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Original Article

Association of nocturnal blood pressure and left ventricular hypertrophy in Iranian hypertensive patients

Shideh Anvari[1], Ehsan Noroozi[1], Mohammad Amin Karimi[2], Reza Khademi[3], Seyyed Kiarash Sadat Rafiei[4], Samira Pirzad[5], Nima Zabihi[6], Niloofar Deravi[4], Arezou Soltanattar[7], Fariba Samadian[1], Elham Keikha[1]

[1]Department of Internal Medicine, Shahid Labbafinezhad Hospital, Shahid Beheshti University of Medical Science, Tehran, Iran; [2]School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran; [3]Student Research Committee, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran; [4]Student Research Committee, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran; [5]School of Medicine, Islamic Azad University, Tehran, Iran; [6]Shahid Modarres Cardiovascular Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran; [7]Ali Ibn Abitaleb Medical School, Islamic Azad University Yazd, Yazd, Iran

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Abstract: Objectives: Hypertension is a prevalent risk factor for cardiovascular mortality and morbidity, often leading to left ventricular hypertrophy (LVH). As ambulatory blood pressure monitoring (ABPM) gains prominence in hypertension management, it is crucial to explore its association with LVH occurrence to enhance clinical understanding and treatment strategies. This study aims to investigate the correlation between nocturnal blood pressure patterns and presence of LVH in hypertensive patients, offering insights into optimizing hypertension management strategies. Methods: Fifty-four patients with confirmed hypertension were included in this study. All participants underwent transthoracic echocardiography within two days of admission and 48-hour ABPM within one week of admission. Based on the presence of LVH, patients were categorized into LVH and non-LVH groups. Nocturnal systolic/diastolic BP were compared between the two groups using the appropriate statistical tests. Results: Among the 54 hypertensive patients, those with LVH (n = 22) demonstrated a significantly higher nocturnal average SBP (124.04 ± 11.92 mmHg) and DBP (76.24 ± 9.76) compared to those without LVH (n = 32, SBP = 116.78 ± 13.92 mmHg, DBP = 72.45 ± 9.76, P < 0.001). Conclusion: This research shows a significant association between nocturnal BP patterns and the presence of LVH in hypertensive individuals. Nocturnal SBP and DBP were identified as independent risk factors for LVH. Further research, particularly on the timing of antihypertensive medication, is warranted to confirm causal relationships and improve management strategies.

Keywords: Nocturnal blood pressure, left ventricular hypertrophy, LVH, hypertension

Introduction

Hypertension (HTN) is a leading and modifiable risk factor contributing significantly to cardiovascular disease (CVD) morbidity and mortality. The current guidelines define HTN as a systolic blood pressure (SBP) of 130 mmHg or higher or a diastolic blood pressure (DBP) of 80 mmHg or higher, resulting in a substantial increase in the prevalence of HTN, now affecting 46% of U.S. adults [1, 2]. Left ventricular hypertrophy (LVH), a target organ damage, is frequently observed in individuals with HTN. Research findings suggest that more than 40% of individuals with HTN exhibit LVH [3-5]. LVH is characterized by an increase in the mass of the left ventricle (LV) due to either thickening of the LV walls, enlargement of the LV cavity, or both. This structural change typically occurs as a response to prolonged pressure or volume overload, leading to myocardial fibers enlargement. LVH is recognized as an independent risk factor for the early onset of cardiovascular complications, including chronic heart failure, cardiac arrhythmias, and sudden cardiac death. Therefore, its presence elevates the risk of both morbidity and mortality [6-8]. Early detection, evaluation, and treatment of LVH are crucial for preventing coronary heart disease among hypertensive individuals.

