Disparate Estimates of Hypertension Control From Ambulatory and Clinic Blood Pressure Measurements in Hypertensive Kidney Disease

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Abstract—Ambulatory blood pressure (ABP) monitoring provides unique information about day-night patterns of blood pressure (BP). The objectives of this article were to describe ABP patterns in African Americans with hypertensive kidney disease, to examine the joint distribution of clinic BP and ABP, and to determine associations of hypertensive target organ damage with clinic BP and ABP. This study is a cross-sectional analysis of baseline data from the African American Study of Kidney Disease Cohort Study. Masked hypertension was defined by elevated daytime (≥135/85 mm Hg) or elevated nighttime (≥120/70 mm Hg) ABP in those with controlled clinic BP (<140/90 mm Hg); nondipping was defined by a ≥10% decrease in mean nighttime systolic BP; reverse dipping was defined by a higher nighttime than daytime systolic BP. Of the 617 participants (mean age: 60.2 years; 62% male; mean estimated glomerular filtration rate: 43.8 mL/min per 1.73 m²) with both clinic BP and ABP, 498 participants (80%) had a nondipping or reverse dipping profile. Of the 377 participants with controlled clinic BP (61%), 70% had masked hypertension. Compared with those with controlled clinic BP or white-coat hypertension, target organ damage (proteinuria and left ventricular hypertrophy) was more common in those with elevated nighttime BP, masked hypertension, or sustained hypertension. In conclusion, clinic BP provides an incomplete and potentially misleading assessment of the severity of hypertension in African Americans with hypertensive kidney disease, in large part because of increased nighttime BP. Whether lowering nighttime BP improves clinical outcomes is unknown but should be tested given the substantial burden of BP-related morbidity in this population. (Hypertension. 2009;53:00-00.)

Key Words: blood pressure ■ chronic kidney disease ■ African American ■ nighttime hypertension

The lifetime risk for developing end-stage renal disease is 4-fold greater in African Americans as compared with whites.1–3 Hypertension causes 34% of all end-stage renal disease in African Americans, and hypertensive kidney disease in African Americans accounted for 9% of all end-stage renal disease in the United States from 1995 to 1998.4 These data suggest greater susceptibility of African Americans to progressive kidney disease from hypertension in comparison with non–African Americans. One possible explanation for this greater susceptibility of African Americans to kidney damage from hypertension is abnormal day-night patterns of blood pressure (BP), which cannot be detected by traditional clinic-based BP measurements and which may be abnormal even in the setting of controlled clinic BP. Few studies have examined the prevalence of masked hypertension, defined as a normal clinic BP with abnormally elevated ambulatory BP (ABP), in patients with chronic kidney disease.

In the African American Study of Kidney Disease and Hypertension (AASK) Trial,5 we reported previously that a lower BP goal (mean arterial pressure <92 mm Hg) did not result in slower progression of chronic kidney disease compared with a usual BP goal (mean arterial pressure of 102 to 107 mm Hg). After completion of the AASK Trial, a prospective cohort study including 691 AASK Trial participants was initi-
alyzed. An important goal of the cohort study was to evaluate novel factors that were associated with decline in renal function. Putative risk factors included abnormal ABP profiles.

The objectives of this article were 3-fold, namely, to describe ABP patterns in African Americans with hypertensive kidney disease, to examine the joint distribution of clinic BP and ABP, and to determine the associations of hypertensive target organ damage with clinic BP and ABP.

Methods

Study Design
The AASK Cohort Study is a prospective, observational study that is an extension of the AASK Trial. A major objective of the cohort study is to identify risk factors for kidney disease progression in the setting of recommended BP therapy. Data collection for the cohort phase of AASK began in April 2002. A detailed description of study participants has been published. In brief, AASK Trial participants were African Americans with hypertension (diastolic BP of ≥95 mm Hg), aged 18 to 70 years, with glomerular filtration rate (GFR) between 20 and 65 mL/min per 1.73 m². Major exclusion criteria were diabetes mellitus, urinary protein:creatinine ratio (milligrams of protein per milligram of creatinine) >2.5, evidence of renal disease other than hypertensive nephrosclerosis, or an absolute indication or contraindication for any of the randomized drugs. Participants who were alive without end-stage renal disease at the end of the AASK Trial were eligible to participate in the cohort study.

