Prevalence and Correlates of Left Ventricular Hypertrophy in the African American Study of Kidney Disease Cohort Study

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Abstract—African Americans with hypertensive renal disease represent a high-risk population for cardiovascular events. Although left ventricular hypertrophy is a strong predictor of adverse cardiac outcome, the prevalence and associated factors of left ventricular hypertrophy in this patient population are not well described. The African American Study of Kidney Disease Cohort Study is a prospective, observational study that is an extension of the African American Study of Kidney Disease randomized clinical trial that was conducted from 1994 to 2001 in African Americans with hypertension and mild-to-moderate renal dysfunction. Echocardiograms and 24-hour ambulatory blood pressure monitoring were performed at the baseline visit of the cohort. Of 691 patients enrolled in the cohort study, 599 patients had interpretable baseline echocardiograms and ambulatory blood pressure data. Left ventricular hypertrophy was defined using a cut point for left ventricular mass index >49.2 g/m² in men and >46.7 m²/m² in women. The majority of patients had left ventricular hypertrophy (66.7% of men and 73.9% of women). In a multiple regression analysis, higher average day and nighttime systolic blood pressure, younger age, and lower predicted glomerular filtration rate were associated with left ventricular hypertrophy, but albuminuria was not. These data demonstrate a striking prevalence of left ventricular hypertrophy in the African American Study of Kidney Disease Cohort and identify potential targets for prevention and therapeutic intervention in this high-risk patient population. (Hypertension. 2007;50:1033-1039.)

Key Words: hypertension ■ hypertrophy ■ left ventricular ■ echocardiography ■ African Americans ■ kidney failure ■ chronic ■ blood pressure monitoring ■ ambulatory

African Americans with hypertension are at increased risk of cardiovascular morbidity and mortality and have more frequent progression to end-stage renal disease compared with other ethnic groups in the United States.1–5 In addition, those patients who develop chronic kidney disease are at even greater risk for cardiovascular mortality.6 Why African Americans are a particularly high-risk group is not fully understood. Left ventricular hypertrophy (LVH) is a strong independent predictor of adverse cardiovascular events, and several studies have found a higher prevalence for LVH in African Americans compared with whites.7–9

The African American Study of Kidney disease (AASK) cohort involves African American patients with nondiabetic, hypertensive renal disease. The prevalence and associated factors of target-organ damage, such as LVH, in this patient population have not been well described previously. In addition, echocardiographic data in this patient population are virtually nonexistent; to our knowledge only 1 other study has specifically assessed echocardiographic findings in a small number of African Americans with hypertensive renal disease.10 Because this population is at high risk for cardiovascular events, the AASK cohort is an ideal group of patients in which to evaluate to identify the frequency and correlates of LVH to help target those patients who may benefit from aggressive therapy for risk reduction.

Methods

Study Population

The AASK Cohort Study is a prospective, observational study that is an extension of the AASK, which tested the effects of 3 medications used as initial antihypertensive therapy (ramipril, metoprolol, and amlopidine) and usual (mean arterial pressure of 102 to 107 mm Hg)
or lower (mean arterial pressure <93 mm Hg) blood pressure (BP) control. Eligible patients enrolled in the AASK were African American men and women with hypertension, between the ages of 18 and 70, diastolic BP of ≥95 mm Hg, with a glomerular filtration rate (GFR) of 20 to 65 mL/min per 1.73 m² and no apparent cause of renal insufficiency other than hypertension. The clinical composite outcome was reduction in GFR by 50% from baseline, the development of end-stage renal disease, or death. During the trial, mean BP was 128/89 mm Hg (±12/8 mm Hg) in the lower BP group and 141/85 mm Hg (±12/7 mm Hg) in the usual BP group. There was no difference in the progression of kidney disease or in the clinical composite outcome compared with the metoprolol and amiodipine groups. Based on the results, in the absence of contraindications, patients in the cohort phase were treated with an angiotensin-converting enzyme inhibitor.

