Orthostatic hypertension: profile of a Nigerian population

Imarhiagbe FA, MBChB, FMCP
Formerly of the Department of Medicine, Federal Medical Centre, Owo, Ondo State, Nigeria
Kubeyinje EP, MBBS, FRCP
Department of Medicine, University of Benin Teaching Hospital, Benin City, Nigeria
Correspondence to: Frank Aiwansoba Imarhiagbe, e-mail: dnimei@yahoo.com
Keywords: orthostatic hypertension, prevalence, BP, Nigerians

Abstract

Background: The aim of this study was to determine the prevalence, age, sex distribution and blood pressure (BP) pattern of patients with orthostatic hypertension in a cohort of hypertensives.

Method: A total of 179 patients on follow-up treatment in a hypertension clinic were assessed for age, sex and BP in the seated position, and after two minutes in the erect position, on three consecutive visits. Orthostatic hypertension was defined as an increase in systolic blood pressure (SBP) of ≥ 20 mmHg on more than one occasion in the erect position. Orthostatic hypotension was defined as a decrease in BP on more than one occasion, between the seated and erect SBP, of ≥ 20 mmHg. The mean ages of the participants with and without orthostatic hypertension were compared by t-test for any significant difference. The means of the seated SBP of participants with and without orthostatic hypertension were also compared with the t-test. The effect of gender on orthostatic hypertension was tested with a chi-square (χ^2). The differences between the mean seated and mean erect SBPs of participants with and without orthostatic hypertension were compared with the paired t-test.

Results: Thirty-eight (21.23%) of the participants had orthostatic hypertension. The mean age of those with orthostatic hypertension was not significantly different from that of the participants without orthostatic hypertension (p-value = 0.789). There was no significant effect of gender on orthostatic hypertension (p-value = 0.795). The mean of the seated SBP was significantly lower in the participants with orthostatic hypertension (p-value = 0.008). The mean seated SBP was significantly different from the mean erect SBP for those with orthostatic hypertension, compared to those without orthostatic hypertension (p-value = 0.000 vs. p-value = 0.169). Five (2.79%) of the participants had orthostatic hypotension.

Conclusion: Orthostatic hypertension, a form of BP dysregulation, may be more common among treated hypertensives than what is presently known.

Ⓟ **Peer reviewed. (Submitted: 2010-07-27, Accepted: 2010-09-29). © SAFP** **S Afr Fam Pract 2011;53(2):161-164**

Introduction

Orthostatic hypertension is an increase in blood pressure (BP) in the erect position from that measured in the seated or supine position. The subject of orthostatic hypertension (unlike orthostatic hypotension which is well known) is seldom discussed and so there is a relative dearth of literature on it. There appears to be no conventional definition of orthostatic hypertension at the moment, therefore most of the definitions have been operational within study designs. However, what is consistent among these definitions is the increase in BP in the erect position. Orthostatic hypertension has attracted attention largely because the few available studies have associated it with diverse clinical conditions such as essential hypertension, silent intracerebral infarcts, cognitive decline, type 2 diabetes mellitus and increased target organ damage in individuals who are hypertensive. Although the exact pathophysiology of orthostatic hypertension has not yet been elucidated completely, overcompensation of the sympathetic alpha adrenergic baroreflex mechanism response to the haemodynamic effect of the erect posture is believed to be central to it.

In this preliminary study, we describe the basic characteristics of orthostatic hypertension in a cohort of hypertensive Nigerians.

Method

One hundred and seventy-nine (179) patients on follow-up treatment in a hypertension clinic at the Federal Medical

Centre, Owo, a semi-urban setting in south-west Nigeria, were consecutively assessed for demographic variables of age, sex, occupation and domicile, and for BP in the seated position and after two minutes in the erect position on three consecutive visits over a period of 12 weeks, regardless of the antihypertensive medication they were on. BP was recorded manually with a standard Accoson® mercury sphygmomanometer between the hours of 09h00 and 12h00 by one of the investigators. The left arm was used, and the sphygmomanometer mercury column was placed at arm level, in both the seated and erect positions, to mitigate the effect of posture and gravity. The time interval between the two positions was determined with a stopwatch. All the study participants were asked to empty their bladders and rest for at least 30 minutes prior to the BP checks. Potential stimulants, such as coffee and cigarette smoking, were avoided on the day of evaluation. Orthostatic hypertension was defined as an increase in systolic blood pressure (SBP) of ≥ 20 mmHg in the erect position on more than one occasion. Orthostatic hypotension was defined as a decrease in BP between seated and erect SBP of ≥ 20 mmHg on more than one occasion.