The protocol was approved by the institutional review boards of the participating centers. All of the study participants provided written informed consent. An independent data and safety monitoring board for the clinical phase and a scientific advisory board for the cohort study provided oversight. Of the 795 participants eligible for the AASK Cohort Study, 691 (86.9%) enrolled. An antihypertensive regimen including an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker was recommended along with a clinic BP goal of <140/90 mm Hg (subsequently lowered to <130/80 mm Hg).

All of the measurements in this report were obtained at the baseline visit of the AASK Cohort Study. Data collection included questionnaire responses, clinic BP, ABP, weight, fasting blood, and a 24-hour urine collection. A 2D, M-mode pulsed Doppler and pulsed tissue Doppler echocardiogram were obtained and processed by the Cardiovascular Research Foundation and then read centrally by trained echocardiographers.

Clinic BP Measurement
All of the BP measurements were performed by trained and certified staff using a Tycos classic handheld aneroid device. The device was calibrated every 6 months. Three consecutive seated readings were recorded. In our analyses, clinic BP was the mean of the last 2 readings.

ABP Measurement
Twenty-four-hour ABP monitoring was performed with the SpaceLabs Medical Model 90207. Arm circumference was measured to ensure that an appropriate size cuff was used, and the center of the inflatable bladder was placed over the brachial artery. The monitor was programmed to record BP every 30 minutes and was set to not display monitor readings. The ABP reading was considered adequate if the monitor had been worn for a full 24 hours and if there were ≥14 acceptable readings between 6 AM and 12 AM (daytime) and 6 acceptable readings between 12 AM and 6 AM (nighttime). To assess the impact of alternative ABP definitions, we performed a sensitivity analysis in which we used ABP definitions proposed by Fagard et al.10 (daytime BP between 10 AM and 8 PM and nighttime BP between 12 AM and 6 AM).

Definitions
Controlled clinic BP was defined as a level of <140/90 mm Hg. ABP was considered normal if the daytime value was <135/85 mm Hg, nighttime value <120/70 mm Hg, and 24-hour value <130/80 mm Hg.11 Masked hypertension was defined as a controlled clinic BP and either an elevated daytime (≥135/85 mm Hg) or an elevated nighttime (≥120/70 mm Hg) ABP. Nocturnal dipping was defined as a ratio of mean nighttime:mean daytime systolic BP ratio of <0.90 (>10% decrease in mean systolic BP at night). Nondipping was defined as a ratio >0.9 (<10% decrease in nighttime systolic BP), and reverse dipping was defined as a ratio >1.0.

Statistical Analysis
Baseline characteristics were compared between BP category groups using ANOVA procedure or a χ² test, as appropriate. Multiple logistic regression models were used to relate BP category status to estimated GFR, gender, duration of hypertension, BMI, age, 24-hour urinary sodium and potassium excretion, and statin use. Multiple linear regression models were used to explore the relationship between the daytime-nighttime difference in systolic BP and nighttime systolic BP with these variables. All of the P values were 2-sided, without adjustment of multiple comparisons.

Results
Of the 619 participants (89.5%) who agreed to have ABP monitoring, 617 had an acceptable study. The mean (SD) number of BP measurements contributed to ABP averages was 36.6 (7.2) for daytime, 11.3 (1.4) for nighttime, and 47.9 (7.4) for 24-hour ABP. Table 2 displays the baseline characteristics of participants overall and stratified by dipping status.

BP Levels
Mean clinic BP was 134.0/79.8 mm Hg (Table 2). Average ambulatory daytime systolic pressure was 138.0 mm Hg, and average nighttime systolic pressure was only slightly lower, at 134.0 mm Hg. Average daytime diastolic pressure was 82.5 mm Hg, and average nighttime diastolic pressure was 77.3 mm Hg. The 24-hour average BP was 137.0/81.2 mm Hg. With control defined as a clinic BP <140/90 mm Hg, 377 participants (61%) were controlled by clinic BP, 255 (41.3%) were controlled by ambulatory daytime BP, and 150 (24.3%) were controlled by ambulatory nighttime BP.