All participants in the AASK who had not died or reached end-stage renal disease were invited to enroll in the cohort study. The study was approved by the institutional review committee at each participating site and adhered to the principles of the Declaration of Helsinki and Title 45, US Code of Federal Regulations, Part 46, Protection of Human Subjects. All of the subjects gave informed consent, and procedures followed were in accordance with institutional guidelines. Patients enrolled in the AASK Cohort Study had excellent BP control during the trial. The average mean BP of cohort participants during the trial was 98 mm Hg (±8 mm Hg; 93 ±7 mm Hg in the lower BP group and 104 ±5 mm Hg in the usual BP group). Details of the study design and inclusion/exclusion criteria have been reported elsewhere.[1]

**Echocardiography**

Patients enrolled in the cohort had an echocardiogram performed at their baseline visit. M-mode, 2D, and Doppler echocardiography examinations were performed at each of the 21 enrolling centers using commercially available equipment. To ensure data quality, echocardiograms were obtained by trained technologists using a standardized protocol and read centrally at the AASK Cardiovascular Core Laboratory. At the core laboratory, echocardiographic tracings were coded, digitized, and interpreted blindly using the DigiView Digital/Video System(s), Model DV-DVRS-100 (Digisonics, Inc). An average of 3 measurements was made of each variable. The end-diastolic (LVIDd) and end-systolic LV internal diameters (LVIDs), interventricular septal thickness, and the posterior wall thickness (PWT) were measured from 2D guided M-mode tracings, according to the American Society of Echocardiography guidelines.[12] When optimal orientation was not possible, measurements were made using 2D views.[13] Left ventricular mass (LVM) was calculated using an anatomically validated formula[14] and normalized to allometric height.[15] LVH as defined according to gender-specific criteria as LVM index >49.2 g/m² in men and >46.7 g/m² in women.

**Methods**

Relative wall thickness (RWT) was calculated using the formula (2 PWT)/LVIDd. A concentric remodeling pattern was defined as an RWT ≥ 0.40. LV sphericity index was calculated by endocardial fractional shortening [(LVIDd−LVIDs)/LVIDd]. Echocardiograms were read by 1 of 5 echocardiographers. Intraclass correlation statistics showed good agreement between measurements (M-mode LVM: 0.72; mitral E wave to A wave ratio: 0.93). Likewise, there was good intrarater variability (eg, mean difference in M-mode LVM was 17.35 g [SD: 27.81], and mean difference of mitral E/A ratio was 0.03 [SD: 0.08]).

**BP Measurements**

Office BP was measured in a standardized method by trained and certified observers using the Tycox Classic handheld Aneroid device. Two BP measurements were obtained at the seated position and 1 in the upright position. Patients were encouraged to have their BP managed by the AASK cohort staff based as outlined previously.[11]

Ambulatory BP monitoring (ABPM) was performed over a 24-hour period, using the SpaceLabs 90217 Ultralite or SpaceLabs 90207 devices. During each 24-hour recording, measurements were obtained every 30 minutes from which awake and asleep averages were calculated, along with dipping status. Nighttime BP was defined as 12:00 AM to 6:00 AM. A blunted BP at night, the so-called nondipping pattern, was defined as ≤10% fall in average systolic BP (SBP).

**Other Measurements**

Urinary albumin excretion was determined by measuring 24-hour albumin and creatinine levels. A urine albumin: creatinine ratio of >0.22 was used to define albuminuria.

Estimated GFR (eGFR) was calculated from serum creatinine using an equation developed from baseline data in the AASK trial.[17] Specifically, eGFR = 392 × (serum creatinine)^(−1.094) × (age)^−0.283 × (0.742 for women).

**Statistical Analysis**

Baseline characteristics were summarized as means and SDs for continuous variables and as frequencies and percentages for categorical variables and were compared between those with and without LVH using a 2-sample t test or χ² test, as appropriate. Multiple linear regression analysis was used to relate LVM to the following prespecified predictor variables: average daytime SBP, average nighttime SBP, eGFR, age, hematocrit (%), gender, and the log-transformed serum albumin:creatinine ratio. Results of all of the statistical tests are reported as 2-sided P values, without adjustment for multiple comparisons.

