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White Coat Hypertension Persistence in Children and Adolescents: The Pediatric Nephrology Research Consortium Study

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Abstract

Objectives—To determine whether youth with white coat hypertension on initial ambulatory blood pressure monitoring (ABPM) continue to demonstrate the same pattern on repeat ABPM.

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Study design—Retrospective longitudinal cohort study of patients referred for high blood pressure (BP) and diagnosed with white coat hypertension by ABPM who had follow-up ABPM 0.5-4.6 years later at 11 centers in the Pediatric Nephrology Research Consortium. We classified ABPM phenotype using the American Heart Association guidelines. At baseline, we classified those with hypertensive BP in the clinic as "stable white coat hypertension," and those with normal BP as "intermittent white coat hypertension." We used multivariable generalized linear mixed effect models to estimate the association of baseline characteristics with abnormal ABPM phenotype progression.

Results—Eighty-nine patients met the inclusion criteria (median age, 13.9 years; 78% male). Median interval time between ABPM measurements was 14 months. On follow-up ABPM, 61% progressed to an abnormal ABPM phenotype (23% ambulatory hypertension, 38% ambulatory prehypertension). Individuals age 12-17 years and those with stable white coat hypertension had greater proportions progressing to either prehypertension or ambulatory hypertension. In the multivariable models, baseline wake systolic BP index ≥0.9 was significantly associated with higher odds of progressing to ambulatory hypertension (OR 3.07, 95% CI 1.02-9.23).

Conclusions—The majority of the patients with white coat hypertension progressed to an abnormal ABPM phenotype. This study supports the 2017 American Academy of Pediatrics Clinical Practice Guideline's recommendation for follow-up of ABPM in patients with white coat hypertension.

White coat hypertension is defined by the finding of hypertensive clinic blood pressure (BP) but normal BP outside of clinical settings. In pediatric hypertension specialty clinics, white coat hypertension is most often diagnosed when patients referred for evaluation of high clinic BP are found to have a normal phenotype on ambulatory blood pressure monitoring (ABPM). The prevalence of white coat hypertension has been reported to be as high as 52% among referred patients.[1,2] The 2017 American Academy of Pediatrics Clinical Practice Guideline recommends using ABPM to evaluate patients suspected of having white coat hypertension.[3] From a survey of pediatric nephrologists in North America, 94% of respondents regularly use ABPM for this purpose.[4]

However, few data exist on outcomes of youth with white coat hypertension, and there are no evidence-based practice guidelines addressing follow-up for children and adolescents with white coat hypertension. The European Societies of Cardiology and Hypertension, the American Heart Association, and the American College of Cardiology suggest periodic follow-up with home BP measurements and ABPM for white coat hypertension in adults,[5,6] and the American Academy of Pediatrics Clinical Practice Guideline suggests follow-up ABPM in 1-2 years.[3] In a retrospective longitudinal study assessing ABPM stability in 124 otherwise healthy children and adolescents, we found that 12 out of 24 subjects who initially had normal ABPM progressed to either prehypertension or ambulatory hypertension on follow-up ABPM.[7] Our objectives were to determine whether youth with white coat hypertension remain normotensive on repeat ABPM, and if not, to identify baseline clinical indicators associated with change in ABPM phenotype. We hypothesized based on our previous work that many of those with white coat hypertension would progress to an abnormal phenotype.

Methods

We performed a retrospective longitudinal cohort study of patients at 11 centers in the Pediatric Nephrology Research Consortium who were diagnosed with white coat hypertension by initial ABPM between January 2007 and September 2019 and had a follow-up ABPM at least 6 months later and no more than 5 years later. All patients were under 18 years of age at the time of the initial ABPM study. For patients who were <13 years old at the time of the initial ABPM, the upper limit of follow-up ABPM for inclusion was reduced to within 2 years because of the rapid growth and increase in BP expected in preadolescents.[8] Exclusion criteria included known kidney disease or other causes of secondary hypertension, any history of malignancy, type 1 and 2 diabetes mellitus, and treatment with antihypertensive medication at the time of either ABPM. The number of patients included per site is reported in Table I (available at www.jpeds.com). Indication for the initial ABPM was evaluation of high BP in the referring providers' clinics and/or in the pediatric nephrology clinic. Patients receiving treatment with stimulant medications for attention deficit hyperactivity disorder (ADHD) were included in the study if their medication doses remained stable during the study period; those on clonidine or guanfacine were excluded.

