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Masked hypertension and kidney function decline: the Jackson Heart Study

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Abstract

Background—Hypertension diagnosed by blood pressure (BP) measured in the clinic is associated with rapid kidney function decline (RKFD) and incident chronic kidney disease (CKD). The extent to which hypertension defined using out-of-clinic BP measurements is associated with these outcomes is unclear.

Methods—We evaluated the association of any masked hypertension (daytime SBP/DBP ≥ 135/85 mmHg, night-time SBP/DBP ≥ 120/70 mmHg or 24-h SBP/DBP ≥ 130/80 mmHg) with RKFD and incident CKD among 676 African-Americans in the Jackson Heart Study with clinic-measured SBP/DBP less than 140/90 mmHg who completed ambulatory BP monitoring in 2000–2004. RKFD was defined as a decline in estimated glomerular filtration rate (eGFR) at least 30% and incident CKD was defined as development of eGFR less than 60 ml/min per 1.73 m^2 with an at least 25% decline in eGFR between 2000–2004 and 2009–2013.

Results—The mean age of participants was 57.6 years, 28.8% were men and 52.7% had any masked hypertension. After a median follow-up of 8 years, 13.8 and 8.6% of participants had RKFD and incident CKD, respectively. In unadjusted analyses, masked hypertension was associated with an increased odds for incident CKD [odds ratio (OR) 2.20, 95% confidence interval (CI) 1.22, 3.97]. This association remained statistically significant after adjustment for demographic characteristics, baseline eGFR and albumin-to-creatinine ratio (OR 1.95, 95% CI

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Conflicts of interest

There are no conflicts of interest.

1.04, 3.67) but was eliminated after propensity score adjustment (OR 1.62, 95% CI 0.87, 3.00). There was no association between masked hypertension and RKFD.

Conclusion—Masked hypertension may be associated with the development of CKD in African-Americans.

Keywords

African-Americans; ambulatory blood pressure monitoring; chronic kidney disease; estimated glomerular filtration rate; masked hypertension; rapid kidney function decline

Ambulatory blood pressure monitoring (ABPM) can provide additional prognostic information on cardiovascular disease (CVD) risk to blood pressure (BP) measured in the clinic setting [1]. One phenotype identified using ABPM in conjunction with clinic-measured BP is masked hypertension. Initially, masked hypertension was defined by not having high clinic-measured BP (SBP <140 mmHg and DBP <90 mmHg) but having high daytime BP (SBP/DBP ≥ 135/85 mmHg) measured by ABPM [2]. Using this definition, the prevalence of masked hypertension has been reported to be 15–30% [1,3]. In 2013, the European Society of Hypertension/European Society of Cardiology recommended extending the definition of masked hypertension to include high daytime BP (mean SBP/DBP ≥ 135/85 mmHg), night-time BP (mean SBP/DBP ≥ 120/70 mmHg) and/or 24-h BP (mean SBP/DBP ≥ 130/80 mmHg) [3]. In a prior analysis of the Jackson Heart Study (JHS), a cohort comprised exclusively of African-Americans, the prevalence of masked hypertension, defined using daytime, night-time and 24-h BP, was 52.2% [4]. In two cohorts of African-Americans with established chronic kidney disease (CKD), the prevalence of masked hypertension, defined using 24-h ambulatory BP, was 27.8 and 26.0% [5,6].

Masked hypertension has been associated with an increased risk for cardiovascular target organ damage, including left ventricular (LV) hypertrophy, increased LV mass index [7], LV wall thickness, carotid intima–media thickness and pulse wave velocity [7-11]. It has also been associated with increased risk for cardiovascular and cerebrovascular events [12,13]. However, the effect of masked hypertension on the risk for CKD is less clear as prior studies have been cross-sectional or were conducted among people with established CKD [5,14,15]. In addition, there are few data on the association of masked hypertension on kidney outcomes among African-Americans. Determining the association of masked hypertension with kidney function decline and the development of CKD may lead to interventions aimed at reducing the burden of CKD. In this study, we examined the association between masked hypertension and rapid kidney function decline (RKFD) and incident CKD in the JHS.

METHODS

The JHS is a single-site, prospective, cohort study of risk factors for CVD among African-Americans residing in the Jackson, Mississippi metropolitan area. Details of the JHS have been described previously [16]. Briefly, the JHS enrolled 5306 participants from four groups in the community: participants enrolled in the Atherosclerosis Risk in Communities Study (31%), randomly selected community-dwelling adults (17%), family members of the participants (22%) and volunteers (30%). Recruitment was restricted to adults 35–84 years

old except for family members where those at least 21 years old were eligible for enrollment [17]. Different age criteria were employed for the recruitment of family members to facilitate a JHS Family Study, which was designed to identify genes influencing the risk factors for heart, lung and blood disorders. Enrollment of families was restricted to the relatives of those who had already become JHS participants [18]. In addition to completing a baseline clinic visit (exam 1) in 2000–2004, participants returned for two additional clinic visits, exam 2 (October 2005–December 2008) and exam 3 (February 2009–January 2013). The institutional review boards at the participating institutions (Jackson State University, Tougaloo College and University of Mississippi Medical Center) approved the study protocol. Written informed consent was obtained from all participants at each study visit.

