Study on the orthostatic changes in blood pressure among hypertensive patients aged 40 and above

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Abstract

Background: Numerous international studies have demonstrated that orthostatic hypotension and orthostatic hypertension are risk factors for cardiovascular events and mortality. In Vietnam, awareness and research on this issue remain limited. **Objectives:** 1) To assess changes in blood pressure from supine to standing. 2) To investigate the relationship between orthostatic blood pressure changes and risk factors and cardiac impairment. **Methods:** A cross-sectional study with a comparison group. The study group consisted of 70 hypertensive patients from the Department of Cardiology from June 2023 to December 2023. The control group consisted of 70 patients without hypertension who attended the Internal Medicine Clinic. **Results:** The prevalence of orthostatic hypertension in the hypertensive group (12.9%) was significantly higher than in the non-hypertensive group (2.9%) ($p = 0.028 < 0.05$). The prevalence of orthostatic hypotension in the hypertensive group (15.7%) was slightly lower than in the non-hypertensive group (18.6%). Among the hypertensive group, there was a significant association between a history of diabetes mellitus and orthostatic blood pressure changes ($p = 0.034 < 0.05$; OR = 3.17; 95% CI: 1.06 – 9.43); between left ventricular hypertrophy on ECG and orthostatic blood pressure changes ($p = 0.038 < 0.05$). In the hypertensive group, the prevalence of left ventricular hypertrophy was higher in those with orthostatic blood pressure changes (25.0%) compared to those without changes (6.0%). There was a positive correlation between standing systolic blood pressure and heart rate ($r = 0.388$; $p = 0.001 < 0.05$); between supine and standing systolic blood pressure with EF ($r = 0.352$; $p = 0.003 < 0.05$), ($r = 0.319$; $p = 0.007 < 0.05$); between supine, standing systolic blood pressure and supine diastolic blood pressure with PWV ($r = 0.289$; $p = 0.015 < 0.05$), ($r = 0.344$; $p = 0.004 < 0.05$), ($r = 0.313$; $p = 0.008 < 0.05$). **Conclusion:** The prevalence of orthostatic blood pressure changes in hypertensive patients is relatively high. Hypertensive patients need to be assessed for arterial stiffness indices, including PWV and ABI, to enable early detection and prediction of systolic blood pressure difference, thereby improving clinical monitoring. However, treatment-related factors, such as medication type, were not assessed and should be considered in future studies.

Keywords: *orthostatic hypertension, orthostatic hypotension, orthostatic blood pressure changes.*

1. INTRODUCTION

According to the World Health Organization, it is estimated that by 2023, approximately 1.28 billion adults aged 30 - 79 worldwide will suffer from hypertension, most of whom live in low- and middle-income countries [1]. Each year, over 7 million people die from hypertension and its complications.

According to the American Autonomic Society and the American Academy of Neurology, orthostatic hypotension (OH) is defined as a decrease in systolic blood pressure (SBP) of at least 20 mmHg and/or a reduction in diastolic blood pressure (DBP) of at least 10 mmHg within 3 minutes of standing [2]. Orthostatic hypotension in hypertensive patients is a common disorder yet is frequently overlooked during clinical evaluation. Orthostatic hypotension affects 6% of the general population and increases significantly with age [3]. To date, studies worldwide have shown that OH is a risk factor for cardiovascular events and mortality [4].

Additionally, the concept of orthostatic hypertension (OHT) has also been identified as a cardiovascular risk factor in some studies. According to the latest consensus from the American Autonomic Society and the Japanese Society of Hypertension in 2022, OHT is defined as an increase in SBP of at least 20 mmHg when transitioning from the supine to the standing position or an SBP increase of 140 mmHg or more in the standing position. However, attention to the issue of orthostatic blood pressure changes (OBP) in Vietnam remains limited. Community studies have mainly focused on hypertension risk

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DOI: 10.34071/jmp.2025.4.14
Received: 11/11/2024; Accepted: 25/07/2025; Published: 30/082025

factors and treatment due to their direct relation to cardiovascular events and mortality, while OBP changes are often overlooked. Notably, there is a lack of domestic research on OHT.