Oscillometric devices were used for all ABPM studies (models 90207, 90217, and 90227; Spacelabs Healthcare). ABPM recordings were classified according to the approach recommended in the pediatric American Heart Association statement on interpretation of ABPM.[9] For patients 17 years of age and younger, ABPM studies were classified as normal and consistent with white coat hypertension if the mean wake and sleep systolic and diastolic BP were below the 95th percentiles for sex and height and BP loads were all <25%. Prehypertension was defined as mean wake and sleep systolic and diastolic BP below the 95th percentiles for sex and height and BP loads ≥25%. For patients 18 years of age and older, the wake hypertension threshold was set at 135/85 mm Hg and sleep threshold was set at 120/70 mm Hg.[10] For descriptive purposes, nocturnal BP dipping was defined as normal if ≥10%, blunted if 0 to <10%, and reversed if mean sleep BP exceeded mean wake BP.[9] The nephrologist interpreting the ABPM determined the adequacy of each recording. To facilitate comparisons between patients across the pediatric age range, BP indices were calculated by dividing the mean wake and sleep systolic and diastolic BP readings by appropriate 95th percentile values, wherein BP index ≥1.0 was consistent with hypertension.

Variables abstracted from the electronic health record included clinic BP measurements (auscultatory and/or oscillometric) at the initial and follow-up pediatric nephrology clinic visits as well as age, sex, height, height percentile for age and sex, body mass index (BMI), BMI z score for age and sex, history of obstructive sleep apnea, ADHD diagnosis and names and doses of ADHD medications, interval time between ABPM, and family history of hypertension, diabetes, and hyperlipidemia. Clinic BP index was defined similarly as ABPM BP index (ie, BP index of clinic BP at the 95th percentile = 1.0). The 2004 Fourth Report normative data were used for all patients due to the timing of patient assessments and data collection.[11] Oscillometric clinic BP was used to calculate clinic BP index if no auscultatory measurement was entered. We did not obtain data on clinic BP values or the type of measurement (auscultatory vs oscillometric) from referring providers. Once

local institutional review boards approved this study, data were collected manually by each center's investigators and submitted to the coordinating institution (Seattle Children's Hospital, University of Washington). Data were stored securely in a REDCap database[12] with study unique identification numbers and no patient identifiers.

Based on clinic BP measured at the initial pediatric nephrology visit, we divided the cohort into 2 groups, "stable white coat hypertension" and "intermittent white coat hypertension."[13] The stable white coat hypertension group was defined as those patients referred and confirmed to have pediatric nephrology clinic BP in the hypertension range (clinic BP index ≥1.0) during their initial visit. The intermittent white coat hypertension group was defined as those referred, but pediatric nephrology clinic BP index was <1.0.

Statistical Analyses

Baseline demographic and clinical characteristics were summarized by their initial pediatric nephrology clinic presentation, by white coat hypertension status (stable vs intermittent), and by follow-up ABPM phenotypes. Continuous variables were summarized using median with IQR. Categorical variables were summarized using counts and percentages. We dichotomized the age groups: <12 years old vs 12-17 years old because of an observation that rise in BP differs during puberty than in early life,[8] BP during the adolescent years tend to track stronger into adulthood,[14] and to be consistent with our previous longitudinal ABPM study.[7] We classified subjects with a BMI z score >1.64 as obese. Comparisons between groups were performed using Wilcoxon rank-sum tests or Kruskal-Wallis tests for continuous variables and Fisher exact tests for categorical variables. Histograms were examined to select ABPM systolic and diastolic BP index cut points of interest. Previously described cut points were also considered.[7] Spearman correlation coefficients were calculated across baseline ABPM systolic and diastolic BP index variables. We used generalized linear mixed effect models to explore the associations between baseline characteristics and progression to ambulatory hypertension (model A) or a combination of either prehypertension or ambulatory hypertension (model B) at follow-up ABPM, with a random intercept term for center in each model. In each model, we included the prespecified baseline covariates baseline age <12, BMI z score, and baseline white coat hypertension group, along with time between baseline and follow-up ABPM. ABPM systolic and diastolic BP index variables were added to the model based on a univariable regression *P* value of <.15. In the case of 2 or more highly correlated BP variables being significant in univariable analysis, the more statistically significant variable was chosen. A 2-sided *P* value of <.05 was considered statistically significant, and all analyses were performed with R v 3.6.2.