Dipping Status
As shown in Table 2, 122 participants (20%) were “dippers.” Remarkably, 495 participants (80%) were either “nondippers” (41%) or “reverse dippers” (39%). There was no significant difference in clinic systolic BP and average daytime systolic BP among the 3 groups; however, nighttime systolic BP was significantly higher in the reverse-dipper (146±20 mm Hg) and the nondipper (132±16 mm Hg) groups compared with the dipper group (116±15 mm Hg; P<0.0001; Table 2 and Figure 1).

Participants in the reverse-dipper category were older, more likely to be men, had a longer duration of hypertension, and were prescribed more antihypertensive medications than those in the dipper and nondipper categories. The distribution of classes of antihypertensive drugs and the dosing of antihypertensives more than once a day did not differ by dipping status (Table 1). Also, there were no differences in smoking, prevalence of left ventricular hypertrophy on echocardiography, or body mass index among the 3 groups.
Participants in the reverse-dipper and nondipper groups were more likely to have a urinary protein:creatinine ratio >0.22 g/g than the dipper group. Estimated GFR, urinary sodium excretion, and other biochemical parameters were not significantly different among the 3 groups.

In sensitivity analyses with daytime BP averaged between 10 AM and 8 PM instead of 6 PM to 12 AM, the distribution of dipping status was essentially unchanged, with 21% dippers, 37% nondippers, and 41% reverse dippers. With the exception of current smoking, which was associated with a higher
prevalence of reverse dipping using the alternate definition ($P=0.04$), and proteinuria, which was no longer associated with dipping status ($P=0.26$), the remaining 24 bivariate relationships in Table 1 were similar for the 2 classifications of daytime BP.

### Comparison of Clinic and ABP and the Prevalence of Masked Hypertension

Assessment of concordance of clinic and ABP (Table 3) demonstrated agreement between the 2 methods in only 338 participants (54.7%): 226 individuals (36.6%) were considered controlled by both clinic and ABP, and 112 (18.1%) were considered uncontrolled by both measurements. Clinic and ABP yielded discordant results in 279 study participants (45.2%): 14 (2.2%) had elevated clinic BP but controlled ambulatory pressures (white coat hypertension), and 265 (42.9%) had controlled clinic but elevated ambulatory pressures (masked hypertension). Importantly, among those with controlled clinic BP, 70% had either uncontrolled daytime or nighttime BP. In contrast, those with elevated clinic BP usually had elevated ABP as well; of the 240 individuals who were uncontrolled by clinic BP criteria, 226 (94%) were also uncontrolled by ABP.

Of the 265 participants with masked hypertension, 151 had uncontrolled daytime BP, and 250 had uncontrolled nighttime BP. Hence, elevated nighttime BP, with or without elevated daytime BP, was the most common cause of masked hypertension; only 15 individuals had masked hypertension from isolated elevation of daytime BP. Although elevation of either systolic or diastolic BP could result in masked hypertension, elevation of systolic BP was the predominant finding. Figure 2 illustrates the relationship between nighttime ambulatory systolic BP and clinic systolic BP at baseline. As shown in Figure 2, elevated nighttime systolic BP was found in the majority of participants with normal clinic systolic BP (249 of 377 [66%]) and explains the high prevalence of masked hypertension.

Participants with masked hypertension had similar demographic characteristics and degree of target organ damage as those with sustained hypertension (Table 4). Compared with those with controlled or white coat hypertension, those with masked hypertension were older and more likely to be men. Although pattern of drug classes was similar, compared with those with controlled BP or white coat hypertension, subjects with masked and sustained hypertension used a greater total number of drugs ($P=0.01$) and tended to more frequently receive antihypertensives more than once daily ($P=0.05$). Individuals with masked hypertension were also more likely to have abnormal dipping patterns and to have a greater prevalence of left ventricular hypertrophy and a protein:creatinine ratio $>0.22$ g/g.

