What Is the Significance of Masked Hypertension Versus Incident Hypertension in Blacks?

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Treatment-naive masked hypertension has been defined as discordant in-office normotension (<140/<90 mm Hg) versus out-of-office hypertension (≥135/85 mm Hg) for either home blood pressure (BP) monitoring (HBPM) or ambulatory BP monitoring (ABPM) of daytime ABPM (≥135/85 mm Hg), night-time ABPM (≥120/70 mm Hg), 24-hour ABPM (≥130/80 mm Hg), or a combination of these out-of-office BP subtypes. Masked hypertension may be a precursor of sustained hypertension, remain masked hypertension for a prolonged period of time, and occasionally revert to normotension.1 However, there are at least 5 other aspects of masked hypertension of clinical importance: (1) it has been considered an intermediate phenotype between sustained normotension and sustained hypertension, which most frequently arises from antecedent high-normal BP (130–139/85–89 mm Hg), less frequently from antecedent normal BP (120–129/80–84 mm Hg), and least frequently from antecedent optimal BP (<120/<80 mm Hg);2 (note that normal and high-normal BP when combined are defined as concordant in-office normotension (<140/<90 mm Hg); 24-hour ABPM ≥130/80 mm Hg); (2) it frequently is associated with hypertensive target organ damage even without progressing to hypertension, perhaps because of increased pressure burden in daily life, and despite a long-dormant period before transitioning to sustained hypertension;3 (3) it has been associated with many cardiometabolic abnormalities, including obesity, metabolic syndrome, diabetes mellitus, obstructive sleep apnea, and chronic renal disease;4 (4) it frequently has been associated with nocturnal hypertension and impaired nocturnal dipping of BP—a particularly high-risk phenotype;5 and (5) there is an especially high prevalence of masked hypertension in people of African descent, approaching 30% to 50% in some series.3,6

It is with this background that the present investigators used ABPM to define masked hypertension as a possible precursor of incident clinic hypertension in 317 treatment-naive participants from the Jackson Heart Study, a prospective cohort of blacks.7 The mean age of this group was 54.8 years, mean body mass index was 30.1 U, prevalence of diabetes mellitus was 11.7%, any masked hypertension subtype was 45.4%, and the presence of left ventricular hypertrophy was 11.1%. Incident clinic hypertension was defined as the first visit with either clinic systolic/diastolic BP ≥140/90 mm Hg or initiation of antihypertensive medication. With a median follow-up of 8.1 years, representing study visits in 2005 to 2008 and 2009 to 2012, there were 187 (59%) incident cases of clinic hypertension: 79% and 42% of participants with and without any masked hypertension, respectively. Thus, the major new finding is that individuals with any subtype of masked hypertension—daytime, nighttime, or 24-hour ABPM—had twice the risk for developing incident clinic hypertension when compared with those with antecedent sustained normotension. These proportions between masked hypertension and sustained normotension were generally consistent when assessing daytime, nighttime, or 24-hour ABPM; but, after multivariable-adjustment, nighttime masked hypertension had a somewhat greater predictive power for incident clinic hypertension. Using any definition of masked hypertension, 51.9% of participants with prehypertension and 14.7% of those with normal clinic BP (often referred to as optimal BP in other studies), developed incident hypertension during the 8.1-year follow-up. Multivariable-adjusted hazard ratios were significant for incident clinic hypertension in any masked hypertension subtype. Finally, after stratifying individuals by prehypertension versus normal clinic BP, the association between masked hypertension and incident clinic hypertension remained significant in both groups. The authors suggested that preventive strategies, that is, lifestyle modification might be useful to prevent progression of masked hypertension to clinic hypertension not only in those with prehypertensive clinic BP but even in those with optimal clinic BP. However, if evidence of target organ damage such as left ventricular hypertrophy was present, antihypertensive treatment might be a particularly important consideration. Furthermore, this study should be interpreted within the context of its limitations.

Role of Nocturnal Hypertension

There is an evidence that nocturnal hypertension plays an important role in the development of masked hypertension, particularly in blacks. In a previous Jackson Heart Study that included 425 untreated normotensive and hypertensive individuals undergoing ABPM, masked hypertension was associated with isolated nocturnal hypertension in 19% of untreated individuals; these subjects had a greater prevalence of left ventricular hypertrophy than their normotensive counterparts in age- and sex-adjusted models.1 In a more recent Jackson Heart Study, conducted in 972 blacks, masked hypertension was noted in 34.4% of participants with normal clinic BP.4 Male sex, smoking, diabetes mellitus, and antihypertensive...
Role of Lifestyle Intervention in Masked Hypertension

The authors advocated lifestyle intervention for masked hypertension in the absence of target organ damage, to prevent its progression to incident hypertension. Importantly, a lifetime risk study showed that primordial lifestyle intervention in young adults was more effective in preventing future cardiometabolic events than primary prevention in older adults. By the same token, lifestyle intervention in this study would be more effective if begun earlier to prevent the development of masked hypertension.

The major strengths of this study include both the application of ABPM to a sizeable cohort of black individuals and the novelty and potential importance of the above findings. The observational data underscore the urgent need for the first proper randomized controlled trial to address the nagging question: does medication management of masked hypertension prevent heart attacks, strokes, and other major cardiovascular and renal complications of hypertension?

Disclosures

None.

References

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