Results

Baseline Demographic and Clinical Characteristics

A total of 89 patients met the inclusion criteria. Table II summarizes the baseline characteristics of the cohort. The median age at the time of initial ABPM was 13.9 years, with 16 patients <12 years of age and 73 patients age 12-17 years. The cohort was predominantly male (78%). Overall, the median interval time between ABPM was 14 months (IQR: 12-22 months); 18 months (IQR: 12-24 months) for the <12-year-old group,

and 13 months (IQR: 12-19 months) for the 12- to 17-year-old group. The difference in the interval time between these 2 age groups was not statistically significant ($P = .23$, Wilcoxon rank-sum test). For both auscultatory and oscillometric clinic BP, median systolic BP indices exceeded 1.0 and median diastolic BP indices were below 1.0. Descriptive statistics of the initial ABPM studies are summarized in Table II.

Stable White Coat Hypertension vs Intermittent White Coat Hypertension

At the baseline pediatric nephrology clinic visit, 80% of patients met the case definition of stable white coat hypertension, and 20% met the intermittent white coat hypertension case definition. Table III summarizes baseline characteristics and ABPM data based on white coat hypertension category. There were no statistically significant differences in patient characteristics including age, sex, BMI z score, obesity prevalence, and any of the ABPM variables.

Follow-Up ABPM

Most patients (61%) progressed to an abnormal ABPM phenotype. Follow-up ABPM in 20 patients (23%) demonstrated ambulatory hypertension and in 34 patients (38%) showed prehypertension. Thirty-five patients (39%) had normal follow-up ABPM. Table IV summarizes the baseline patient characteristics grouped by follow-up ABPM phenotypes. A higher percentage of patients age 12-17 years and those with stable white coat hypertension at the time of initial ABPM were found among those who progressed to prehypertension or ambulatory hypertension at follow-up (overall Fisher exact test P values of .04 and .03, respectively). Baseline BMI z score and obesity were not associated with progression to an abnormal follow-up ABPM phenotype.

Table IV also provides a breakdown of the temporality of the ABPM abnormalities. Of the 20 patients who developed ambulatory hypertension phenotype, 65% demonstrated sleep-only hypertension, 10% had wake-only hypertension, and 25% had both wake and sleep hypertension. Of the 34 patients who developed prehypertension on follow-up ABPM, 41% had sleep-only prehypertension, 44% had wake-only prehypertension, and 15% had both wake and sleep prehypertension.

In multivariable analysis (Table V), wake systolic BP index ≥0.9 on baseline ABPM was significantly associated with higher odds of progressing to ambulatory hypertension (OR 3.07, 95% CI 1.02-9.23, model A). When combining prehypertension with ambulatory hypertension, wake systolic BP index ≥0.9 on baseline ABPM was again associated with higher odds of progression (OR 4.37, 95% CI 1.35-14.15, model B). In addition, baseline stable white coat hypertension was associated with higher odds of progressing to either prehypertension or ambulatory hypertension (OR 6.67, 95% CI 1.67-25.00).

Discussion

In this longitudinal follow-up study of youth with white coat hypertension who had a repeat ABPM at least 6 months later, we found that most patients progressed to an abnormal ABPM phenotype, including 23% progressing to ambulatory hypertension after a median follow-up time of 14 months. Of note, isolated sleep-hypertension, a subtype of masked

hypertension, was the most prevalent type of ambulatory hypertension found on follow-up ABPM. Patient characteristics at the initial pediatric nephrology evaluation that may be associated with risk for progression to an abnormal ABPM phenotype include age ≥12 years, stable white coat hypertension as determined by pediatric nephrology clinic BP, and wake systolic BP index ≥0.9. These data suggest that in children and adolescents with an initial diagnosis of white coat hypertension, follow-up ABPM is indicated. In addition, these data support the importance of follow-up ABPM given the high frequency of isolated sleep-hypertension observed.

Our study also demonstrated that neither baseline BMI nor obesity was predictive of abnormal follow-up ABPM, and we found this observation surprising. We believe that the sample size of our study may have been too small to demonstrate any association of obesity with progression to an abnormal ABPM phenotype. In addition, the relatively short time interval between ABPM studies and the wide participant age range, which spanned all pubertal stages, may have further contributed to the failure to demonstrate an independent effect of obesity on ABPM phenotype progression in our cohort.