Analysis population

Participants who underwent ABPM at exam 1 ($n = 1148$) were included in the current analysis. We excluded participants with an incomplete ABPM recording ($n = 102$; defined below), clinic-measured SBP/DBP at least 140/90 mmHg ($n = 202$), unknown hypertension status due to missing clinic-measured BP ($n = 5$), self-report of being on dialysis ($n = 2$) or unknown dialysis status ($n = 4$) at exam 1, or missing serum creatinine measurements at exam 1 ($n = 9$). We also excluded participants who did not attend exam 3 ($n = 140$) and those who were missing a serum creatinine measurement from this visit ($n = 8$). After these exclusions, data from 676 participants were available for the analysis of RKFD. For the analysis of incident CKD, an additional 25 participants with reduced estimated glomerular filtration rate (eGFR) (eGFR <60 ml/min per 1.73 m^2) at exam 1 were excluded, leaving 651 participants (Fig. 1).

Data collection

Exam 1 data were collected during an in-home interview and a clinic exam [19]. Information on age, sex, education, cigarette smoking, alcohol use and physical activity was collected during in-home interviews. A modified Baecke questionnaire was used to record the duration, frequency and intensity of physical activity during living, work, home life and sports [20]. Ideal health status for physical activity was defined using American Heart Association criteria as at least 75 min of vigorous physical activity or at least 150 min of moderate or combined moderate and vigorous physical activity per week.

During exam 1, trained technicians measured BP, height and weight, recorded the names of prescription and over the counter medications taken in the previous 2 weeks, and collected fasting blood and urine samples. A 24-h urine collection was requested from all participants. Beginning in October 2002, random spot urine samples were also collected during exam 1.

Biochemical testing for fasting glucose, serum and urine creatinine were performed using an enzymatic method on a Vitros 950 or 250 Ortho-Clinical Diagnosis analyzer (Raritan, New Jersey, USA). A lipid profile was assayed by oxidase method on a Roche COBAS Fara analyzer (Roche Diagnostics, Indianapolis, Indiana, USA). Hemoglobin A1c (HbA1c) was measured with a TOSOH high performance liquid chromatography system. Urinary albumin was measured on a Dade-Behring BN 11 nephelometer (Dade Behring, Newark, Delaware, USA). Among participants for whom 24-h urine sample was not collected, the random spot

urine sample was used to estimate the urinary albumin-to-creatinine ratio (UACR) [21]. Albuminuria was defined as a UACR at least 30 mg/g. Diabetes was defined as a fasting (≥8 h) plasma glucose at least 126 mg/dl, HbA1c at least 6.5% or use of antidiabetes medication.

Clinic blood pressure measurement

During exam 1, clinic BP was measured with a Hawksley random-zero sphygmomanometer (Hawksley and Sons Ltd, Langing, UK) and an appropriately sized cuff [22]. After the participant had rested for at least 5 min in a seated upright position with their back and arms supported, feet flat on the floor and legs uncrossed, two BP measurements were recorded in the right arm. The average of the two measurements recorded 1 min apart was used to define clinic BP. Quality control was conducted by JHS Coordinating Center and included monitoring digit preference for each staff member and by comparing the mean BP level measured within and between study staff. Other quality control measures included technician certification, recertification and procedural checklists [23]. As previously described, the random-zero sphygmomanometer used for BP measurements in Exam 1 was calibrated to the semiautomatic oscillometric device (Omron HEM-907XL; Omron Healthcare Inc., Lake Forest, Illinois, USA) [24] used for BP measurements at JHS Exams 2 (2005–2008) and 3 (2009–2013). To calibrate BP across two devices, a comparability substudy was conducted. This substudy included 2115 participants for which BP was assessed simultaneously by random-zero sphygmomanometer and the Omron HEM-907XL device using a Y-connector. As described elsewhere [25], the random-zero BP measurements were calibrated to the semiautomatic oscillometric device using robust regression. The calibrated clinic BP values were used for the primary analyses with non-calibrated BP used in sensitivity analysis.

