

Postural Hypertension in Elderly Men- The Honolulu Heart Program

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Abstract

Postural hypertension is a condition in which the blood pressure rises abnormally during standing. The aim of this study is to determine the prevalence of postural hypertension and its association with morbidity and subsequent mortality. At the fourth examination (1991-1993) of the Honolulu Heart Program longitudinal cohort, 3741 Japanese-American men aged 71-93 were examined. Prevalence of postural hypertension in this cohort was 39%. Although there was a high prevalence of postural hypertension in this cohort, its clinical significance is questionable as there were no significant correlations with morbidity and subsequent mortality.

Introduction

Postural hypertension or orthostatic hypertension is a condition in which the blood pressure rises during standing. There are only a few case reports and studies documenting this condition and it is not well recognized. The definition of orthostatic hypertension is not yet standardized. One study defines it as an increase in diastolic blood pressure from below 90 to above 90 mm Hg on standing.¹ Another study defines it as an increase in systolic blood pressure by 10 mm Hg or more by the tilt test,² and another by marked hypertension in the upright position with normal pressure in the supine position.³ Kozo et al define it as an elevation of systolic BP >20mm Hg at 2 minutes after standing.⁴

Postural hypertension was first reported by Schneider et al in 1922, in 4.2% of 2000 apparently healthy airmen examined after World War I.⁵ The prevalence of postural hypertension varies. In one study, orthostatic hypertension was found in 71% (15 of 21 patients) of borderline hypertensives, while another study had a prevalence 8.7% (29 of 334).⁴ Streeten et al showed that as many as 10% of some hypertensive populations may have orthostatic hypertension.¹

Various mechanisms for postural hypertension have been suggested. One study showed that decreased venous return causes decreased cardiac output, increased sympathetic stimulation and excessive arteriolar constriction.¹ Other studies showed that patients have increased vascular adrenergic activity⁶ and excessive increase in peripheral vascular resistance during tilting.^{7,8} Therefore, the exact pathogenic mechanisms of postural hypertension are not clear, even though abnormalities of autonomic nervous system activity may be etiologically related.

Various studies have looked at the clinical significance of postural hypertension. One study demonstrated that neurobehavioral functioning and activities of daily living were significantly lowered in people with postural hypertension. Additionally, more periventricular hyperintensities or advanced leukoaraiosis were seen on MRI of the brain when compared with a normotensive group.³ In another study, an association with extreme dipping (marked fall in nocturnal blood pressure) and cerebrovascular disease has been demonstrated.^{2,9}

The aim of this analysis is to determine the prevalence of postural hypertension in elderly men of the Honolulu Heart Program longitudinal cohort and to study the association with morbidity and subsequent mortality.

Methods

Study Population

The Honolulu Heart Program is a longitudinal cohort study of cardiovascular disease which began in 1965. Participants were men of Japanese ancestry, living on the island of Oahu, Hawaii in 1965, and born between 1900 and 1919. The first examination was performed between 1965-1968 and 8006 men were examined. The entire cohort has undergone 6 examinations so far. At the fourth examination (1991-1993), 3741 men aged 71-93 years were examined, representing 80% of the survivors. For this analysis, all variables of interest were available in 3522 participants. The study was approved by the institutional review board of Kuakini Medical Center, and procedures followed were in accordance with institutional guidelines.

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Data Collection

The fourth examination included demographic questions, medical and psychosocial interview assessment of cognitive function, fasting blood and 2hour GTT, EKG, anthropometry, blood pressures (seated, ankle-arm ratio, orthostatics). Diabetes mellitus was defined by history (as told to the participant by a physician), or by fasting glucose ≥ 140 mg/dl or by 2 hour post prandial glucose ≥ 200 mg/dl. Body mass index (BMI) was calculated as weight (kg)/height (square meter).

Morbidity and mortality surveillance by the monitoring of hospital discharge records and death certificates has been performed since the beginning of the study. For this report, mortality data were accumulated through December 1997. Data collection is believed to be essentially complete for all cause mortality. Attrition in this cohort is known to be very small; at the fourth examination, only 5 men were lost to follow-up.