Therefore, we conducted this study titled: ***"Study on the orthostatic changes in blood pressure among hypertensive patients aged 40 and above"*** with the objectives:

1. To assess orthostatic blood pressure changes from supine to standing positions in hypertensive patients aged 40 and older.

2. To examine the relationship between orthostatic blood pressure changes and cardiovascular risk factors and target organ damage in hypertensive patients aged 40 and older.

2. SUBJECTS AND METHODS

2.1. Study Subjects

The study was conducted on 140 patients, divided into two groups. The research group comprised 70 patients with hypertension (HTN) receiving treatment at the Cardiovascular Department of Hue University of Medicine and Pharmacy Hospital from June 2023 to December 2023. The control group consisted of 70 patients without hypertension who visited the Internal Medicine Clinic at Hue University of Medicine and Pharmacy Hospital.

2.2. Research Methods

Study Design: Cross-sectional descriptive study with comparative control group.

Inclusion Criteria:

- Consent to participate in the study.

- Hypertensive group: patients with systolic/diastolic blood pressure ≥ 140/90 mmHg on admission and/or a prior diagnosis of hypertension under treatment.

- Non-Hypertensive group: Individuals aged 40 and above with no history of hypertension diagnosis or treatment and not meeting the criteria for hypertension diagnosis according to VNHA 2022 guidelines at the time of examination [5].

Exclusion Criteria:

- Patients with secondary hypertension: acute or chronic kidney disease, thyroid, parathyroid, pituitary disorders, drug-induced, etc.

- Incomplete clinical data and test results.

- Patients or family members who do not consent to participate in the study.

Definitions of Key Study Variables:

Blood pressure was measured using the Omron HEM-907 electronic sphygmomanometer (Japan), with appropriately sized cuffs based on the subject's arm circumference. In patients with arrhythmias (e.g., atrial fibrillation), blood pressure and heart rate were measured manually using a stethoscope and mercury sphygmomanometer to ensure accuracy. Heart rate was recorded from the first standing measurement.

- **Orthostatic Hypotension:** A decrease in SBP of at least 20 mmHg and/or a reduction in DBP of at least 10 mmHg upon standing from a supine position after 3 minutes [6].

- **Orthostatic Hypertension:** An increase in SBP of at least 20 mmHg when transitioning from supine to standing, or an SBP increase to 140 mmHg or higher in the standing position [7, 8].

- **Orthostatic Blood Pressure Change**: Refers to any change in blood pressure when transitioning from supine to standing position, including both OH and OHT.

- **Family History of Early Cardiovascular Disease:** Considered early if it occurs before age 65 for females and before age 55 for males [5].

- **Supine Blood Pressure:** Calculated as the average of three blood pressure measurements taken after five minutes of rest in the supine position, with each measurement spaced at least 2 minutes apart [8].

- **Standing Blood Pressure:** Blood pressure is measured after standing for 3 to 5 minutes, with the arm in the same position as during supine measurement. The average of the measurements taken at 3 and 5 minutes is used to determine standing systolic blood pressure [8].

- **Systolic Blood Pressure Difference:** The difference between supine and standing systolic blood pressure.

- **Diastolic Blood Pressure Difference:** The difference between supine and standing diastolic blood pressure.

- **Pulse Pressure:** The difference between systolic and diastolic blood pressure [5].

- **Left Ventricular Hypertrophy on ECG:** Defined by meeting the Sokolow-Lyon criteria: S.V1 + R.V5/V6 ≥ 35 mm [5].

- **Pulse wave velocity (PWV) and ankle-brachial index (ABI)**: These parameters are measured by the non-invasive Vascular Screening Device VP-1000plus from Omron.

2.3. Data Analysis:

The collected data were processed using the medical statistics software SPSS version 26.0.

3. RESULTS

Table 1. Orthostatic Blood Pressure Change Status

Orthostatic BP Change Status	Hypertensive Group (n = 70) n (%)	Non-Hypertensive Group (n = 70) n (%)	p
Orthostatic Hypertension	9 (12.9)	2 (2.9)	0.028
Orthostatic Hypotension	11 (15.7)	13 (18.6)	0.654
Orthostatic BP Change	20 (28.6)	15 (21.4)	0.329

The prevalence of OHT in the hypertensive group (12.9%) was higher than in the non-hypertensive group (2.9%), with a statistically significant difference ($p = 0.028 < 0.05$). The prevalence of OH in the hypertensive group (15.7%) was lower than in the non-hypertensive group (18.6%), but this difference was not statistically significant ($p = 0.654 > 0.05$).