Ambulatory blood pressure monitoring

Upon completion of exam 1, each participant was invited to undergo ABPM. ABPM was performed using a portable, noninvasive oscillometric device (Spacelabs 90207; Medifacts International Ltd, Rockville, Maryland, USA) with a cuff fitted to the participant's nondominant arm. The device was programed to take BP measurements every 20 min.

Trained technicians instructed participants on the general procedures and function of the ABPM device to ensure compliance and successful collection of data. Participants returned to the clinic 24 h later for the removal of the ABPM device. The ABPM device was connected to a computer and the BP recordings were downloaded using commercially available software (Medicom, version 3.41) [16,19]. Consistent with the International Database of Ambulatory Blood Pressure in relation to Cardiovascular Outcome (IDACO) criteria, we defined a complete ABPM recording as having at least 10 daytime (1000 to 2000 h) and at least five nighttime (0000 to 0600 h) BP measurements [26]. IDACO criteria were applied, rather than more stringent criteria, to include the maximum sample size available [27].

Masked hypertension

Masked daytime hypertension was defined as a mean daytime SBP/DBP at least 135/85 mmHg; masked night-time hypertension was defined as a mean night-time SBP/DBP at least

120/70 mmHg; and masked 24-h hypertension was defined as a mean SBP/DBP at least 130/80 mmHg using all BP readings taken over the ABPM recording period [3]. Participants with masked daytime, night-time or 24-h hypertension were categorized as having any masked hypertension.

Outcomes

The two outcomes included RKFD and incident CKD. Both endpoints were evaluated at exam 3 as serum creatinine was not measured at exam 2. Serum creatinine was measured using an enzymatic method at exam 1 and calibrated to the isotope-dilution mass spectrometry-traceable method used at exam 3 as previously described [28]. RKFD was defined as a decline in eGFR at least 30% from exam 1 to exam 3 [21]. Incident CKD was defined as a decline from eGFR at least 60 ml/min per 1.73 m^2 at exam 1 to eGFR less than 60 ml/min per 1.73 m^2 at exam 3 in conjunction with a decline in eGFR at least 25% over this time period [29]. eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration creatinine equation [30]. The percentage change in eGFR was calculated as 100 × (eGFR at exam 1 − eGFR at exam 3) divided by eGFR at exam 1. eGFR values more than 120 ml/min per 1.73 m^2 were truncated at 120 ml/min per 1.73 m^2 to avoid large changes among participants with high eGFR at exam 1 [31,32].

Statistical analysis

Baseline characteristics of the participants with and without any masked hypertension were calculated and compared using two-sample *t* tests for continuous variables and chi-square tests for categorical variables. The percentage of participants having RKFD and incident CKD was determined by masked hypertension status for any, daytime, night-time and 24-h masked hypertension. We calculated odds ratios (ORs) and 95% confidence intervals (CIs) for RKFD and incident CKD comparing participants with versus without any masked hypertension in the following sequential models: Model 1, unadjusted; Model 2, adjusted for age, sex and education; Model 3, adjusted for age, sex, education, eGFR and UACR; and Model 4, adjusted for a propensity score. In Model 4, we used a propensity score to simultaneously adjust for age, sex, education, eGFR, UACR, BMI, diabetes, total cholesterol, high-sensitive C reactive protein, physical activity, current cigarette smoking, alcohol use and self-reported use of antihypertensive medication given the large number of covariates relative to the number of outcome events. The sequential modeling was repeated for masked daytime, night-time and 24-h hypertension, separately. All covariates were defined using exam 1 values. Different propensity scores were developed for each type of masked hypertension using logistic regression models, with the type of masked hypertension as the dependent variable (i.e. daytime, night-time, 24-h and any masked hypertension, separately) and all covariates as independent variables. Quintiles of the propensity score were included in Model 4 in place of the values of individual covariates. The above analyses were repeated using the noncalibrated BP values. Missing covariates at exam 1 (n = 141 participants did not have UACR and an additional n = 49 participants missing other covariates) were imputed using the Markov Chain Monte Carlo method [33]. Imputation was performed for each combination of outcomes and exposures analyzed. The imputation model included the outcome variable, the exposure variable and all the covariates used to create the propensity scores. Twenty (20) imputed datasets were generated for analysis. In a final

analysis, ORs for RKFD and incident CKD were calculated stratified by diabetes status and use of antihypertensive medication at exam 1. All data analyses were conducted using SAS version 9.4 (SAS Institute, Cary, North Carolina, USA).

RESULTS

Participant baseline characteristics

Among the 676 participants included in the analysis, the mean age was 57.6 ± 10.7 years, 28.8% were men and 52.7% had any masked hypertension (masked daytime hypertension, 29.9%; masked night-time hypertension, 48.1%; masked 24-h hypertension, 33.7%). Compared with participants without any masked hypertension, those with any masked hypertension were older, more likely to be men, have diabetes and self-report taking antihypertensive medication (Table 1). In addition, participants with any masked hypertension had higher HbA1c and fasting plasma glucose, and lower eGFR levels compared with their counterparts without any masked hypertension.