Studies in adults demonstrate that individuals with white coat hypertension are at increased risk for progression to sustained hypertension.[15,16] However, the relevance of these findings for children is not certain considering that most participants in these studies were middle aged or older and thus at higher risk for incident hypertension. Longitudinal data on the risk of progression in children and adolescents with white coat hypertension are scant. In accordance with our findings, a small study from Sweden with a longer follow-up time of 9.3 years demonstrated that in 30 otherwise healthy school-age children with white coat hypertension, 7 subjects (23%) progressed to sustained hypertension.[17] Our findings, indicating that progression to ambulatory hypertension may occur in some patients over a shorter time frame, demonstrate the need for longitudinal follow-up with serial ABPM, and confirm the recommendation in the American Academy of Pediatrics Clinical Practice Guideline for follow-up ABPM 1-2 years following a diagnosis of white coat hypertension.[3]

Meta-analyses of studies conducted in adults have demonstrated that white coat hypertension conveys a cardiovascular risk that is intermediate between normotension and sustained hypertension. Adults with white coat hypertension have slightly higher rates of cardiovascular morbidity and mortality,[18] higher left ventricular mass index,[19] and greater common carotid artery intimal-media thickness.[20] Evidence for target organ changes in children with white coat hypertension is mixed. However, even though left ventricular hypertrophy prevalence is not increased in white coat hypertension, several studies have shown that left ventricular mass index in children with white coat hypertension is intermediate between that of children with normal BP and those with hypertension.[2,21-24] Similarly, children with white coat hypertension have greater carotid artery intimal-media thickness compared with children with normal BP,[24,25] and youth with white coat hypertension may have endothelial dysfunction similar to that observed in children with hypertension.[26] The Swedish white coat hypertension longitudinal study discussed above importantly compared the prevalence of left ventricular hypertrophy, carotid-femoral artery pulse wave velocity, and carotid intimal-media thickness at the time of follow-up between those who progressed to ambulatory hypertension and those who continued to have normal

ABPM. Although still within the normal range, those who progressed to ambulatory hypertension had significantly higher pulse wave velocity and carotid intimal-media thickness than the normotensive group, suggesting early-stage target organ changes.[17]

We also found a possible association between stable white coat hypertension at the initial pediatric nephrology clinic visit with progression to abnormal follow-up ABPM phenotype. Similarly, an adult 16-year follow-up study demonstrated that stable white coat hypertension was associated with increased risk for cardiovascular mortality and all-cause mortality when compared with normotensive controls and intermittent white coat hypertension was not.[13] Although few of the patients in our study had normal clinic BP at their initial pediatric nephrology clinic visit (20%), our data suggest these individuals may be at less risk for progression in this limited time frame.

Limitations of this study include its retrospective design, potential for selection bias, small sample size, and imperfect ABPM reproducibility. First, given the high frequency of white coat hypertension reported in the literature, it seems likely that many children from each center did not return for follow-up evaluation and ABPM. Also, providers within the participating centers may conduct follow-up differently,[4] thus, limiting the number of patients undergoing follow-up ABPM. Therefore, there is a high likelihood of selection bias. As mentioned in the methods section, we also set an upper limit of interval time between ABPM of 2 years for patients <13 years, and this may have introduced time-dependent bias. Because this study was a retrospective review of electronic health records from pediatric nephrology clinics, we did not have access to actual BP readings or measurement technique from referring providers' records. As a result, subjects were likely referred to the pediatric nephrologists and entered the study cohort in a nonstandard way. Although it might have been interesting to assess changes in target organ damage between those with stable vs changing ABPM phenotypes, this was not possible given the study design. Second, although this longitudinal cohort of youth with white coat hypertension is one of the largest to date, the sample size was small and likely reduced our power to detect small effect size differences. We note that our estimates are imprecise with wide 95% CIs in the multivariable models, and this may have been improved with a larger sample size. Our study was based on a convenience sample where study data were collected voluntarily from retrospective chart reviews at multiple Pediatric Nephrology Research Consortium centers, and thus, sample size and power calculations were not performed prior to data collection. Third, imperfect ABPM reproducibility could have contributed to a relatively high percentage of patients who progressed to abnormal ABPM phenotype. Both adult and pediatric studies have investigated reproducibility,[27-30] and overall, ABPM has superior reproducibility over clinic BP measurements in the short term.[31] However, a longitudinal study in adults showed low reproducibility for white coat hypertension and masked hypertension over a 4-year period,[32] supporting the need for periodic re-evaluation. In the previously mentioned pediatric longitudinal ABPM study, the phenotype changed in 58 of 124 children, with 16% worsening and 31% improving.[7] Whether the changes in phenotypes observed in our study were due to inherent reproducibility issues vs evolution of the individuals' phenotypes is unknown. Regardless, it is important for clinicians to perform ABPM follow-up for patients with white coat hypertension who may develop additional hypertension and cardiovascular risk factors in their adolescent and young adult years. Lastly, we acknowledge that we chose

to use age as a categorical variable (<12 years old vs 12-17 years old) rather than more biological continuous variable for the reasons described above in the method section.