Table 2. Blood Pressure Distribution by Position

	Hypertensive Group (n = 70)		Non-Hypertensive Group (n = 70)		Total (n = 140)	
SBP (mmHg)	**Supine SBP**	**Standing SBP**	**Supine SBP**	**Standing SBP**	**Supine SBP**	**Standing SBP**
$X \pm SD$	137.57 ± 19.42	137.46 ± 20.95	117.21 ± 10.46	114.82 ± 13.37	127.39 ± 18.60	126.14 ± 20.87
p	0.944		**0.040**		0.190	
DBP (mmHg)	**Supine DBP**	**Standing DBP**	**Supine DBP**	**Standing DBP**	**Supine DBP**	**Standing DBP**
$X \pm SD$	81.71 ± 11.43	83.61 ± 12.12	78.86 ± 10.43	79.32 ± 10.07	80.29 ± 10.99	81.46 ± 11.31
p	**0.028**		0.498		**0.032**	

In the hypertensive group, supine and standing SBP were similar ($p = 0.944 > 0.05$). However, there was a statistically significant change in DBP from supine to standing ($p = 0.028 < 0.05$). In the non-hypertensive group, SBP showed a substantial change from supine to standing ($p = 0.04 < 0.05$), while DBP remained consistent ($p = 0.498 > 0.05$).

Table 3. Factors Associated with Orthostatic BP Changes

		Hypertensive Group		Non-Hypertensive Group	
		Orthostatic BP Change (n = 20)	**No Orthostatic BP Change (n = 50)**	**Orthostatic BP Change (n = 15)**	**No Orthostatic BP Change (n = 55)**
Smoking	n(%)	1 (5.0)	9 (18.0)	5 (33.3)	15 (27.3)
	p	0.262		0.749	
Low Physical Activity	n(%)	9 (45.0)	29 (58.0)	3 (20.0)	17 (30.9)
	p	0.324		0.528	
Dyslipidemia	n(%)	6 (30.0)	12 (24.0)	5 (33.3)	14 (25.5)
	p	0.604		0.531	
Diabetes Mellitus	n(%)	10 (50.0)	12 (24.0)	1 (6.7)	2 (3.6)
	p	0.034		0.521	
Coronary Artery Disease	n(%)	5 (25.0)	9 (18.0)	0 (0)	1 (1.8)
	p	0.522		1	
Heart Failure	n(%)	0 (0)	4 (8.0)	1 (6.7)	2 (3.6)
	p	0.193		0.521	

Cerebrovascular Disease	n(%)	1 (5.0)	9 (18.0)	1 (6.7)	1 (1.8)
	p	0.262		0.385	
Peripheral Artery Disease	n(%)	0 (0)	3 (6.0)	0 (0)	0 (0)
	p	0.552			
Family History of Early Cardiovascular Disease	n(%)	1 (5.0)	3 (6.0)	1 (6.7)	2 (3.6)
	p	1		0.521	
Left Ventricular Hypertrophy on ECG	n(%)	5 (25.0)	3 (6.0)	0 (0)	3 (5.45)
	p	**0.0388**		1	

In the hypertensive group, a significant association was found between diabetes mellitus and orthostatic blood pressure changes ($p = 0.034 < 0.05$; OR = 3.17; 95% CI: 1.06–9.43). No significant associations were found between orthostatic BP changes and other risk factors in the non-hypertensive group ($p > 0.05$). Left ventricular hypertrophy on ECG was significantly associated with orthostatic BP changes in the hypertensive group ($p = 0.038 < 0.05$).