Masked hypertension and rapid kidney function decline

During a median follow-up of 8.0 years (range: 5.9–12.2 years), eGFR on average declined 12.1% (median: 10.0%, interquartile range: 1.3–21.7%). Ninety-three participants (13.8%) experienced RKFD (Fig. 2, panel a). Overall, 15.5 and 11.9% of participants with and without any masked hypertension, respectively, experienced RKFD ($P = 0.178$). The unadjusted OR for RKFD comparing participants with versus without any masked hypertension was 1.36 (95% CI 0.87–2.11) (Table 2). No association was present between any masked hypertension and RKFD after full multivariable adjustment (OR, 1.07; 95% CI 0.67–1.70). The multivariable-adjusted ORs for RKFD associated with daytime, nighttime and 24-h masked hypertension were 1.23 (95% CI, 0.76–1.99), 1.24 (0.78–1.96) and 1.37 (0.86–2.18), respectively. Results were similar when using noncalibrated BP values (Supplemental Table 1, http://links.lww.com/HJH/A917).

Masked hypertension and incident chronic kidney disease

Fifty-six participants (8.6%) developed CKD during follow-up. Incident CKD occurred more often among participants with versus without any masked hypertension (11.4 versus 5.5%; $P = 0.008$) (Fig. 2, panel b). The unadjusted OR for developing CKD comparing participants with versus without any masked hypertension was 2.20 (95% CI 1.22–3.97) (Table 3). The association remained statistically significant after adjustment for age, sex, education, eGFR and UACR (OR, 1.95; 95% CI 1.04–3.67). However, this association was eliminated after propensity score adjustment (OR, 1.62; 95% CI 0.87–3.00). When masked daytime, night-time and 24-h hypertension were analyzed separately, only the association of masked night-time hypertension with incident CKD was statistically significant after adjustment for age, sex, education, eGFR and UACR (OR, 1.86; 95% CI 1.01–3.42). The fully adjusted OR (95% CI) for incident CKD association with masked daytime, night-time and 24-h hypertension were 1.34 (0.74–2.42), 1.71 (0.95–3.09) and 1.55 (0.87–2.75), respectively. Results were similar when using noncalibrated BP values (Supplemental Table 2, http://links.lww.com/HJH/A917).

Subgroup analysis

The propensity-score adjusted ORs for RKFD comparing participants with versus without any masked hypertension were 0.84 (95% CI 0.49–1.45) and 2.20 (95% CI 0.82–5.91) among participants taking and not taking antihypertensive medication, respectively (Table 4, *P* value for interaction =0.074). The propensity-score adjusted ORs for incident CKD comparing participants with versus without any masked hypertension were 1.38 (95% CI 0.66–2.88) and 3.24 (95% CI 0.91–11.5) among those taking and not taking antihypertensive medication, respectively (*P* value for interaction =0.187). The associations between any masked hypertension with RKFD and incident CKD were not statistically significantly different between participants with and without diabetes (each *P* value for interaction >0.6).

DISCUSSION

In the current study, having any masked hypertension was associated with an increased risk for incident CKD after adjustment for age, sex, education, eGFR and UACR. However, this association was eliminated after propensity score adjustment. The results were consistent for masked daytime, night-time and 24-h hypertension. No statistically significant association was present between any, daytime, night-time and 24-h masked hypertension and RKFD. Although there was a suggestion of a higher risk for RKFD and incident CKD among participants who were not taking antihypertensive medication, the differences in the ORs across subgroups defined by use of antihypertensive medication were not statistically significant. The association of masked hypertension and RKFD and incident CKD did not differ by diabetes status.

Masked hypertension has been associated with reduced eGFR and higher levels of urine protein excretion in cross-sectional studies [9] and an increased risk for end-stage renal disease, death or doubling of serum creatinine in longitudinal studies [6,34,35] of adults with established CKD. In the Ohasama study, a population-based cohort, prevalent CKD at baseline, defined as eGFR less than 60 ml/min per 1.73 m^2 and/or testing positive for proteinuria using a dipstic were more common among those with versus without masked daytime hypertension defined by a daytime SBP/DBP at least 140/85 mmHg [14]. Over a median follow-up of 8.3 years and among participants without CKD at baseline, the risk for incident CKD increased with progressively higher baseline 24-h and night-time SBP but not with higher levels of daytime or clinic SBP. The association between night-time SBP and incident CKD remained statistically significant after adjusting for daytime SBP. Similar results were present with night-time DBP and incident CKD [36]. Although the Ohasama study did not specifically evaluate the risk for incident CKD among participants with masked hypertension, the current results are consistent with the Ohasama study, suggesting a possible association between any and night-time masked hypertension and the development of CKD. Other risk factors may be mediators between masked hypertension and CKD, indicated by the elimination of this association with propensity score adjustment in the current study. Risk factors for CKD, including smoking, obesity, physical activity and psychosocial factors [37], have been associated with masked hypertension. In JHS, male sex, smoking, diabetes, antihypertensive medication use and clinic BP were associated with an increased prevalence of masked hypertension [38]. In addition, a high prevalence of obesity