Given the limitations above, future studies with a larger cohort and prospective design are needed to confirm these findings, to evaluate additional predictors of progression, and to properly determine risk factors. These studies will aid in formulating future pediatric ABPM clinical practice guidelines to define optimal follow-up frequency, methodology, and interval time.

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Glossary

ABPM	Ambulatory blood pressure monitoring
ADHD	Attention deficit hyperactivity disorder
BMI	Body mass index
BP	Blood pressure

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Table I.

The number of patients from each pediatric nephrology center

Medical centers	Number of subjects
Seattle Children's Hospital	24
University of Rochester	15
University of Texas San Antonio	9
University of Pittsburgh Medical Center Children's Hospital of Pittsburgh	8
Kaiser Permanente	8
Texas Children's Hospital	7
Nationwide Children's Hospital	6
University of Texas Houston	5
Boston Children's Hospital	4
Levine Children's Hospital	2
Brenner Children's	1

Table II.

Patient characteristics, pediatric nephrology clinic BP, and ABPM results at baseline

Patient characteristics	Median [IQR] or n (%)
Demographics and history	
Female	20 (22%)
Age 12-17 y	73 (82%)
BMI z score	1.7 [0.7-2.2]
Obesity	49 (55%)
Height percentile	72.0 [41.3-88.3]
ADHD	6 (7%)
Family history of HTN	61 (71%)
Family history of diabetes	29 (34%)
Clinic BP	
Number of patients with auscultatory BP	56 (63%)
Clinic auscultatory SBP (mm Hg)	131 [122-140]
Clinic auscultatory SBP index	1.02 [0.97-107]
Clinic auscultatory DBP	70 [62-80]
Clinic auscultatory DBP index	0.86 [0.77-0.96]
Number of patients with clinic oscillometric BP	65 (73%)
Clinic oscillometric SBP	135 [122-144]
Clinic oscillometric SBP index	1.04 [0.98-1.12]
Clinic oscillometric DBP	73 [68-77]
Clinic oscillometric DBP index	0.88 [0.83-0.95]
Stable white coat hypertension	71 (80%)
ABPM	
ABPM wake SBP index	0.89 [0.85-0.92]
ABPM wake DBP index	0.83 [0.79-0.87]
ABPM sleep SBP index	0.88 [0.85-0.91]
ABPM sleep DBP index	0.83 [0.78-0.88]
Systolic nocturnal dipping %	12.0 [8.7-16.4]
Diastolic nocturnal dipping %	20.1 [14.6-24.0]
Adequate SBP nocturnal dipping	60 (67%)
Adequate DBP nocturnal dipping	79 (89%)

DBP, diastolic blood pressure; *HTN*, hypertension; *SBP*, systolic blood pressure.

One patient had missing ADHD status; 3 patients had missing family history of HTN/diabetes.

Obesity defined by BMI z score >1.64.

Table III.

Patient characteristics, pediatric nephrology clinic BP, and ABPM results at baseline grouped by white coat hypertension status

Patient characteristics	Stable white coat hypertension (n = 71, 80%)	Intermittent white coat hypertension (n = 18, 20%)	*P* value
Female	14 (20%)	6 (33%)	.22
Age <12 y	11 (15%)	5 (28%)	.3
Age 12-17 y	60 (85%)	13 (72%)	
BMI z score	1.8 [0.7-2.2]	1.7 [0.9-2.0]	.81
Obesity	39 (55%)	10 (56%)	>.99
Height percentile	65.4 [34.6-87.3]	76.2 [70.1-94.1]	.06
ADHD	5 (7%)	1 (6%)	>.99
Family history of HTN	48 (70%)	13 (76%)	.77
Family history of diabetes	20 (29%)	9 (53%)	.09
Clinic auscultatory SBP index	1.05 [1.0-1.08]	0.92 [0.9-0.97]	<.001
Clinic auscultatory DBP index	0.87 [0.79-0.96]	0.76 [0.72-0.85]	.016
Clinic oscillometric SBP index	1.08 [1.03-1.14]	0.92 [0.9-0.97]	<.001
Clinic oscillometric DBP index	0.91 [0.84-0.96]	0.87 [0.8-0.89]	.09
ABPM wake SBP index	0.89 [0.85-0.92]	0.89 [0.87-0.91]	.65
ABPM wake DBP index	0.83 [0.77-0.87]	0.83 [0.81-0.88]	.30
ABPM sleep SBP index	0.88 [0.85-0.91]	0.89 [0.86-0.92]	.54
ABPM sleep DBP index	0.83 [0.77-0.88]	0.83 [0.79-0.88]	.65
Adequate SBP nocturnal dipping	47 (66%)	13 (72%)	.78
Adequate DBP nocturnal dipping	63 (89%)	16 (89%)	>.99

Median [IQR] or n (%).