**Results**

**Study Participants**

Of the 1094 participants in the AASK, 787 patients were alive and not on dialysis, and 691 individuals agreed to enrollment in the cohort study. Baseline echocardiograms were obtained in 649 patients, of whom 41 lacked ABPM data and 8 lacked measurable LVM. Therefore, 599 patients were evaluated for this report.

Tables 1 to 3 present characteristics of participants overall and stratified by LVH. Overall, the mean age of participants was 60.1 ± 10.1 years, and 38.4% were women. Average body mass index was 31.2 ± 6.98 kg/m². The mean hematocrit was 38.4 ± 5.3%. eGFR averaged 44.2 ± 16.5 mL/min per 1.73 m², and average serum creatinine was 2.25 ± 1.35 mg/dL. Albuminuria was present in 12.6% of patients. Additional characteristics are shown in Table S1 (please see http://hyper.ahajournals.org).

BP measurements are displayed in Table 2. Average clinic BP was 135 ± 21.5/80 ± 12.4 mm Hg. Average ambulatory daytime SBP was 138 ± 17 mm Hg, and average nighttime SBP was 134 ± 21 mm Hg. Average 24-hour pulse pressure was 56.1 ± 12.5 mm Hg.

Echocardiographic characteristics are shown in Table 3. The majority of patients had normal left ventricular systolic function as measured by fractional shortening (average: 38 ± 10%). A high proportion of patients had evidence for diastolic dysfunction, with a median E wave to A wave ratio of 0.92 ± 0.38. Left atrial size was enlarged (mean area: 19.2 ± 5.12 cm²). Concentric remodeling was common in this population (mean RWT: 0.53 ± 0.13). Of the patient population, 69.4% had LVH (66.7% of men and 73.9% of women). The mean LVM index was 60.9 ± 21.6 g/m² overall; 60.5 ± 21.0 g/m² in men, and 61.7 ± 22.5 g/m² in women.
Factors Associated With LVH

Variables predictive of LVH were younger age, greater body mass index, the presence of albuminuria, higher average day and average nighttime SBP, and higher 24-hour pulse pressure. Table 2 shows the correlations between LVH and clinic and ABPM values. Average 24-hour SBP, average day SBP, and average night SBP were all highly predictive of LVH. The difference between daytime and nighttime BP was not significantly associated with LVH.

Echocardiographic characteristics associated with the presence of LVH (Table 3) include greater internal diastolic and systolic dimensions, greater wall thicknesses, larger aortic root diameter and left atrial size, higher RWT, and higher Doppler stroke volume. Patients with LVH had lower heart rates but a similar cardiac index and fractional shortening.

The Figure shows the joint relationship of daytime and nighttime BP by quartiles with mean LVM. Independent relationships were observed between the degree of LVH and progressive increases in both BP indices as fixed levels of the other index.

Multivariable Associations

On multiple regression analysis (adjusted $R^2=0.10$), greater average nighttime and daytime SBP, lower estimated eGFR, and younger age were independent correlates of LVH (Table 4). Hematocrit and the presence of microalbuminuria were not independently associated with LVH.

Discussion

This study represents the first comprehensive systematic examination of the prevalence and correlates of LVH in African American patients with hypertensive kidney dysfunction, a population at very high risk for cardiovascular disease. Despite adequate BP control over 5 years before evaluation, there was an extraordinarily high prevalence of LVH in the AASK cohort. In most series, only $\approx 30\%$ of patients with hypertension have LVH$^{18}$ compared with $\approx 70\%$ in the AASK Cohort Study.

Our findings point to both direct and potentially indirect effects of BP on LVH. In particular, ABPM values were highly correlated with LVH. We found that the most predictive value for LVH in this patient population was average daytime SBP based on ABPM. Our finding is consistent with other studies that have shown a direct relationship between LVH and SBP as measured either by ABPM or office BP averaged over a 30-year period.$^{19-21}$ In the Framingham population, a continuous association between SBP and LVH

Table 1. Demographic and Clinical Data: Comparison Between Patients With and Without LVH