and high levels of perceived stress might also explain the increased prevalence of masked hypertension in African-Americans [39,40].

Although few prospective studies have reported on masked hypertension and CKD, several cohort studies, including the JHS, have investigated the association between masked hypertension and incident CVD [10,41-45]. A meta-analysis of 7961 adults estimated the risk for CVD among participants with masked hypertension to be 2.09 (95% CI 1.55–2.81) times higher when compared with normotensive participants [41]. Similarly, in a pooled analyses of four population cohorts, Hansen *et al.* [10] showed that cardiovascular risk was higher in participants with masked hypertension, hazard ratio of 1.62 (95% CI 1.35–1.96), when compared with their counterparts with normotension. In the JHS, participants with any masked hypertension had a 2.49 (95% CI 1.26–4.93) times higher risk of CVD compared with their counterparts without masked hypertension. The risk for CVD was statistically significantly elevated among participants with masked daytime, night-time or 24-h hypertension [46]. Furthermore, night-time BP has been associated with cardiovascular mortality after adjustment for daytime BP [42]. As CVD and CKD share many risk factors, these studies provide further support for the plausibility of an association between masked hypertension and CKD. Considering that African-Americans have a high prevalence of masked hypertension and CKD and increased risk for incident CKD, the lack of association observed in the current study may indicate no association truly existing. However, this should be confirmed in other cohorts.

There are several strengths of the current study. The JHS enrolled a large community-based sample of African-Americans. This population has been well characterized, and we were able to control for multiple potential confounders. Despite these strengths, the results should be interpreted in the context of possible limitations, including the conduct of only a single ABPM procedure, which may have resulted in misclassification of participants' masked hypertension status. However, this misclassification would most likely be nondifferential (i.e. not dependent on risk for future RKFD or incident CKD). Therefore, the association between masked hypertension and RKFD and incident CKD may be stronger than we report. Only a subset of JHS participants completed the ABPM procedure. Differences were present in demographic and clinical characteristics between participants who did and did not complete the ABPM procedure [38]. Although this may limit the generalizability of the study results, the association with RKFD and incident CKD for participants with versus without masked hypertension should remain internally valid. Clinic BP was measured in a single occasion; some participants may have different clinic BP if measured on a separate day. In addition, clinic BP measured using random-zero sphygmomanometer at Exam 1 was calibrated to an oscillometric device. However, results were similar when using non-calibrated BP values. Further, interarm differences in BP may have affected the current results. Specifically, the ABPM cuff was placed on each participant's nondominant arm to minimize the effect of daily activities on readings and clinic BP was measured in the right arm. Too few participants had clinic measured BP in the hypertensive range to study the association of white-coat hypertension and renal outcomes. The JHS cohort enrolled African-Americans exclusively and the current results may not be generalizable to other racial/ethnic groups. In addition, serum creatinine levels were measured at only two time

points separated by 8 years, limiting the ability to know the actual timing and rate of decline of kidney function.

In summary, the results of the current study indicate that masked hypertension, particularly masked night-time hypertension, may be associated with an increased risk for CKD but not RKFD. Currently, data are not available to indicate whether antihypertensive medication reduces the risk for adverse renal outcomes or CVD among people with masked hypertension. As the prevalence of masked hypertension and CKD is high among African-Americans, identifying modifiable risk factors for, and evaluating treatment of, this condition may help reduce the overall burden of CKD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

ABPM	ambulatory blood pressure monitoring
BP	blood pressure
CI	confidence interval
CKD	chronic kidney disease
OR	odds ratio
RKFD	rapid kidney function decline