Table 4. Correlation Factors with Blood Pressure by Position

			Supine SBP	**Standing SBP**	**Supine DBP**	**Standing DBP**
Waist Circumference (cm)	Hypertensive Group (n = 70)	r	0.169	0.307	0.126	0.309
		p	0.162	0.010	0.300	0.009
	Non-Hypertensive Group (n = 70)	r	0.156	0.233	0.133	0.095
		p	0.197	0.052	0.272	0.433
BMI (kg/m^2)	Hypertensive Group (n = 70)	r	0.222	0.234	0.157	0.301
		p	0.065	0.051	0.195	0.011
	Non-Hypertensive Group (n = 70)	r	0.167	0.230	0.224	0.171
		p	0.166	0.056	0.063	0.156
Triglyceride (mmol/L)	Hypertensive Group (n = 70)	r	0.174	0.094	0.163	0.103
		p	0.149	0.437	0.177	0.396
	Non-Hypertensive Group (n = 70)	r	0.186	-0.001	0.197	0.043
		p	0.124	0.991	0.101	0.724
LDL-C (mmol/L)	Hypertensive Group (n = 70)	r	0.110	0.016	0.083	0.133
		p	0.366	0.897	0.494	0.271
	Non-Hypertensive Group (n = 70)	r	0.022	0.062	0.000	0.043
		p	0.855	0.611	0.999	0.721
LVMI (g/m^2)	Hypertensive Group (n = 70)	R	-0.021	-0.070	-0.013	0.103
		p	0.863	0.565	0.914	0.938
EF (%)	Non-Hypertensive Group (n = 70)	r	0.352	0.319	0.148	0.235
		p	0.003	0.007	0.222	0.050
PWV (m/s)	Hypertensive Group (n = 70)	r	0.289	0.344	0.313	0.226
		p	0.015	0.004	0.008	0.060
	Non-Hypertensive Group (n = 70)	r	0.254	0.261	0.119	0.164
		p	0.034	0.029	0.328	0.174
ABI (Left)	Hypertensive Group (n = 70)	r	-0.131	-0.042	-0.034	0.017
		p	0.279	0.731	0.779	0.890
	Non-Hypertensive Group (n = 70)	r	0.135	0.015	0.121	0.026
		p	0.266	0.905	0.317	0.831

ABI (Right)	Hypertensive Group (n = 70)	r	-0.013	0.126	0.42	0.132
		p	0.917	0.298	0.732	0.277
	Non-Hypertensive Group (n = 70)	r	0.200	0.029	0.245	0.129
		p	0.096	0.813	0.041	0.286

Notes: BMI: Body mass index, LVMI: Left ventricular mass index, EF: Ejection fraction, PWV: Pulse wave velocity, ABI: Ankle-Brachial index.

There was a statistically significant positive correlation between standing SBP, standing DBP, and waist circumference in the hypertensive group (r = 0.307; p = 0.010 < 0.05 for SBP; r = 0.309; p = 0.009 < 0.05 for DBP). Additionally, a significant positive correlation was found between standing DBP and BMI in the hypertensive group (r = 0.301; p = 0.011, p < 0.05). No correlation was observed between SBP and DBP, in both the supine and standing positions, with LVMI (p > 0.05).

There was a statistically significant positive correlation between supine SBP and standing SBP with EF (r = 0.352; p = 0.003 < 0.05 for supine SBP; r = 0.319; p = 0.007 < 0.05 for standing SBP). In the hypertensive group, there was a significant positive correlation between supine SBP and PWV (r = 0.289; p = 0.015 < 0.05), as well as between standing SBP and supine DBP with PWV (r = 0.344; p = 0.004 < 0.05 for standing SBP; r = 0.313; p = 0.008 < 0.05 for supine DBP).

In the non-hypertensive group, there was a statistically significant positive correlation between supine SBP and standing SBP with PWV (r = 0.254; p = 0.034 < 0.05 for supine SBP; r = 0.261; p = 0.029 < 0.05 for standing SBP). Additionally, there was a significant positive correlation between supine DBP and right-side ABI in the non-hypertensive group (r = 0.245; p = 0.041 < 0.05).