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Patients (n=599)</th>
<th>LVH (n=416)</th>
<th>No LVH (n=183)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean±SD</td>
<td>60.1±10.1</td>
<td>59.4±10.1</td>
<td>61.9±10.0</td>
<td>0.0055</td>
</tr>
<tr>
<td>Gender, n (%), % female</td>
<td>230.0 (38.4)</td>
<td>170.0 (40.9)</td>
<td>60.0 (32.8)</td>
<td>0.0611</td>
</tr>
<tr>
<td>Body mass index, mean±SD, kg/m²</td>
<td>31.2±6.98</td>
<td>32.6±7.09</td>
<td>27.9±5.48</td>
<td>0.0001</td>
</tr>
<tr>
<td>Smoking status, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoked</td>
<td>244.0 (40.8)</td>
<td>176.0 (42.4)</td>
<td>68.0 (37.2)</td>
<td></td>
</tr>
<tr>
<td>Currently smoking</td>
<td>105.0 (17.6)</td>
<td>76.0 (18.3)</td>
<td>29.0 (15.8)</td>
<td></td>
</tr>
<tr>
<td>Past smoker</td>
<td>249.0 (41.6)</td>
<td>163.0 (39.3)</td>
<td>86.0 (47.0)</td>
<td></td>
</tr>
<tr>
<td>Hematocrit, mean±SD %</td>
<td>38.4±5.27</td>
<td>38.2±5.12</td>
<td>38.9±5.59</td>
<td>0.193</td>
</tr>
<tr>
<td>eGFR, mean±SD, mL/min/1.73 m²</td>
<td>44.2±16.5</td>
<td>43.4±16.3</td>
<td>46.1±17.0</td>
<td>0.0734</td>
</tr>
<tr>
<td>Serum creatinine, mean±SD, mg/dL</td>
<td>2.25±1.35</td>
<td>2.30±1.48</td>
<td>2.14±1.00</td>
<td>0.1164</td>
</tr>
<tr>
<td>Serum cholesterol, mean±SD, mg/dL</td>
<td>201.0±45.5</td>
<td>202.0±45.8</td>
<td>199.0±44.7</td>
<td>0.3787</td>
</tr>
<tr>
<td>Albuminuria, n (%), UACR &gt;0.22</td>
<td>75.0 (12.6)</td>
<td>66.0 (15.9)</td>
<td>9.0 (4.9)</td>
<td>0.0002</td>
</tr>
</tbody>
</table>

UACR indicates urinary albumin:creatinine ratio.

Table 2. Association of BP Measurements with LVH

<table>
<thead>
<tr>
<th>Variable, mm Hg</th>
<th>All Patients (n=599), Mean±SD</th>
<th>LVH (n=416), Mean±SD</th>
<th>No LVH (n=183), Mean±SD</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline clinic SBP</td>
<td>135.0±21.5</td>
<td>138.0±21.1</td>
<td>131.0±21.6</td>
<td>0.0003</td>
</tr>
<tr>
<td>Baseline clinic DBP</td>
<td>80.7±12.4</td>
<td>81.6±12.8</td>
<td>78.2±11.3</td>
<td>0.0068</td>
</tr>
<tr>
<td>Baseline clinic pulse pressure</td>
<td>54.6±16.6</td>
<td>55.9±16.5</td>
<td>51.8±16.7</td>
<td>0.0057</td>
</tr>
<tr>
<td>24-h pulse pressure</td>
<td>56.1±12.5</td>
<td>58.0±12.5</td>
<td>51.6±11.4</td>
<td>0.0001</td>
</tr>
<tr>
<td>24-h average SBP</td>
<td>136.0±18.0</td>
<td>139.0±18.0</td>
<td>130.0±16.6</td>
<td>0.0001</td>
</tr>
<tr>
<td>24-h average DBP</td>
<td>80.0±11.2</td>
<td>80.7±11.5</td>
<td>78.3±10.3</td>
<td>0.0126</td>
</tr>
<tr>
<td>Average day SBP</td>
<td>138.0±17.0</td>
<td>140.0±16.8</td>
<td>132.0±16.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Average day DBP</td>
<td>82.6±10.8</td>
<td>83.2±10.9</td>
<td>81.3±10.5</td>
<td>0.0388</td>
</tr>
<tr>
<td>Average night SBP</td>
<td>134.0±21.0</td>
<td>137.0±21.1</td>
<td>128.0±19.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Average night DBP</td>
<td>77.3±13.0</td>
<td>78.2±13.6</td>
<td>75.4±11.6</td>
<td>0.01</td>
</tr>
<tr>
<td>Average day-night SBP</td>
<td>3.26±12.8</td>
<td>2.80±12.9</td>
<td>4.31±12.7</td>
<td>0.1818</td>
</tr>
</tbody>
</table>
was found even at BP levels considered to be in the normal or near-reference range.22 Most investigators have found that SBP is better correlated with LVH than diastolic BP19,20 and that ABPM is a better predictor of LVH than clinic BP.20