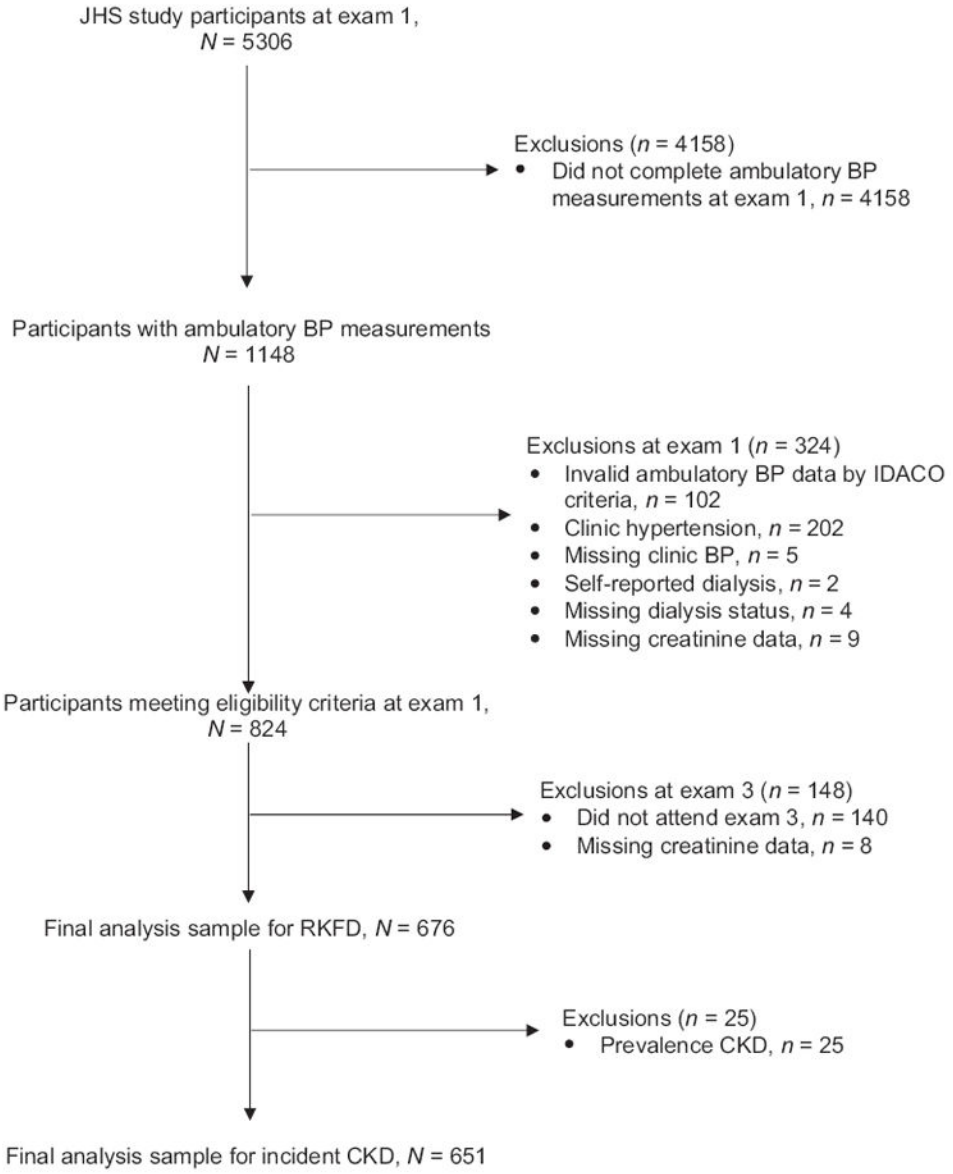

FIGURE 1.

CONSORT flow diagram for the analysis of masked hypertension and rapid kidney function decline and incident chronic kidney disease. BP, blood pressure; CKD, chronic kidney disease; RKFD, rapid kidney function decline.

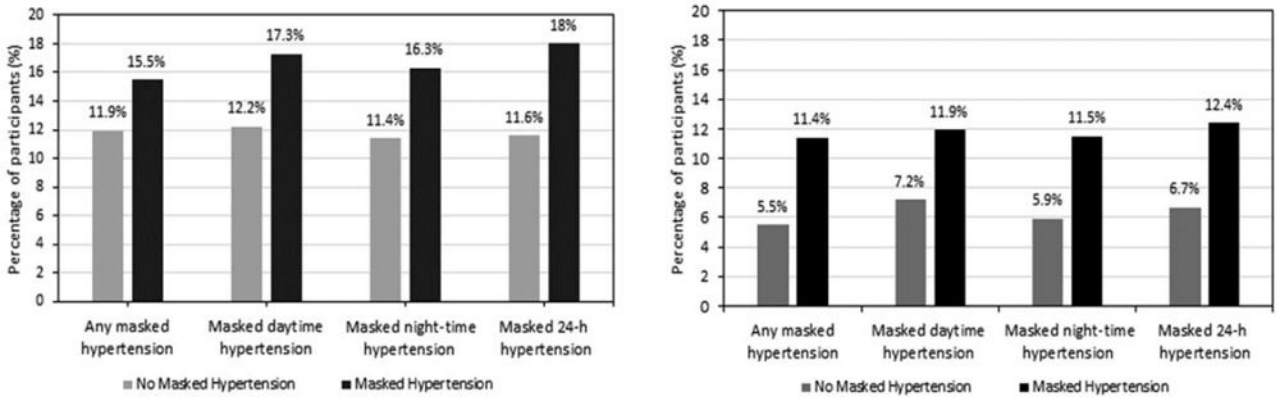

FIGURE 2.