Exclusion criteria for the study were clinically manifested renal failure, or a serum creatinine level of above 2 mg/dl, systemic corticosteroid therapy and diabetes mellitus. The study was approved by the ethics committee (Institutional Review Board) of the Federal Medical Centre, Owo.

Statistics

Mean age, sex distribution, prevalence of orthostatic hypertension and orthostatic hypotension were described in a frequency table (see Table I). Orthostatic hypertension was computed as the mean of the orthostatic increases in BP ≥ 20 mmHg, and orthostatic hypotension as the mean of the orthostatic decreases in BP ≥ 20 mmHg. The mean ages of the participants with and without orthostatic hypertension were compared by means of an independent t-test for any significant differences. The mean of the seated SBPs of the participants with and without orthostatic hypertension were also compared with an independent t-test. The mean of the seated and erect BPs of the participants with and without orthostatic hypertension were compared with a paired t-test for any significant differences. The effect of gender on orthostatic hypertension was tested with a chi-square (χ^2). Analyses were done with IBM® SPSS version 16, and p-value ≤ 0.05 was taken as significant.

Results

The lower mean seated SBP of the participants with orthostatic hypertension compared to the participants without orthostatic hypertension is noted, and suggests that an orthostatic increase can occur regardless of the seated BP.

Table I: Baseline characteristics of study participants

	Orthostatic hypertension	No orthostatic hypertension
Number of participants (n)/total	38/179	141/179
% of total	21.23%	78.77%
Mean age ([a]SD)	61.00 (14.17) years	60.38 (12.13) years
Sex (female/male)	24/14	83/58
Mean of the seated [b]SBP (SD)	129.47 (23.47) mmHg	140.22 (21.47) mmHg
Mean of the erect SBP (SD)	150.78 (24.42) mmHg	141.21 (21.95) mmHg
Number with orthostatic hypotension (%)	Not applicable	5 (2.79%)

[a] = standard deviation
[b] = systolic blood pressure

The preponderance of participants without orthostatic hypertension compared to those with orthostatic hypertension is shown in Figure 1.

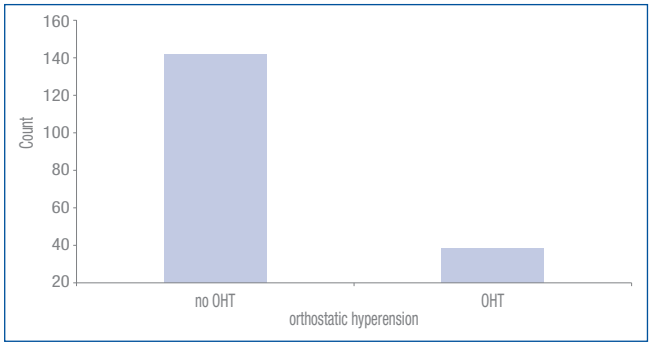

Figure 1: Bar chart showing the distribution of participants with and without orthostatic hypertension

No significant difference was found between the mean ages, as shown in Table II.

Table II: The independent t-test results of the mean ages of the participants with and without orthostatic hypertension

	Levene's test for equality of variances		T-test for equality of means		
	F	Significance	T-test	Degrees of freedom	Significance (Two-tailed)
Age (Equal variances assumed)	1.169	0.281	0.268	177	0.789

No significant effect of gender was found on orthostatic hypotension, as is shown in Table III.

Table III: Chi-square showing the effect of gender on orthostatic hypertension

	Value	Degrees of freedom	Significance (Two-sided)
Pearson chi-square	0.459	2	0.795
Number of valid cases	179		

The significant differences between the means of the seated SBPs are shown in Table IV.