Average nighttime SBP was also strongly predictive of LVH. Although average daytime SBP was reasonably well controlled in our population, average nighttime SBP was elevated. In other studies, African Americans have been shown to have higher nighttime BP than whites despite similar daytime BP.23,24 The majority of patients with uncomplicated hypertension experience a fall in their nighttime BP of >10% of the daytime mean arterial BP. The absence of a fall in nocturnal BP has been linked to target organ damage, such as carotid artery structural alterations and LVH in some studies25–29 but not others.30,31 Observational studies have shown that hypertensive patients with elevated nighttime BP are at greater risk for experiencing cardiovascular outcomes and stroke compared with hypertensive patients with a normal decrease in nocturnal BP.25,32 In our patient population there was an additive relationship between nighttime and daytime SBP, with the greater LVH occurring in patient in the upper quartiles of both night and day SBP.

Unlike previous studies, we found that younger age was associated with greater LVM in this cohort population. The AASK cohort population is derived from a high-risk, older population followed through the 5-year AASK and is composed of survivors who had not died or reached end-stage renal disease. Therefore, this unexpected finding may represent a survival bias; those patients who were older with lower LVM and those who were younger (with or without high LVH) may have been less likely to reach an outcome in the trial, and, therefore, were eligible for enrollment in the cohort study.

We did not find an independent relationship between the presence of albuminuria and LVH. This observation contrasts with some33,34 but not other35,36 studies where albuminuria correlated with LVH. Many of these studies measured clinic rather than ambulatory BP.33 ABPM values (particularly ambulatory SBP) have been shown to have a closer association with LVH compared with office measurements.35,37–39 The use of ABPM data in our analysis may be one reason for the lack of an independent association between urinary albumin excretion and LVH. Other studies using ABPM in their analysis have also found a lack of independent association between albuminuria and LVH.35,36 Unlike other studies evaluating albuminuria, the vast majority of patients (75%) in the AASK cohort were treated with angiotensin-converting enzyme inhibitors. Increased renin-angiotensin activity has been associated with the presence of microalbuminuria and target organ damage;40 and it is possible that the widespread use of angiotensin-converting enzyme inhibitors in our patient population accounted in part for the lack of association in our study. Previous studies also excluded patients with renal insufficiency,33–35 whereas all of the AASK cohort patients had established hypertensive renal dysfunction.