Percentage of participants experiencing rapid kidney function decline (panel a) and incident chronic kidney disease (panel b) by type of masked hypertension. Rapid kidney function decline was defined as a decrease in estimated glomerular filtration rate at least 30% from exam 1 to exam 3. Incident chronic kidney disease was defined as a decline in estimated glomerular filtration rate from at least 60 ml/min per 1.73 m^2 at exam 1 to less than 60 ml/min per 1.73 m^2 at exam 3 with at least 25% decline during this time period.

TABLE 1

Baseline characteristics for Jackson Heart Study participants with and without any masked hypertension

	Any masked hypertension		
	No, *n* = 320	**Yes, *n* = 356**	***P* value**
Participant characteristics			
Age (years)	56.1 ± 11.1	58.9 ± 10.1	0.001
Male	74 (23.1)	121 (34.0)	0.002
Education ≥high school	275 (86.5)	295 (82.9)	0.195
BMI (kg/m^2)	31.2 ± 6.2	31.0 ± 6.3	0.693
Diabetes	47 (14.7)	95 (26.8)	<0.001
Self-reported use of antihypertensive medication use	144 (46.0)	205 (59.1)	0.001
Laboratory measures			
HbA1c	5.8 ± 1.0	6.2 ± 1.4	<0.001
Fasting plasma glucose (mg/dl)	98.1 ± 24.9	102.7 ± 31.8	0.043
Fasting total cholesterol (mg/dl)	200.3 ± 39.9	199.8 ± 40.0	0.856
Fasting HDL-C (mg/dl)	54.7 ± 14.5	54.0 ± 14.9	0.545
Fasting LDL-C (mg/dl)	124.4 ± 34.8	124.7 ± 38.7	0.922
Fasting triglycerides (mg/dl)	106.8 ± 101.9	106.8 ± 67.7	0.993
HsCRP > 3.0 mg/l	152 (47.8)	180 (50.6)	0.474
UACR[2] ≥ 30 mg	14 (5.5)	25 (9.0)	0.115
eGFR (ml/min per 1.73 m^2)	95.9 ± 20.4	92.8 ± 19.3	0.038
Behavioral factors			
Current smoking	25 (7.8)	36 (10.2)	0.277
Alcohol use (past 12 months)	152 (47.5)	162 (45.6)	0.627
Physical activity meeting AHA guidelines	74 (23.1)	64 (18.0)	0.097
Clinic BP (mmHg)			
SBP	118.2 ± 10.5	125.0 ± 9.3	<0.001
DBP	71.9 ± 6.7	74.5 ± 7.9	<0.001
ABPM, (mmHg)			
Daytime SBP	119.0 ± 7.7	133.2 ± 10.6	<0.001
Daytime DBP	72.8 ± 6.0	81.3 ± 8.6	<0.001
Night-time SBP	107.4 ± 7.3	126.4 ± 12.2	<0.001
Night-time DBP	61.1 ± 5.1	72.8 ± 8.4	<0.001
24-h SBP	114.5 ± 6.8	130.7 ± 9.8	<0.001
24-h DBP	68.1 ± 5.0	78.0 ± 7.6	<0.001

Results are presented as mean ± SD for continuous variables or number of participants (column %) for categorical variables. ABPM, ambulatory blood pressure monitoring; AHA, American Heart Association; eGFR, estimated glomerular filtration rate; HbA1c, haemoglobin A1c; HsCRP, high-sensitivity c-reactive protein; UACR, Urine albumin–creatinine ratio.