Seated blood pressure was measured twice with a mercury sphygmomanometer using a standardized protocol. Supine measurements taken after at least 15 minutes of supine rest. Standing measurements were taken 3 minutes after standing.

Definition of Postural Hypertension

Normally on standing, there is a fall in systolic BP of 10 mm Hg and a rise in diastolic BP of 2-3 mm Hg.¹⁰ Postural hypertension is a syndrome in which the blood pressure (systolic or diastolic) rises by 10 mm Hg or more on standing.

Statistical Methods

Subjects with postural hypotension were excluded. We compared subjects with and without postural hypertension. We studied cross sectional associations of postural hypertension with age, BMI, smoking status, seated hypertension, abnormal ankle brachial index, diabetes, prevalent CHD, CVA and cognitive impairment, and subsequent 6 year mortality.

Data are expressed as means \pm standard deviation (SD) and as percentages. Univariate and multivariate logistic regression models were used to calculate Odds Ratios (OR) and 95% confidence interval (CI) to study cross-sectional associations. Cox proportional hazards models were used to study the association with subsequent 6-year mortality.

Results

Prevalence of postural hypertension in this cohort was 39%. Prevalence of postural hypertension was 38% between 71-74 years of age, 38.9% between 75-79 years of age, 41.5% between 80-84 of age, and 37.5% in persons over 85 years of age. (Fig. 1). There was no significant difference in prevalence rates between the 5 year age groups (chi square $p = 0.522$). The baseline characteristics of variables by postural hypertension status are shown in Table 1. There was a significant difference in prevalence of seated hypertension, 76.8% of those with postural hypertension had seated hypertension compared to 73% in those without postural hypertension. BMI is also significantly associated with postural hypertension. There were no significant differences among those with and without postural hypertension among variables like age, smoking status, abnormal ankle-brachial index (ABI), prevalent diabetes, prevalent CHD, prevalent CVA, and cognitive impairment.

Figure 1.— Prevalence of postural hypertension by 5-year age groups in the Honolulu Heart Program Cohort (n=3522). Chi square $p=0.522$.

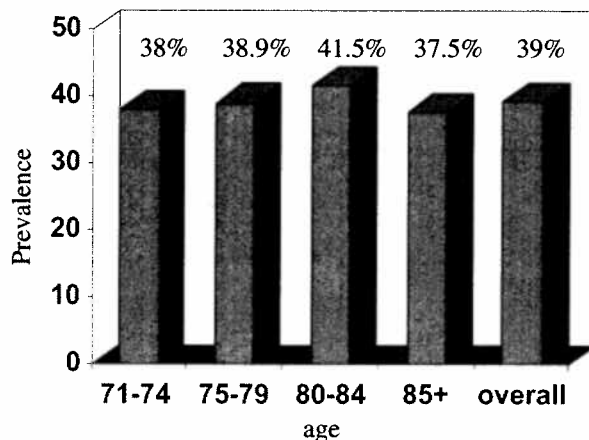


Table 1.— Baseline characteristics of variables by Postural Hypertension Status

Variables	Postural Htn Present	Postural Htn Absent
Age (years)	77.6 (+4.4)	77.5 (+4.5)
BMI*	23.4 (+3.1)	23.7 (+3.2)
Smoking Status	7.41%	7.60%
Seated Htn.*	76.84%	73%
Abnormal ABI	11.89%	13.20%
Prevalent Diab.	28.17%	28.10%
Prevalent CHD	17.45%	15.90%
Prevalent CVA	5.24%	4.50%
Cognitive Imp.	12.28%	12.40%

Continuous Variables- Means (\pm SD), t tests

Categorical Variables-Percentages, chi square analysis

* $p < 0.05$

Htn.= Hypertension

Diab.= Diabetes

Univariate logistic regression models showed that postural hypertension was significantly associated with seated hypertension (OR 1.23, 95% CI 1.04-1.45) and BMI (OR 1.03, 95% CI 1.002-1.05). There were no significant associations with age, prevalent coronary artery disease, stroke, cognitive impairment, diabetes, abnormal ankle-brachial index and smoking status. (Table 2).

