low-dose
(1.25 mg/d by mouth) ramipril did not alter potassium
level, but high-dose (10 mg/d by mouth) ramipril re-
sulted in an increase in potassium level from 4.53 to 4.78
mEq/L. We did not find a significant difference in the
rate of hyperkalemia between dose levels of ACEI, but
the power to detect a difference was limited because more
than two-thirds of our patients were receiving high-
dose ACEI. Next, we recognize that our result may not
be generalizable to all drugs in the classes we studied.

Figure 4. Distribution of serum potassium levels at the occurrence
of incident hyperkalemia events. To convert potassium to millimoles per liter,
multiply by 1.0. ACEI indicates angiotensin-converting enzyme inhibitor; BB,
β-blocker; CCB, dihydropyridine calcium channel blocker.

Table 4. Association of Risk of Hyperkalemia With
Time-Dependent Factors in Multivariable Analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hazard Ratio (95% Confidence Interval)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up diuretic use</td>
<td>0.41 (0.22-0.78)</td>
<td>.006</td>
</tr>
<tr>
<td>Follow-up GFR &lt;30 vs &gt;50 mL/min/1.73 m²</td>
<td>9.07 (3.18-25.88)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Follow-up GFR &gt;30 to ≤40 vs &gt;50 mL/min/1.73 m²</td>
<td>3.67 (1.21-11.15)</td>
<td>.02</td>
</tr>
<tr>
<td>Follow-up GFR &gt;40 to ≤50 vs &gt;50 mL/min/1.73 m²</td>
<td>1.98 (0.59-6.61)</td>
<td>.27</td>
</tr>
<tr>
<td>Follow-up UP/Cr &gt;0.08 vs ≤0.22</td>
<td>2.01 (0.92-4.39)</td>
<td>.08</td>
</tr>
<tr>
<td>vs ≤0.08</td>
<td>1.50 (0.62-3.63)</td>
<td>.37</td>
</tr>
<tr>
<td>Follow-up UP/Cr &gt;0.22 to ≤0.66</td>
<td>1.84 (0.78-4.30)</td>
<td>.16</td>
</tr>
<tr>
<td>vs ≤0.08</td>
<td>7.25 (1.72-30.58)</td>
<td>.007</td>
</tr>
<tr>
<td>Follow-up potassium level 4-5</td>
<td>2.01 (0.59-6.61)</td>
<td>.27</td>
</tr>
<tr>
<td>vs ≤0.08</td>
<td>30.83 (6.89-138.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Follow-up potassium level &gt;5</td>
<td></td>
<td></td>
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<tr>
<td>vs ≤0.08</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: GFR, glomerular filtration rate; UP/Cr, urinary protein to
creatinine ratio.

SI conversion factor: To convert potassium to millimoles per liter, multiply
by 1.0.

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In conclusion, in the setting of non-diabetic, hypertensive CKD, the risk of hyperkalemia is inversely related to GFR and BMI, regardless of antihypertensive treatment.

Table 5. Time From Randomization Until First Serum Potassium Measurement Higher Than 6.0 mEq/L, Stratified by GFR, BMI, and ACEI Therapy

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of Patients With at Least 1 Follow-up Serum Potassium Measurement &gt;5.5 mEq/L</th>
<th>No. of Patients With at Least 1 Serum Potassium Measurement &gt;6.0 mEq/L</th>
<th>No. of Patients With Antihypertensive Therapy</th>
<th>Time (mo) From Randomization Until First Recorded Serum Potassium Measurement &gt;6.0 mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>1094</td>
<td>1053</td>
<td>50</td>
<td>15</td>
</tr>
<tr>
<td>All ACEI-treated patients</td>
<td>436</td>
<td>417</td>
<td>29</td>
<td>8</td>
</tr>
<tr>
<td>All ACEI-treated patients with a GFR ≤40</td>
<td>148</td>
<td>139</td>
<td>22</td>
<td>7</td>
</tr>
<tr>
<td>All ACEI-treated patients with a GFR &gt;40</td>
<td>288</td>
<td>278</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>All ACEI-treated patients with a BMI ≤25</td>
<td>80</td>
<td>75</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>All ACEI-treated patients with a BMI &gt;25</td>
<td>356</td>
<td>342</td>
<td>19</td>
<td>4</td>
</tr>
</tbody>
</table>

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); GFR, glomerular filtration rate.

SI conversion factor: To convert potassium to millimoles per liter, multiply by 1.0.

African American Study of Hypertension and Kidney Disease Collaborative Research Group

and during treatment is higher than 40 mL/min/1.73 m², routine monitoring of serum potassium level is sufficient, even in those treated with ACEIs. Conversely, the results of this study would justify more frequent monitoring of serum potassium in the following subcategories: (1) patients with a GFR of 40 mL/min/1.73 m² or lower who have a low BMI and are receiving treatment with an ACEI; (2) patients with a GFR of 30 mL/min/1.73 m² or lower, irrespective of treatment, but especially with a BMI of 25 or lower; (3) older patients; (4) those with higher levels of microalbuminuria; and (5) those patients in whom a diuretic is not part of the medication regimen.

In conclusion, in the setting of non-diabetic, hypertensive CKD, the risk of hyperkalemia is inversely related to GFR and BMI, regardless of antihypertensive treat-
ment. After initiation of antihypertensive therapy, the risk of hyperkalemia is greatest with ACEI use, intermediate with BB use, and lowest with CCB use. Including a diuretic as part of the medication regimen may markedly reduce the risk of hyperkalemia.

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Error in Abstract. In the article titled “Risk of Hyperkalemia in Nondiabetic Patients With Chronic Kidney Disease Receiving Antihypertensive Therapy” by Weinberg et al, published in the September 28 issue of the Archives (2009;169[17]:1587-1594), an error occurred in the second sentence of the “Results” section of the Abstract. The corrected sentence should read as follows: “Compared with a GFR higher than 50 mL/min/1.73 m², after multivariable adjustment, the hazard ratio (HR) for hyperkalemia in patients with a GFR between 31 and 40 mL/min/1.73 m² and a GFR lower than 30 mL/min/1.73 m² was 3.61 (95% confidence interval [CI], 1.42-9.18 [P=.007]) and 6.81 (95% CI, 2.67-17.35 [P < .001]), respectively; there was no increased risk of hyperkalemia if GFR was 41 to 50 mL/min/1.73 m².”