### Table 3. Echocardiographic Data: Comparison Between Patients With and Without LVH

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Patients (n=599)</th>
<th>LVH (n=416)</th>
<th>No LVH (n=183)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVDD, mean±SD, mm</td>
<td>48.4±6.70</td>
<td>50.1±6.49</td>
<td>44.3±5.30</td>
<td>0.0001</td>
</tr>
<tr>
<td>LVDS, mean±SD, mm</td>
<td>28.8±6.85</td>
<td>30.1±7.04</td>
<td>25.8±5.34</td>
<td>0.0001</td>
</tr>
<tr>
<td>IVST, mean±SD, mm</td>
<td>13.4±2.91</td>
<td>14.2±2.81</td>
<td>11.6±2.30</td>
<td>0.0001</td>
</tr>
<tr>
<td>PWT, mean±SD, mm</td>
<td>12.7±2.46</td>
<td>13.4±2.39</td>
<td>11.1±1.81</td>
<td>0.0001</td>
</tr>
<tr>
<td>Aortic root diameter, mean±SD, mm</td>
<td>34.2±5.10</td>
<td>34.8±5.04</td>
<td>33.0±5.03</td>
<td>0.0001</td>
</tr>
<tr>
<td>Left atrium diameter, mean±SD, mm</td>
<td>41.2±6.95</td>
<td>43.0±6.55</td>
<td>37.2±6.08</td>
<td>0.0001</td>
</tr>
<tr>
<td>Left atrial area, mean±SD, cm²</td>
<td>19.2±5.12</td>
<td>20.4±5.21</td>
<td>16.6±3.72</td>
<td>0.0001</td>
</tr>
<tr>
<td>LV mass index, mean±SD, g/m²²</td>
<td>60.9±21.6</td>
<td>70.0±19.6</td>
<td>40.2±6.07</td>
<td>0.0001</td>
</tr>
<tr>
<td>RWT, mean±SD</td>
<td>0.53±0.13</td>
<td>0.55±0.14</td>
<td>0.51±0.11</td>
<td>0.0003</td>
</tr>
<tr>
<td>RWT=0.44, n (%)</td>
<td>467.0 (78.0)</td>
<td>329.0 (79.1)</td>
<td>138.0 (75.4)</td>
<td>0.3173</td>
</tr>
<tr>
<td>Fractional shortening, mean±SD, %</td>
<td>0.38±0.10</td>
<td>0.38±0.10</td>
<td>0.38±0.10</td>
<td>0.8899</td>
</tr>
<tr>
<td>Doppler SV, mean±SD, mL</td>
<td>98.9±58.9</td>
<td>103.0±60.2</td>
<td>89.8±55.0</td>
<td>0.0124</td>
</tr>
<tr>
<td>Doppler cardiac index, mean±SD, L/min/m²</td>
<td>3.03±1.67</td>
<td>3.08±1.72</td>
<td>2.92±1.54</td>
<td>0.3413</td>
</tr>
<tr>
<td>SV/PP ratio, mean±SD</td>
<td>1.72±1.12</td>
<td>1.73±1.12</td>
<td>1.69±1.14</td>
<td>0.6902</td>
</tr>
<tr>
<td>SV/PP ratio, mean±SD, 24-h pulse pressure</td>
<td>1.83±1.14</td>
<td>1.84±1.12</td>
<td>1.81±1.17</td>
<td>0.7632</td>
</tr>
<tr>
<td>Heart rate, mean±SD</td>
<td>67.0±13.4</td>
<td>66.1±13.0</td>
<td>69.5±13.9</td>
<td>0.0117</td>
</tr>
</tbody>
</table>

IVST indicates intraventricular wall thickness; PWT, posterior wall thickness; SV, stroke volume; SV/PP ratio, stroke volume:pulse pressure ratio.

**Figure.** Joint association of mean LVM with quartiles of day and night SBP.
Therefore, in patients with established renal dysfunction, the additional presence of albuminuria may be less strongly associated with LVH.

We found GFR to be a predictor of LVH independent of BP (measured at the time of enrollment in the cohort) and the hemodynamic effects of anemia. An association between renal dysfunction and LVH has been described by others in recent studies. Data from the Framingham Study, a predominately white population, has shown that patients with even mild renal impairment have twice the prevalence of coronary disease, heart failure, ischemic stroke, and LVH, although the results were not adjusted for BP. In an exclusively white population with normal serum creatinine, abnormal renal function as defined by GFR <60 mL/min per 1.73 m² or the presence of microalbuminuria was associated with increased LVM. The only other study to date identifying a relationship between GFR and LVH in an African American population is the Atherosclerosis Risk in Communities Study. In this population study of 1968 African Americans, those with mildly reduced kidney function (32 patients) had greater eGFR than those who had normal GFR (P = 0.036). The association between renal dysfunction and LVH may thus be observed, but its full importance remains to be confirmed.

The association between renal dysfunction and LVH may explain in part the high cardiovascular morbidity and mortality observed in African Americans with hypertensive kidney disease. Although the mechanism of this association is not fully understood, factors associated with renal impairment, including increased calcium-phosphorus product, hyperphosphoremia, parathormone, sympathetic nervous system activity, and activation of the renin-angiotensin-aldosterone system, may contribute to the target-organ damage and cardiovascular risk in these patients as well. The relationship of reduced eGFR with LVH in this population may also represent an indirect effect of sustained BP elevation over time. Our results emphasize the importance of early identification of renal impairment in targeting those at greater risk for cardiovascular events. In conclusion, in African Americans with hypertension and chronic kidney disease, LVH is strongly associated with both higher average daytime and nighttime SBP and lower eGFR.