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Circadian rhythm is an important factors influencing blood pressure values, with BP typically being lowest during the night [9]. According to this fluctuation, individuals are categorized into two groups: dippers and non-dippers. Dippers characterized by a decrease in mean systolic/diastolic nighttime BP by more than 10% compared to daytime BP values, while, non-dippers show a BP fall of less than 10% [10]. In recent years, research has highlighted the importance of circadian blood pressure patterns, especially the non-dipping and reverse dipping patterns, in predicting cardiovascular risk. A recent meta-analysis of 6918 hypertensive patients revealed that those with a reverse dipping pattern faced a 2.5-fold higher risk of cardiovascular events compared to dippers, and a 2.0-fold higher risk compared to non-dippers [11, 12].

Over the past three decades, 24-hour ABPM has become the preferred method for measuring BP outside clinical settings, significantly enhancing HTN management by providing insights into nocturnal BP levels that are inaccessible through traditional methods. To align with current HTN guidelines, it is recommended to include ABPM as part of the assessment, alongside the evaluation of clinical blood pressure [1, 2, 13, 14]. Previous studies have suggested that abnormal BP circadian rhythm, including nocturnal HTN, are linked to both structural and functional changes in the LV. Non-dipping and reverse dipping BP patterns have been associated with reduced left ventricular strain and increased LVMI, indicating impaired ventricular function and hypertrophy. Specifically, reverse dipping patterns were linked to significant reductions in both longitudinal and circumferential LV strains and increased risk of LVH [15-17].

Nevertheless, several other investigations have shown that the correlation between circadian rhythms of blood pressure and left LVH, as well as its therapeutic implications, remains uncertain [18, 19]. ABPM enable a comprehensive 24-hour evaluation of blood pressure parameters, encompassing both daytime and nighttime measurements, while capturing the circadian rhythm of blood pressure fluctuations. Analyzing and identifying these patterns using ABPM may yield valuable insights into the relationship between nocturnal HTN and organ damage, such as LVH.

Therefore, this observational cohort study was conducted to explore the relationship between 48-hour ABPM and the circadian rhythm of blood pressure with LVH in patients diagnosed with HTN.

Materials and methods

Study participants

The primary objective of this investigation was to explore the relationship between 48-hour ABPM and cardiovascular mortality and morbidity within the Iranian population. The study was conducted at Labbafinejad Hospital, affiliated with Shahid Beheshti University of Medical Sciences. A cohort of 54 participants was recruited for this analysis and divided into two distinct groups. The first group comprised 32 hypertensive patients without LVH, while the second group consisted of 22 hypertensive patients who exhibited LVH.

Inclusion criteria for participation encompassed individuals with HTN who had confirmed elevated BP and LVH through ECG, underwent transthoracic echocardiography within two days of their inpatient visit, and had 48-hour ABPM obtained within one week of their index inpatient visit. Exclusion criteria involved individuals with secondary HTN and those with various forms of CVD, including but not limited to restrictive or dilated cardiomyopathy, myocardial infarction, coronary heart disease, intracranial hemorrhage, ischemic stroke, valvular heart disease, heart failure, hypertrophy, myocarditis, and congenital heart disease.

Clinical and biochemical data collection

Comprehensive clinical data were collected from all patients, including measurements of DBP, SBP, gender, age, and body mass index (BMI). BMI was calculated as weight in kilograms divided by the square of the height in meters. Blood samples were collected after an overnight fasting period. Biochemical parameters, including serum triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), were determined using an automatic biochemical analyzer (Roche modular 7600 automatic biochemistry analyzer).

Echocardiography measurement

Echocardiographic measurements were performed utilized the Color Doppler Ultrasonic Diagnostic Apparatus (SEQUOIA512, American Siemens Co., Ltd.) operating at a frequency range of 3.5 to 5 MHz. The chordae tendineae of the mitral valve were evaluated using the parasternal long-axis view, with patients were positioned in the left-lateral decubitus position. The parameters assessed included interventricular septal thickness (IVST), left ventricular end-systolic dimension (LVESD), left ventricular end-diastolic dimension (LVEDD), and posterior wall thickness (PWT). These measurements were used to calculate fractional shortening (FS) and left ventricular ejection fraction (EF). Mean values were calculated from continuous cardiac cycles measurments. According to Devereux et al. [20], the following formulas were utilized to calculate the Left ventricular weight or left ventricular mass (LVM):

$$LVM = 0.8 \{1.04 [((LVEDD + IVSd + PWd)^3 - LVEDD^3)]\} + 0.6$$

LVM: left ventricular mass; LVEDD: Left ventricular end-diastolic dimension; IVSd: interventricular septal thickness at end-diastole; PWd: posterior wall thickness at end-diastole.