Table IV.

Baseline patient characteristics grouped by follow-up ABPM phenotype

Patient characteristics	Hypertension (n = 20, 22.5%)	Prehypertension (n = 34, 38.2%)	Normal (n = 35, 39.3%)	*P* value
Female	3 (15%)	5 (15%)	12 (34%)	.13
Age <12 y	2 (10%)	3 (9%)	11 (31%)	.04
Age 12-17 y	18 (90%)	31 (91%)	24 (69%)	
BMI z score	1.3 [0.2-2.2]	2.0 [1.1-2.2]	1.7 [0.8-2.0]	.33
Obesity	9 (45%)	22 (65%)	18 (51%)	.35
Height percentile	75.6 [57.2-95.8]	72.5 [50.9-93.8]	65.4 [27.9-81.1]	.12
ADHD	2 (10%)	3 (9%)	1 (3%)	.64
Family history of HTN	15 (75%)	18 (56%)	28 (82%)	.07
Family history of diabetes	6 (30%)	9 (28%)	14 (41%)	.54
Clinic auscultatory SBP index	1.01 [0.98-1.04]	1.06 [0.93-1.08]	1.02 [0.98-1.09]	.79
Clinic auscultatory DBP index	0.83 [0.74-0.94]	0.85 [0.75-0.95]	0.91 [0.82-1.00]	.20
Stable white coat hypertension	17 (85%)	31 (91%)	23 (66%)	.03
Clinic oscillometric SBP index	1.05 [1.02-1.12]	1.06 [1.01-1.11]	1.01 [0.93-1.12]	.30
Clinic oscillometric DBP index	0.92 [0.82-0.94]	0.89 [0.82-0.95]	0.87 [0.83-0.96]	.90
ABPM wake SBP index	0.91 [0.84-0.93]	0.90 [0.85-0.92]	0.88 [0.85-0.90]	.16
ABPM wake DBP index	0.87 [0.82-0.89]	0.82 [0.78-0.85]	0.83 [0.78-0.87]	.06
ABPM sleep SBP index	0.90 [0.86-0.94]	0.88 [0.84-0.91]	0.88 [0.85-0.90]	.21
ABPM sleep DBP index	0.87 [0.83-0.91]	0.80 [0.76-0.84]	0.84 [0.79-0.88]	.002
Adequate SBP nocturnal dipping	14 (70%)	26 (76%)	20 (57%)	.24
Adequate DBP nocturnal dipping	19 (95%)	32 (94%)	28 (80%)	.19
Specific follow-up ABPM abnormalities				
Wake-only abnormality	2 (10%)	15 (44%)	N/A	
Sleep-only abnormality	13 (65%)	14 (41%)	N/A	
Both wake and sleep abnormalities	5 (25%)	5 (15%)	N/A	

N/A, not applicable.

Table V.

Multivariable model results of the association between baseline characteristics and follow-up ABPM phenotype

	Model A. HTN (n = 20) vs pre-HTN/Normal (n = 69)		Model B. HTN/pre-HTN (n = 54) vs normal (n = 35)	
Variables	**OR (95% CI)**	***P* value**	**OR (95% CI)**	***P* value**
Interval time between ABPM	1.05 (0.54, 2.07)	.88	0.65 (0.32, 1.32)	.23
Baseline stable white coat hypertension	1.96 (0.44, 8.33)	.38	6.67 (1.67, 25.00)	.01
Baseline age <12 y	0.52 (0.1, 2.69)	.43	0.27 (0.06, 1.11)	.07
BMI z score	0.84 (0.49, 1.44)	.53	1.18 (0.70, 1.98)	.54
Wake SBP Index ≥0.9	3.07 (1.02, 9.23)	.046	4.37 (1.35, 14.15)	.01
Sleep DBP Index ≥0.9	3.31 (0.88, 12.43)	.08		
Sleep DBP dipping ≥10%			3.6 (0.63, 20.63)	.15