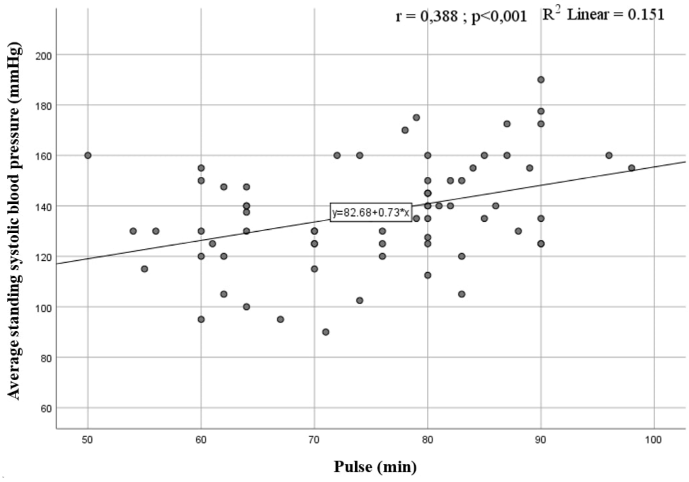

Figure 1. Correlation Between Average Standing Systolic Blood Pressure and Heart Rate in the Hypertensive Group

There was a statistically significant, moderate positive correlation between standing SBP and heart rate in the hypertensive group (r = 0.388; p = 0.001, p < 0.05).

Table 5. Linear Regression Model Predicting the Level of Systolic Blood Pressure Difference (n=140)

Risk Factor	Unstandardized Coefficient		Standardized Coefficient Beta	t	p
	B	Standard Error			
Constant	40.65	9.44		4.31	< 0.001
Triglyceride (mmol/L)	0.85	0.33	0.20	2.58	0.011
Waist Circumference (cm)	-0.31	0.09	-0.27	-3.33	0.001
Heart rate (bpm)	-0.31	0.09	-0.29	-3.59	< 0.001
Supine Pulse Pressure (mmHg)	0.15	0.06	0.20	2.46	0.015
			R^2	18.20%	

This model exhibits no multicollinearity, with an adjusted R-squared value of 18.20%. All variables are statistically significant ($p < 0.05$). The predictive model for the level of systolic blood pressure difference is as follows:

Systolic BP Difference = 40.65 + 0.85 (Triglyceride) - 0.31*(Waist Circumference) - 0.31*(Heart rate) + 0.15*(Supine Pulse Pressure).

4. DISCUSSION

In our study, the prevalence of OHT in the hypertensive group (12.9%) was higher than in the non-hypertensive group (2.9%). The prevalence of OH in the hypertensive group (15.7%) was lower than in the non-hypertensive group (18.6%). A study by Dang Le Minh Tri (2021) on the prevalence and risk factors of OH in elderly hypertensive patients reported an OH prevalence of 26.2% [9]. Another study by Hong-Jie Chi (2019) involving 1997 subjects aged 60 and above found OH and OHT prevalences of 23.1% and 9.5%, respectively [10]. This suggests that OH occurs more frequently than OHT, though OHT remains significant, albeit with limited attention.

Diabetes can lead to OH due to damage to the autonomic nervous system. In cases with increased sympathetic activity and autonomic jumpy degeneration, as seen in diabetes, it may also be considered a factor contributing to OH [11], [12]. In this study, we found a statistically significant association between diabetes and orthostatic BP changes ($p = 0.015 < 0.05$; OR = 3.17; 95% CI: 0.14 – 0.83). In Dang Le Minh Tri's study (2021), diabetes was also identified as a risk factor for OH ($p = 0.006 < 0.05$; OR = 1.86; 95% CI: 1.19 – 2.9) [9]. Similarly, Guerin's study (2016) showed an association between OH and diabetes ($p = 0.03 < 0.05$; OR = 4.23; 95% CI: 1.10-16.24) [13].

A study by Nguyen Trong Hieu (2023) on the prevalence of left ventricular hypertrophy (LVH) on ECG in hypertensive individuals aged 18 and older found that 10,4% of subjects were diagnosed with LVH [14]. Another study by J Julien (2004) on the prevalence of LVH in hypertensive patients reported a prevalence of >15% for LVH on ECG [15]. In our study, among hypertensive patients, the rate of LVH on ECG was higher in those with orthostatic BP changes (25%) than in those without (6%), a statistically significant difference ($p = 0.038 < 0.05$). This finding suggests that many hypertensive patients with LVH on ECG also exhibit orthostatic BP changes, possibly due to autonomic dysfunction, myocardial hypertrophy, and arterial stiffness, which impair BP regulation in the standing position.