Perspectives
We documented an extremely high prevalence of LVH in African Americans with hypertensive kidney disease, despite excellent BP control, as measured by office BP, during the preceding 5 years. The high prevalence of LVH is likely multifactorial but may relate in part to a genetic predisposition in African Americans. Recently identified candidate genes that are more common in African Americans and also seem to be associated with higher LVM or inappropriate LVH include a variant of the corin gene, a polymorphism in the calcineurin gene, and the 894T allele of endothelial NO synthase gene.

The high prevalence of LVH in this population raises the question of whether LVH itself should be a therapeutic target. Regression of LVM with effective BP reduction has been demonstrated in >400 clinical studies, but <10% have been double-blind, placebo-controlled studies. Data indicate that LVM regression reduced adverse clinical outcomes in hypertensive patients. In a prospective cohort study, a decreased risk of cardiac events with LVM regression was found independent of baseline LVM, BP, and the degree of BP reduction. In the Losartan Intervention for Endpoint Reduction Trial, patients who had LVM regression with treatment had significantly less cardiac morbidity and mortality. A mechanism that might explain these findings is that midwall fractional shortening, a sensitive measure of intrinsic myocardial systolic performance, improves with LV mass regression. Our own findings that LVH and LVM were significantly associated with daytime and nighttime SBP provide a strong rationale for additional research, particularly clinical trials, that test whether reduction in ambulatory BP and targeted treatment of nighttime BP reduce LVM and prevent clinical cardiovascular events.

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Disclosures
S.R. has ownership interest in Merck and Pfizer. The remaining authors report no conflicts.
References


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Prevalence and Correlates of Left Ventricular Hypertrophy in the African American Study of Kidney Disease Cohort Study

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*deceased

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Table S1. Supplemental demographic and clinical data: comparison between patients with and without LVH

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients (n=599)</th>
<th>LVH (n=416)</th>
<th>No LVH (n=183)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n(%) or mean +/- s.d.</td>
<td>n(%) or mean +/- s.d.</td>
<td>n(%) or mean +/- s.d.</td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$15,000</td>
<td>243 (40.6%)</td>
<td>175 (42.1%)</td>
<td>68 (37.2%)</td>
</tr>
<tr>
<td>$15,000-39,999</td>
<td>163 (27.2%)</td>
<td>110 (26.4%)</td>
<td>53 (29%)</td>
</tr>
<tr>
<td>$40,000+</td>
<td>64 (10.7%)</td>
<td>41 (9.9%)</td>
<td>23 (12.6%)</td>
</tr>
<tr>
<td>Declines to answer</td>
<td>129 (21.5%)</td>
<td>90 (21.6%)</td>
<td>39 (21.3%)</td>
</tr>
<tr>
<td>Education (from trial)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not a HS graduate</td>
<td>235 (39.3%)</td>
<td>161 (38.7%)</td>
<td>74 (40.7%)</td>
</tr>
<tr>
<td>HS Graduate</td>
<td>190 (31.8%)</td>
<td>137 (32.9%)</td>
<td>53 (29.1%)</td>
</tr>
<tr>
<td>College or beyond</td>
<td>173 (28.9%)</td>
<td>118 (28.4%)</td>
<td>55 (30.2%)</td>
</tr>
<tr>
<td>C-reactive protein (CRP)</td>
<td>0.80 +/- 1.22</td>
<td>0.80 +/- 0.98</td>
<td>0.81 +/- 1.65</td>
</tr>
<tr>
<td>(mg/dL)</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>
Appendix. African American Study of Kidney Disease Cohort Investigators

Coordinators – Karen Brittain, Susan Sherer, Laurie Tuason, Cynthia Kendrick, Sharon Bi, Harvey Litowitz, Xianyou Liu, Xuelei Wang, Kimberly Wiggins, Cheryl A. Tatum.