### Table 3. Comparison of Assessment of BP Control by ABP vs Clinic BP

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (N=617), N (%)</th>
<th>Controlled Clinic BP (N=377), N (%)</th>
<th>Uncontrolled Clinic BP (N=240), N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controlled</td>
<td>255 (41.3)</td>
<td>222 (58.9)</td>
<td>33 (13.8)</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>362 (58.7)</td>
<td>155 (41.1)</td>
<td>207 (86.3)</td>
</tr>
<tr>
<td>Nighttime BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controlled</td>
<td>150 (24.3)</td>
<td>127 (33.7)</td>
<td>23 (9.6)</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>467 (75.7)</td>
<td>250 (66.3)</td>
<td>217 (90.4)</td>
</tr>
<tr>
<td>Daytime or nighttime BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Both controlled</td>
<td>126 (20.4)</td>
<td>112 (29.7)</td>
<td>14 (5.8)</td>
</tr>
<tr>
<td>Either uncontrolled</td>
<td>491 (79.6)</td>
<td>265 (70.3)</td>
<td>226 (94.2)</td>
</tr>
</tbody>
</table>

### Association Between Nighttime BP and Hypertensive Target Organ Damage

Participants in the highest tertile of nighttime systolic pressure ($\geq 142$ mm Hg) were compared with those in the middle tertile (126 to 141 mm Hg) and lowest tertile ($<126$ mm Hg; Table 5). Participants in the highest tertile had a rise in systolic BP of 4.8 mm Hg at night compared with a decrease...
of 10.4 mm Hg at night in those in the lowest tertile. In addition, clinic BP and all of the parameters of ABP were higher in the highest tertile of nighttime systolic BP. There were no differences in nonsteroidal anti-inflammatory drug use, exercise frequency, or alcohol use among the 3 tertiles of nighttime systolic pressure. Participants in the highest tertile of nighttime systolic BP were more likely to have left ventricular hypertrophy on echocardiography, higher prevalence of proteinuria, and a lower estimated GFR than those in the middle and lower tertiles. Also, among those in the highest tertile, there was greater use of calcium channel blockers, β-blockers, and more-than-once-daily drugs, as well as total number of drugs.

In multivariable models, we explored the independent correlates of dipper status, daytime-nighttime difference in systolic BP, and nighttime systolic BP (Table S1, available online at http://hyper.ahajournals.org). Female gender (odds ratio: 1.6; P=0.03) and a shorter duration of hypertension (odds ratio: 0.97; P=0.04) were independently associated with a higher likelihood of a dipper profile. A higher estimated GFR and female gender were independently associated with a greater decline in nighttime systolic BP. Higher urinary sodium excretion was independently associated with higher nighttime systolic BP.

**Discussion**

In this study of African American patients with hypertensive kidney disease, we documented a remarkably high prevalence of nondipping or reverse dipping, elevated nighttime BP, and masked hypertension. Higher nighttime BP and masked hypertension were associated with increased severity of hypertension-related target organ damage. Nighttime BP was elevated in most participants, and this elevation in nighttime BP was responsible for the high prevalence of reverse dipping and nondipping. These abnormal circadian rhythms are not explained by patterns of drug use (Tables 1, 4, and 5).
A pattern of BP has been reported to be more common in recent years. In previous studies, masked hypertension was described in detail in a previous publication. Some studies have shown that the alteration of timing of antihypertensive drug therapy can lower nighttime BP and improve the diurnal profile of BP.

Of particular interest was our finding that nondippers and reverse dippers had similar clinic BP as dippers, although reverse dippers and nondippers had elevated 24-hour and nighttime BP compared with dippers. On the basis of large observational studies, elevations in 24-hour systolic pressure of a magnitude found in the current study (6 to 8 mm Hg) would be expected to result in as much as a 30% increase in cardiovascular events and mortality. Elevated nighttime BP is also one possible explanation for the high rate of kidney disease progression that we documented previously and for the lack of benefit of the lower BP goal, based on clinic analyses, as nighttime BP increased, estimated GFR decreased, and the prevalence of elevated proteinuria increased substantially (Table 5). The mechanisms responsible for elevated nighttime BP are uncertain; expanded extracellular volume status, increased sympathetic nervous system activity, and altered circadian pattern of sodium excretion have been proposed. Our data support a potential role of altered sodium handling as a cause of elevated nighttime BP, because higher urinary sodium excretion was associated with elevated nighttime BP.

The high prevalence of masked hypertension in this cohort was striking and underscores the limitations of clinic BP in assessing the severity of hypertension in African Americans with hypertensive kidney disease. As illustrated in Figure 2, the majority of participants with abnormal BP elevation had either masked hypertension (42.9%) or sustained hypertension (18.1%); only 2.2% had white coat hypertension. The phenomenon of masked hypertension has been described recently. In previous studies, masked hypertension was prevalent in 9% to 14% in the general population and has been associated not only with increased hypertensive target organ damage, such as proteinuria, left ventricular hypertrophy, and carotid atherosclerosis, but also with increased cardiovascular morbidity and mortality. The prevalence of masked hypertension in our study is also higher than the prevalence of 25% noted in previous studies of patients with chronic kidney disease.