Our study also found a statistically significant positive correlation between waist circumference and standing SBP and DBP in the hypertensive group, with r and p values as follows: ($r = 0.307$; $p = 0.010 < 0.05$ for SBP) and ($r = 0.309$; $p = 0.009 < 0.05$ for DBP). In a study by Nguyen Duc Hoang and Le Thanh Hai (2014), a correlation was observed between supine and standing SBP and waist circumference, with r and p values of ($r = 0.44$; $p < 0.0001$ for supine SBP) and ($r = 0.335$; $p < 0.0001$ for standing SBP). There was also a correlation between supine DBP and waist circumference ($r = 0.275$; $p < 0.01$), although no correlation was found between standing DBP and waist circumference ($p > 0.05$) [16].

Our study further demonstrated a statistically significant positive correlation between standing DBP and BMI in the hypertensive group ($r = 0.301$; $p = 0.011 < 0.05$). In a study by Vu Van Nga (2016) on 269 individuals aged 20–64, BMI was primarily associated with SBP and DBP, with r values of 0.309 and 0.352, respectively [17]. Similarly, a survey by Le Thanh Chien (2011) indicated a positive correlation between BMI and both SBP and DBP, with r values of 0.41 and 0.38, respectively ($p < 0.05$) [18].

Our research also showed a statistically significant positive correlation between supine DBP and posterior ankle-brachial index (ABI) in the non-hypertensive group ($r = 0.245$; $p = 0.041 < 0.05$). A study by Y. Y. Luo et al. (2007) examining the relationship between ABI and all-cause and cardiovascular mortality in hypertensive patients in China found that, among 3047 subjects, 839 (27.5%) had a low ABI. After 13 months of follow-up, there were 252 deaths, 100 of which were due to cardiovascular disease. Low ABI was associated with increased all-cause and cardiovascular mortality, with RR and 95% CI of (1.619; 1.190 – 2.203) and (2.454; 1.531 – 3.933), respectively [19]. In a study by Worillas et al. (2009) on 1101 hypertensive patients aged 40 and above with acute coronary syndrome, 469 patients (42.6%) had an ABI < 0.9. Patients in this group were older, had higher rates of diabetes, coronary artery disease, or stroke, more severe LVH, and more significant coronary artery lesions. Hospital mortality was higher in hypertensive patients with ABI < 0.9 (2.3% vs. 0.2%; $p < 0.01$) [20].

Typically, when transitioning from a supine to a standing position, sympathetic activation and reduced parasympathetic activity increase heart rate. In patients with neurogenic OH, the heart rate increases by only about 10-15 beats, which is less than in patients with OH not caused by autonomic dysfunction (typically more than 15 beats) [21]. Our study found a statistically significant positive correlation between standing SBP and heart rate (r =

0.388; p = 0.001). In a survey by Nguyen Duc Hoang and Le Thanh Hai (2014), a statistically significant positive correlation was observed between supine SBP (r = 0.34; p < 0.01) and standing SBP (r = 0.33; p < 0.01) and heart rate, as well as a weak positive correlation between supine DBP and heart rate (r = 0.262; p < 0.01) [16].

Among all study participants (140 individuals), a multivariate correlation was found between the level of systolic blood pressure difference and triglycerides, waist circumference, heart rate, and supine pulse pressure. This model showed no multicollinearity, with an adjusted R^2 of 18.20%. All variables were statistically significant ($p < 0.05$). The predictive model for the level of systolic blood pressure difference is as follows:

Systolic BP Difference = 40.65 + 0.85 * (Triglyceride) - 0.31 * (Waist Circumference) - 0.31 * (Heart rate) + 0.15 * (Supine Pulse Pressure).

5. CONCLUSIONS

The prevalence of orthostatic blood pressure changes among hypertensive patients is relatively high. Proper attention should be given to the conditions of orthostatic hypotension and orthostatic hypertension. Hypertensive patients should be assessed for arterial stiffness indices, including PWV and ABI, to enable early detection and prediction of systolic blood pressure difference, thereby improving clinical monitoring. However, treatment-related factors such as medication type were not assessed and should be considered in future studies.

REFERENCES

1. WHO. Hypertension 2021 [Available from: https://www.who.int/news-room/fact-sheets/ detail/ hypertension].

2. R. Freeman et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope, and postural tachycardia syndrome. Clin Auton Res. 2011;21(2):69-72.