Table IV: Independent t-test results comparing the means of the seated SBP of the orthostatic hypertension and non-orthostatic hypertension participants

	Levene's test for equality of variances		T-test for equality of means		
	F	Significance	T-test	Degrees of freedom	Significance (Two-tailed)
Sitting [a]SBP (Equal variances assumed)	0.215	0.643	-2.685	177	0.008

a = systolic blood pressure

The significant differences between the means of the seated and erect SBPs of the participants with orthostatic hypertension are shown on paired t-test statistics in Table V.

No significant differences were found between the mean of the seated and erect SBPs on a paired t-test in participants without orthostatic hypertension (see Table VI).

Discussion

Orthostatic hypertension is the increase in BP that arises when assuming an erect position, after having had a BP measurement taken while in the seated or supine position. On assuming an erect position, BP normally drops insignificantly due to the effect of microgravity, which causes the pooling of about 10% of the blood volume in the lower extremities.[1] Following orthostasis, the resultant decrease in the thoracic blood volume excites the baroreceptor sympathetic mechanisms to compensate for the volume reduction with an increase in the heart rate and peripheral vascular resistance; the effect of which is a negligible decrease in BP in healthy individuals.[1,2] With orthostatic hypertension, there is a significant increase in the BP on assuming the erect position, due partly to baroreceptor overcompensation, although the exact pathophysiology is not yet completely clear.[3] Because the orthostatic increase in BP could be abolished with alpha adrenergic blocking agents, alpha adrenergic activity has been implicated as the predominant pathophysiological mechanism of orthostatic hypertension.[4] At present, there is no standard definition of orthostatic hypertension with regard to the margin of BP increase, and as such, the only available definitions have been operational within study designs. Although the clinical significance of orthostatic hypertension in hypertensives is still not fully known, it has been associated with diverse clinical conditions, ranging from silent cerebral infarcts, and cognitive decline, to target organ damage.[5,6] In normotensive subjects, orthostatic hypertension has been found to be highly predictive of essential hypertension, so much so that it has been described as a prehypertension state.[7] Like isolated systolic hypertension and orthostatic hypotension, orthostatic hypertension has generally been associated with the elderly, early-stage neuropathy and sustained hypertension in type 2 diabetes, morning hypertension, increased neurohumoral activity, and greater cardiac burden in hypertensive patients.[6,7,8]

In this study, we noted that the mean age of those with orthostatic hypertension did not differ significantly from those without orthostatic hypertension (61 ± 14.17 vs. 60.38 ± 12.13, p-value = 0.789). The gender distribution of participants with and without orthostatic hypertension showed a similar trend of female preponderance (24 vs. 14, n = 38 and 83 vs. 58, n = 141). Thirty-eight (21.22%) of the 179 participants who were studied had orthostatic hypertension. A similar study found an 11% prevalence.[3]

In a related population-based study on the prevalence and correlates of orthostatic hypotension and orthostatic hypertension, none of the participants under the age of 40 years had orthostatic hypertension. The prevalence of orthostatic hypertension was 1.1%, and age was found to correlate significantly with orthostatic hypertension.[9] Another population-based study found a prevalence as high as 30%, but this might reflect the difference in the design of the studies, particularly in terms of the definition of orthostatic hypertension.[10] The prevalence of orthostatic hypertension in this study might have been influenced by antihypertensive medications, particularly the alpha adrenergic receptor blockers.[4]

Table V. Paired t-test results of the means of the seated and erect SBPs of participants with orthostatic hypertension

	Paired differences (mean)	Standard deviation	T-test	Degrees of freedom	Significance (two-tailed)
Seated [a]SBP Erect SBP	-21.3158	3.4257	-38.357	37	0.000

[a] = systolic blood pressure

Table VI. Paired t-test results of the mean of the seated and erect SBPs of participants without orthostatic hypertension

	Paired differences (mean)	Standard deviation	T-test	Degrees of freedom	Significance (two-tailed)
Sitting [a]SBP Standing SBP	-0.9929	8.5210	-1.384	140	0.169