Other notable findings were the relationship between nighttime BP and indices of kidney function. In multivariate analyses, as nighttime BP increased, estimated GFR decreased, and the prevalence of elevated proteinuria increased substantially (Table 5). The mechanisms responsible for elevated nighttime BP are uncertain; expanded extracellular volume status, increased sympathetic nervous system activity, and altered circadian pattern of sodium excretion have been proposed. Our data support a potential role of altered sodium handling as a cause of elevated nighttime BP, because higher urinary sodium excretion was associated with elevation of nighttime BP.

Elevated nighttime BP was also associated with a higher likelihood of left ventricular hypertrophy. The association of nighttime BP with left ventricular hypertrophy in AASK is described in detail in a previous publication. Some studies have shown that the alteration of timing of antihypertensive drug therapy can lower nighttime BP and improve the diurnal profile of BP. Whether this can be accomplished in patients with chronic kidney disease remains to be seen.

Table 5. Participant Characteristics by Nighttime Systolic BP Category

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Subjects (N=617)</th>
<th>Lowest Tertile (&lt;126 (N=215)</th>
<th>Middle Tertile 126 to 141 (N=202)</th>
<th>Highest Tertile &gt;142 (N=200)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean±SD</td>
<td>60.2±10.2</td>
<td>59.6±10.3</td>
<td>60.5±9.89</td>
<td>60.6±10.3</td>
<td>0.52</td>
</tr>
<tr>
<td>Gender, n (%), male</td>
<td>383 (62.1)</td>
<td>126 (58.6)</td>
<td>130 (64.4)</td>
<td>127 (63.5)</td>
<td>0.42</td>
</tr>
<tr>
<td>Duration of hypertension, mean±SD, y</td>
<td>20.0±10.0</td>
<td>18.8±9.83</td>
<td>20.8±10.0</td>
<td>20.5±10.1</td>
<td>0.07</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>104 (16.9)</td>
<td>29 (13.5)</td>
<td>33 (16.4)</td>
<td>42 (21.0)</td>
<td>0.12</td>
</tr>
<tr>
<td>LVH (determined from baseline echo), n (%)</td>
<td>413 (68.9)</td>
<td>118 (56.5)</td>
<td>137 (70.3)</td>
<td>158 (81.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Body mass index, mean±SD, kg/m²</td>
<td>31.3±7.04</td>
<td>30.6±7.48</td>
<td>32.1±6.83</td>
<td>31.1±6.69</td>
<td>0.09</td>
</tr>
<tr>
<td>Urinary protein:creatinine ratio, mean±SD</td>
<td>0.35±0.79</td>
<td>0.17±0.39</td>
<td>0.35±0.78</td>
<td>0.55±1.05</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Proteinuria, n (%), urinary protein:creatinine ratio &gt;0.22 g/g</td>
<td>181 (29.4)</td>
<td>34 (15.9)</td>
<td>61 (30.2)</td>
<td>86 (43.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Serum creatinine, mean±SD, mg/dL</td>
<td>2.28±1.40</td>
<td>2.23±1.20</td>
<td>2.14±1.00</td>
<td>2.47±1.86</td>
<td>0.051</td>
</tr>
<tr>
<td>Estimated GFR, mean±SD, mL/min per 1.73 m²</td>
<td>43.8±16.3</td>
<td>44.3±16.7</td>
<td>45.8±16.5</td>
<td>41.3±15.6</td>
<td>0.01</td>
</tr>
<tr>
<td>Serum glucose, mean±SD, mg/dL</td>
<td>103±37.6</td>
<td>100±30.5</td>
<td>104±44.2</td>
<td>105±36.5</td>
<td>0.63</td>
</tr>
<tr>
<td>Calcium, mean±SD, mg/dL</td>
<td>9.64±0.55</td>
<td>9.67±0.51</td>
<td>9.67±0.52</td>
<td>9.58±0.62</td>
<td>0.33</td>
</tr>
<tr>
<td>Phosphorus, mean±SD, mg/dL</td>
<td>3.53±0.70</td>
<td>3.52±0.72</td>
<td>3.56±0.59</td>
<td>3.51±0.77</td>
<td>0.79</td>
</tr>
<tr>
<td>Urine sodium, mean±SD, g/d</td>
<td>3.77±2.97</td>
<td>3.54±1.77</td>
<td>3.77±1.84</td>
<td>4.02±5.41</td>
<td>0.25</td>
</tr>
<tr>
<td>No. of antihypertensive medications, mean±SD</td>
<td>3.53±1.44</td>
<td>3.13±1.31</td>
<td>3.61±1.49</td>
<td>3.86±1.42</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ACE inhibitor/ARB, n (%)</td>
<td>515 (84.8)</td>
<td>179 (85.2)</td>
<td>165 (82.9)</td>
<td>171 (86.4)</td>
<td>0.62</td>
</tr>
<tr>
<td>CCB, n (%)</td>
<td>174 (28.7)</td>
<td>47 (22.4)</td>
<td>70 (35.2)</td>
<td>57 (28.8)</td>
<td>0.02</td>
</tr>
<tr>
<td>Diuretic, n (%)</td>
<td>500 (82.4)</td>
<td>177 (84.3)</td>
<td>160 (80.4)</td>
<td>163 (82.3)</td>
<td>0.59</td>
</tr>
<tr>
<td>Vasodilator, n (%)</td>
<td>177 (29.2)</td>
<td>53 (25.2)</td>
<td>55 (27.6)</td>
<td>69 (34.8)</td>
<td>0.09</td>
</tr>
<tr>
<td>β-Blocker, n (%)</td>
<td>244 (40.2)</td>
<td>71 (33.8)</td>
<td>78 (39.2)</td>
<td>95 (48.0)</td>
<td>0.01</td>
</tr>
<tr>
<td>Antihypertensives more than once per day, n (%)</td>
<td>322 (53.2)</td>
<td>97 (46.0)</td>
<td>95 (48.5)</td>
<td>130 (68.7)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