3. A. Joseph et al. Orthostatic hypotension: A review. Nephrol Ther. 2017;13(1):55-67.

4. Tran Cong Duy. Diagnosis and treatment of orthostatic hypotension. Ho Chi Minh City Cardiology Association. 2013.

5. Vietnam Cardiology Association. Recommendations of the Hypertension Subsection - Vietnam Cardiology Association (VSH/VNHA) 2022 on the diagnosis and treatment of hypertension. Recommendations on Diagnosis and Treatment of Hypertension. 2022.

6. Fan XH, Wang Y, Sun K, Zhang W, Wang H, Wu H, Zhang H, Zhou X, Hui R. Disorders of orthostatic blood pressure response are associated with cardiovascular disease and target organ damage in hypertensive patients. Am J Hypertens. 2010 Aug;23(8):829-37.

7. Jordan J, Ricci F, Hoffmann F, Hamrefors V, Fedorowski A. Orthostatic Hypertension: Critical Appraisal of an Overlooked Condition. Hypertension. 2020 May;75(5):1151-1158.

8. Jordan J, Biaggioni I, Kotsis V, Nilsson P, Grassi G, Fedorowski A, Kario K. Consensus statement on the definition of orthostatic hypertension endorsed by the American Autonomic Society and the Japanese Society of Hypertension. Clin Auton Res. 2023 Feb;33(1):69-73.

9. Dang Le Minh Tri and Le Kieu Minh. Study on the prevalence and risk factors of orthostatic hypotension in elderly hypertensive patients at Military Hospital 175. Journal of Medicine - Practical Pharmacy 175. 2021.

10. H. J. Chi et al. The association between orthostatic blood pressure changes and subclinical target organ damage in individuals aged 60 years or older. J Geriatr Cardiol. 2019;16(5):387-94.

11. N. Magkas et al. Orthostatic hypotension: From pathophysiology to clinical applications and therapeutic considerations. J Clin Hypertens (Greenwich). 2019;21:426-554.

12. Low PA, Tomalia VA. Orthostatic Hypotension: Mechanisms, Causes, Management. J Clin Neurol. 2015 Jul;11(3):220-6.

13. A. Guerin et al. Factors associated with orthostatic hypotension in hospitalized elderly patients. Aging Clin Exp Res. 2016;28(3):513-7.

14. Nguyen Trong Hieu. Prevalence of left ventricular hypertrophy and related factors in hypertensive individuals aged 18 and older in Binh Chanh district, Ho Chi Minh City in 2022-2023. Vietnam Medical Journal. 2023;531.

15. Julien J, Tranche C, Souchet T. [Left ventricular hypertrophy in hypertensive patients: epidemiology and prognosis. Arch Mal Coeur Vaiss. 2004;97(3):221-7.

16. Nguyen Duc Hoang and Le Thanh Hai. Study on the prevalence and risk factors of orthostatic hypotension in elderly hypertensive patients. Vietnamese Journal of Cardiology. 2014;65(50-57).

17. Vu Van Nga and Do Thi Quynh. Some cardiovascular risk factors among university staff in Hanoi in 2016. Journal of Science, VNU: Medical and Pharmaceutical Sciences. 2016;34(2):89-96.

18. Le Thanh Chien and colleagues. Survey on the correlation between hypertension, BMI, waist circumference, and waist-to-hip ratio in residents of Hoa Thanh ward, Tan Phu district. Ho Chi Minh City Medical Journal. 2011;15(4).

19. Luo YY, et al. Risk factors for peripheral arterial disease and the relationship between low ankle-brachial index and mortality from all-cause and cardiovascular disease in Chinese hypertensive patients. J Hum Hypertens. 2007;21:461-6.

20. Morillas P, Cordero A, Bertomeu V, Gonzalez-Juanatey JR, Quiles J, Guindo J, et al. Prognostic value of low ankle-brachial index in hypertensive patients with acute coronary syndromes. J Hypertens. 2009;27(2):341-7.

21. Gibbons CH, Schmidt P, Biaggioni I, Frazier-Mills C, Freeman R, Isaacson S, et al. Recommendations from a consensus panel for the screening, diagnosis, and treatment of neurogenic orthostatic hypotension and associated supine hypertension. J Neurol. 2017;264(8):1567-82.