[a] = systolic blood pressure

The contribution of gender to orthostatic hypertension was also not significant (χ^2 = 0.459, p-value = 0.795). Although orthostatic intolerance has been associated with women more than with men, a related study involving angiotensin-converting enzyme (ACE) genetics in orthostatic hypertension found no difference between males and females with regard to orthostatic hypertension rates.[11,12] Remarkably, the mean seated SBP of the participants with orthostatic hypertension was significantly lower than that of those without orthostatic hypertension (p-value = 0.008), and the difference between the mean seated and erect SBPs in participants with orthostatic hypertension, compared to those without orthostatic hypertension, shown on paired t-test statistics, was also significant (p-value = 0.000 vs. p-value = 0.169). These results suggest that there can be significant orthostatic increases, even at normotensive levels of BP. They may also suggest that unless an orthostatic increase in BP is routinely investigated, this form of BP dysregulation may be missed, particularly in hypertensives with controlled BP. Only five participants, representing 2.79% of the total sample, had orthostatic hypotension.

We recognise the fact that the participants were exposed to diverse classes of antihypertensive medications and that these might have had a possible influence on the changes in orthostatic BP, and in particular, the alpha adrenergic blockers. It bears reiteration that orthostatic hypertension is in dire need of research, both in the elucidation of its exact pathophysiology and clinical significance, and also with regard to a consensus definition to enable comparative studies across different population groups. We conclude that orthostatic hypertension may be more common among hypertensives than previously thought, and as knowledge of this seldom-discussed form of BP dysregulation increases, it may influence the choice of antihypertensive drugs in the near future.

Declaration

We declare no conflict of interest whatsoever in respect of this work, and that this work is not under any consideration for publication in any other journal.

References

1. Smith JJ, Porth CJ. Posture and the circulation: the age effect. Exp Gerontol. 1991;26(2-3):141-162.
2. Smith JJ, Porth CJ, Ericckson M. Haemodynamic response to the upright posture. J Clin Pharmacol. 1994;34(5):375-386.
3. Fessel J, Robertson D. When pressor reflexes overcompensate. Nat Clin Pract Nephrol. 2006;2(8):424-431.
4. Kario K, Pickering TG, Hoshide S, et al. Morning BP surge and hypertensive cerebrovascular disease: role of the alpha adrenergic sympathetic nervous system. Am J Hypertens. 2004;17(8):668-675.
5. Kario K, Eguchi K, Hoshide Y, et al. U-curve relationship between orthostatic BP change and silent cerebrovascular disease in elderly hypertensives: orthostatic hypertension as new cardiovascular risk factor. J Am Coll Cardiol. 2002;40(1):133-141.
6. Yoshinari M, Wakisaka M, Nakamura U, et al. Orthostatic hypertension in patients with type 2 diabetes. Diabetes Care. 2001;24(10):1783-1786.
7. Kario K. Orthostatic hypertension: a measure of BP variation for predicting cardiovascular risk. Circ J. 2009;73(6):1002-1007.
8. Yoshinari M, Wakisaka M, Nakamura U, et al. Orthostatic hypertension in patients with type 2 diabetes. Diabetes Care. 2001;24(10):1783-1786.
9. Wu JS, Yang YC, Lu FH, et al. Population-based study on the prevalence and correlates of orthostatic hypotension/hypertension and orthostatic dizziness. Hypertens Res. 2008;31(5):897-904.
10. Vara-Gonzalez L, Munoz-Cacho P, Sanz de Casstro S. Postural changes in BP in the general population in Cantabria (northern Spain). Blood Press Monit. 2008;13(5):263-267.
11. Grenon SM, Xiao X, Hurwitz S, et al. Why is orthostatic tolerance lower in women than in men? Renal and cardiovascular responses to simulated microgravity and the role of midodrine. J Investig Med. 2006;54(4):180-190.
12. Fan XH, Wang YB, Wang H, et al. Polymorphism of angiotensin-converting enzyme inhibitors and ACE gene are not associated with orthostatic BP dysregulation in hypertensive patients. Acta Pharmacol Sin. 2009;30(9):1237-1244.