We performed multivariate logistic regression analysis (Table 3) with postural hypertension as a dependent variable and age, BMI and seated hypertension as independent variables. Postural hypertension was significantly associated with BMI (OR 1.025, 95% CI 1.002-1.050) and seated hypertension (OR 1.192, 95% CI 1.011-1.405), but not with age.

Two separate Cox proportional hazards models were performed with 6 year all cause mortality as the end point (Table 4). Postural hypertension showed no significant association with 6 year all cause

mortality. In the first model, the relative risk (RR) for all cause mortality associated with orthostatic hypertension was 0.95 (95%CI 0.82- 1.10) after adjustment for age alone. Adjustment for other factors like seated hypertension and BMI (model 2) also did not appreciably alter the results (RR=0.97; 95%CI 0.83-1.12).

Table 2.— Univariate Logistic Regression Analysis, with Postural Hypertension as the dependent variable

Variables	Odds Ratio (95% CI)
BMI	1.030 (1.002-1.050)*
Smoking	1.018 (0.911-1.159)
Seated Hypertension	1.230 (1.040-1.450)*
Abnormal ABI	0.886 (0.716-1.048)
Prevalent Diabetes	0.995 (0.878-1.038)
Prevalent CHD	1.189 (0.859-1.646)
Previous CVA	1.189 (0.859-1.646)
Cognitive Impairment	0.986 (0.797-1.220)

* p < 0.05

Table 3.— Multivariate Logistic Regression Analysis, with Postural Hypertension as the dependent variable

Variables	Odds Ratio
Age	1.009 (0.993-1.026)
BMI	1.025 (1.002-1.050)*
Seated Hypertension	1.192 (1.011-1.405)*

* p < 0.05

Table 4.— Cox Proportional Hazards models for association between Postural Hypertension and 6-year all-cause mortality

Models	Relative Risk	95% CI
Model 1 (Postural hypertension adjusted for age)	0.95	0.82-1.10
Model 2 (Postural hypertension adjusted for age, seated hypertension, and BMI)	0.97	0.83-1.12

Discussion

Physiological mechanisms that ensure fairly small oscillations in blood pressure, with changes in posture are well known. Postural or orthostatic hypotension is commonly encountered and its physiology has been well characterized. The opposite, orthostatic or postural hypertension, has received scant attention and only few case reports and studies have been done.¹⁻⁴ Its clinical significance in the elderly population also remains unclear.

Orthostatic hypertension has not been well defined. Sapru et al defined orthostatic hypertension as marked hypertension in the upright position with normal pressures in the supine position.³ Streeten et al defined orthostatic hypertension as an increase in diastolic BP from below 90 to above 90 mm of Hg.¹ Matsubayashi et al defined orthostatic hypertension as an elevation of SBP \geq 20 mm Hg at 1 or 2 minutes after standing⁴ and Kario et al defined it as an increase in systolic BP by 10 mm Hg by tilt test.⁹ In our study we defined postural hypertension as change in SBP or DBP \geq 10 mm Hg on standing after 3 minutes.

Our cross-sectional data showed that the prevalence of postural hypertension in this cohort is 39%. Postural hypertension is significantly associated with seated hypertension and BMI. There were no significant associations with other variables, prevalent diseases and 6 year all-cause mortality.

There are several strengths to this study. It is population-based, and the numbers are large. It is the only large study done in an elderly population, with almost complete follow-up for morbidity and mortality outcomes.

There are some limitations to this study. Seated blood pressure was measured only twice and not multiple times. Orthostatic blood pressure was measured once. Since blood pressure values are known to vary with repeated measurements multiple measures would be preferable. Secondly, morbidity associations that were studied were cross-sectional. Thirdly, there are no data on cause specific mortality in this analysis. Lastly data reported here can only be generalized to an ambulatory male population, since there were no women in the cohort and orthostatics were not measured at home visits or nursing home visits.

In conclusion, even though there is a high prevalence of postural hypertension in this population of elderly Japanese-American men, its clinical significance is questionable as there were no significant correlations with morbidity and subsequent mortality.

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