LVH indicates left ventricular hypertrophy; CCB, calcium channel blocker; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker.
This study has several strengths. First, it is the largest study of ABP monitoring in patients with chronic kidney disease. Second, the quality of the data was excellent, with only 2 (0.32%) of 619 participants having recordings that could not be evaluated. Third, measurement of clinic BP was carefully standardized and performed by trained staff using frequently calibrated equipment.

Our study also has limitations. Because all of the participants were African American, extrapolations to other racial/ethnic groups should be done with caution. Second, our study was not designed to evaluate biological mechanisms. A more extensive evaluation of sympathetic nervous system activity, salt and water balance, and other homeostatic mechanisms is required. Because we performed 24-hour urine collection without split sampling, we could not examine the relation of nighttime salt excretion with dipping status. We could not evaluate the effects of diuretics on dipping status, because 85% of patients were using diuretics at baseline. Third, it has been noted that antihypertensive therapy can have differential effects on day and night BP; however, the magnitude of the difference is small and unlikely to explain the high prevalence of nondipping in our study. Finally, because the analyses are cross-sectional, we cannot draw causal links between any BP index and target organ damage.

In conclusion, clinic BP provides an incomplete and potentially misleading assessment of the severity of hypertension in African Americans with hypertensive kidney disease, in large part because of increased nighttime BP. The use of ABP monitoring reveals a very high prevalence of masked hypertension, which, in turn, may be associated with hypertension-related target organ damage. Whether lowering nighttime BP improves clinical outcomes is unknown but should be tested given the substantial burden of BP-related morbidity in this population.

Perspectives

Clinic BP provides an incomplete assessment of the severity of hypertension in African Americans with hypertensive kidney disease. The majority of participants with controlled clinic BP had abnormal BP profiles, including nondipping and/or reverse dipping, elevated nighttime BP, and masked hypertension. Higher nighttime BP and masked hypertension were associated with increased severity of hypertension-related target organ damage. Whether lowering nighttime BP can prevent progression of chronic kidney disease and other hypertension-related clinical outcomes is unknown but should be studied given the substantial burden of BP-related morbidity in this population.

Acknowledgment

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Disclosures

None.

References


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