Additionally, the left ventricular mass index (LVMI) was determined by:

$$LVMI = \frac{LVM}{height^{2.7}}$$

Patients were categorized into LVH and non-LVH groups based on the LVMI value. An LVMI greater than 115 for males and 95 for females was considered indicative of LVH [21]. Patient with LVMI below these cutoffs were classified in the non-LVH group.

Ambulatory blood pressure measurement

In this investigation, we employed the American Sun Tech OSCAR 2 24-hour ambulatory blood pressure monitor, equipped with a cuff measuring 22 cm × 12 cm, positioned on the left upper arm of patients. The cuff was inflated within a pressure range of 40 to 220 mmHg, with an outgassing rate set at 2 mmHg/s. Continuous blood pressure monitoring was conducted throughout the day, with measurements automatically taken every 30 minutes during the daytime period (from 6:00 to 22:00) and every hour during the nocturnal period (from 22:00 to 6:00). Monitoring began at 8 a.m. and concluded after 24 hours at 8 a.m the following day. Failure to effectively monitor blood pressure was described as having a SBP equal to or exceeding 260 mmHg and DBP equal to or exceeding 150 mmHg. Parameters obtained from ABPM included the average daytime DBP and SBP (24-hour DBP, 24-hour SBP), and the average nocturnal DBP and SBP.

Statistical methods

Statistical analysis was conducted using the SPSS 13.0 software package (SPSS Inc., Chicago). Continuous variables were presented as means accompanied by standard deviations or as medians with interquartile ranges (IQR), while categorical variables were expressed as absolute frequencies, counts, and percentages. The chi-squared (χ^2) test was utilized to evaluate categorical variables, and disparities in baseline characteristics between groups were assessed using Student's t-test.

Results

Baseline characteristics

In this investigation, out of the 54 individuals diagnosed with HTN, 22 were identified as having concurrent LVH, while 32 did not (40% vs. 74%). The baseline clinical characteristics of both groups are presented in **Table 1**. No notable variations were observed between the two groups concerning baseline features, encompassing age, SBP and DBP, cholesterol levels, triglycerides, LDL-C, and HDL-C among hypertensive patients with and without LVH ($P > 0.05$). Nonetheless, hypertensive patients with LVH displayed a notably elevated body mass index (BMI) (26.2 ± 3.05 vs. 24.1 ± 2.9) and a greater proportion of female individuals (81% vs. 59%) compared to hypertensive patients without LVH ($P < 0.05$).

Nocturnal blood pressure assessment

The evaluation of nocturnal blood pressure parameters in hypertensive individuals with and without LVH reveals significant differences. The results demonstrated that hypertensive patients with LVH (n = 22) exhibited higher

Table 1. Clinical characteristics

Characteristics	Hypertensive patients without LVH (n = 32)	Hypertensive patients with LVH (n = 22)
Age (years)	59.78 ± 12.17	64.09 ± 10.72
Gender (female)	59% (n = 19)	81% (n = 18)
BMI (kg/m^2)	24.1 ± 2.9	26.2 ± 3.05
SBP (mmHg)	150.2 ± 10.3	153.4 ± 10.9
DBP (mmHg)	98.2 ± 7.9	99.5 ± 8.2
HDL-C (mmol/L)	1.11 ± 0.53	1.25 ± 0.35
LDL-C (mmol/L)	3.01 ± 0.57	3.12 ± 0.51
TC (mmol/L)	5.15 ± 0.95	5.23 ± 1.18
TG (mmol/L)	1.42 ± 0.78	1.58 ± 0.85

Abbreviations: BMI: Body Mass Index; BP: Blood Pressure; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol; TC: Total Cholesterol; TG: Triglyceride.