TABLE 2

Odds ratios for rapid kidney function decline associated with any, daytime, night-time and 24-h masked hypertension among Jackson Heart Study participants

		Odds ratio (95% confidence interval)			
	Events/*n* at risk (%)	**Model 1**	**Model 2**	**Model 3**	**Model 4**
Any masked hypertension					
No	38/320 (11.9)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	55/356 (15.5)	1.36 (0.87, 2.11)	1.26 (0.79, 1.99)	1.20 (0.75, 1.91)	1.07 (0.67, 1.70)
Masked daytime hypertension					
No	58/474 (12.2)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	35/202 (17.3)	1.50 (0.95, 2.37)	1.44 (0.89, 2.30)	1.29 (0.79, 2.10)	1.23 (0.76, 1.99)
Masked night-time hypertension					
No	40/351 (11.4)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	53/325 (16.3)	1.52 (0.97, 2.36)	1.41 (0.89, 2.23)	1.35 (0.85, 2.14)	1.24 (0.78, 1.96)
Masked 24-h hypertension					
No	52/448 (11.6)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	41/228 (18.0)	1.67 (1.07, 2.61)	1.59 (1.00, 2.52)	1.46 (0.91, 2.35)	1.37 (0.86, 2.18)

Model 1: unadjusted. Model 2: adjusted for age, sex and education. Model 3: adjusted for age, sex and education, estimated glomerular filtration rate (eGFR) and urine albumin–creatinine ratio (UACR). Model 4: adjusted for quintiles of the propensity score for having masked hypertension. Propensity score quintiles were calculated for each type of masked hypertension, separately, using logistic regression models with the type of masked hypertension as the dependent variable and age, sex, education, eGFR, UACR, BMI, diabetes, high-sensitivity c-reactive protein, total cholesterol, physical activity, current cigarette smoking, alcohol intake and antihypertensive medication use as independent variables. RKFD, rapid kidney function decline.

TABLE 3

Odds ratios for incident chronic kidney disease associated with any, daytime, night-time and 24-h masked hypertension among Jackson Heart Study participants

		Odds ratio (95% confidence interval)			
	Events/*n* at risk (%)	**Model 1**	**Model 2**	**Model 3**	**Model 4**
Any masked hypertension					
No	17/308 (5.5)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	39/343 (11.4)	2.20 (1.22, 3.97)	1.94 (1.05, 3.59)	1.95 (1.04, 3.67)	1.62 (0.87, 3.00)
Masked daytime hypertension					
No	33/457 (7.2)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	23/194 (11.9)	1.73 (0.99, 3.03)	1.52 (0.84, 2.72)	1.55 (0.84, 2.85)	1.34 (0.74, 2.42)
Masked night-time hypertension					
No	20/339 (5.9)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	36/312 (11.5)	2.08 (1.18, 3.68)	1.82 (1.01, 3.30)	1.86 (1.01, 3.42)	1.71 (0.95, 3.09)
Masked 24-h hypertension					
No	29/433 (6.7)	1 (ref)	1 (ref)	1 (ref)	1 (ref)
Yes	27/218 (12.4)	1.97 (1.13, 3.42)	1.70 (0.96, 3.03)	1.71 (0.94, 3.11)	1.55 (0.87, 2.75)

Model 1: unadjusted. Model 2: adjusted for age, sex and education. Model 3: adjusted for age, sex and education, estimated glomerular filtration rate (eGFR) and urine albumin–creatinine ratio (UACR). Model 4: adjusted for quintiles of the propensity score for having masked hypertension. Propensity score quintiles were calculated for each type of masked hypertension, separately, using logistic regression models with the type of masked hypertension as dependent variable and age, sex, education, eGFR, UACR, BMI, diabetes, high-sensitivity c-reactive protein, total cholesterol, physical activity, current cigarette smoking, alcohol intake and antihypertensive medication use as independent variables. CKD, chronic kidney disease.

TABLE 4

Association between any masked hypertension with rapid kidney function decline and incident chronic kidney disease stratified by antihypertensive medication use and by diabetes status at baseline

	RKFD		Incident CKD	
Subgroups	**Propensity score-adjusted OR (95% CI)**	***P* value for interaction**	**Propensity score-adjusted OR (95% CI)**	***P* value for interaction**
Self-reported antihypertensive medication use				
Yes	0.84 (0.49, 1.45)	0.074	1.38 (0.66, 2.88)	0.187
No	2.20 (0.82, 5.91)		3.24 (0.91, 11.5)	
Diabetes status				
Yes	1.20 (0.52, 2.80)	0.606	1.54 (0.47, 5.07)	0.949
No	0.94 (0.53, 1.65)		1.61 (0.79, 3.29)	

Due to small number of cases in the stratified analyses, the propensity score was included in the model as a continuous variable. Propensity score was calculated for each type or masked hypertension using logistic regression models, with the any masked hypertension as the dependent variable and age, sex, education, estimated glomerular filtration rate (eGFR), urinary albumin–creatinine ratio (UACR), BMI, diabetes, high-sensitivity c-reactive protein (HSCRP), total cholesterol, physical activity, current cigarette smoking, alcohol intake and use of antihypertensive medication as independent variables. CI, confidence interval; CKD, chronic kidney disease; OR, odds ration; RKFD, rapid kidney function decline.