Table 2. Comparison of nocturnal blood pressure variables in hypertensive patients with and without LVH

Variables	Hypertensive patients without LVH (n = 32)	Hypertensive patients with LVH (n = 22)	*P*-Value
NSBP (mmHg)	116.78 ± 13.92	124.04 ± 11.92	< 0.001
NDBP (mmHg)	72.45 ± 9.76	76.24 ± 9.76	< 0.001

NSBP: Nocturnal Systolic Blood Pressure; NDBP: Nocturnal Diastolic Blood Pressure; LVH: left ventricular hypertrophy.

mean nocturnal SBP (124.04 ± 11.92 mmHg) compared to those without LVH (116.78 ± 13.92 mmHg), with a *p*-value < 0.001, indicating statistical significance. Similarly, the mean nocturnal DBP was elevated in the LVH group (76.24 ± 9.76 mmHg) compared to the non-LVH group (72.45 ± 9.76 mmHg), also with a *p*-value < 0.001. These findings suggest a strong association between the presence of LVH and increased nocturnal blood pressure levels in hypertensive individuals (**Table 2**).

Discussion

In this study, we examined hypertensive individuals' nocturnal blood pressure profiles and stratified them based on the presence or absence of LVH. Our findings revealed significant discrepancies in nocturnal SBP and DBP between individuals with LVH and those without, underscoring the critical importance of evaluating nocturnal blood pressure patterns in hypertensive individuals, particularly concerning the presence of LVH. These observations suggest a potential association between nocturnal BP patterns and the presence of LVH in hypertensive patients.

Blood pressure exhibits a characteristic nocturnal dipping pattern, wherein it decreases during nighttime hours. While daytime blood pressure targets typically aim for values below 130/80 mmHg, thresholds for nocturnal blood pressure are lower, often set at or above 120/70 mmHg, although more stringent criteria advocate for values surpassing 110/65 mmHg [1, 22, 23]. Nocturnal HTN is prevalent, affecting a substantial proportion, estimated to be between 40-60%, of the population. This condition is associated with adverse effects on end-organ health, notably leading to elevated left ventricular mass and carotid intima-media thickness, which in turn elevate the risks of cardiovascular events and mortality [24-27].

Our investigation identified significant associations between nocturnal SBP and DBP with LVH. Individuals presenting with LVH demonstrated markedly elevated levels of both SBP and DBP compared to those without LVH. The precise pathophysiological mechanisms linking nocturnal HTN and LVH remain unclear. The nocturnal rise in blood pressure is primarily attributed to increased sympathetic nervous system activity and decreased parasympathetic activity during sleep. Studies consistently demonstrate elevated catecholamine (norepinephrine and epinephrine) levels in untreated essential hypertensive patients with reduced nocturnal blood pressure decline, suggesting a role for sympathetic activation in circadian blood pressure fluctuations. Additionally, noc-

turnal sympathetic nervous system activity may stimulate the renin-angiotensin-aldosterone system (RAAS) in hypertensive individuals, leading to heightened Angiotensin II and aldosterone secretion. Angiotensin II directly induces myocardial hypertrophy, while aldosterone promotes myocardial fibrosis through increased collagen production, contributing to LVH.

As mentioned, our findings indicate a significance difference between nocturnal SBP and DBP between patients with and without LVH ($P < 0.001$), aligns with previous studies in this field. For example, Toriumi et al. [28] performed a cross-sectional investigation employing data extracted from the Japan Morning Surge Home Blood Pressure (J-HOP) study, encompassing 1,277 patients subjected to ambulatory blood pressure monitoring and echocardiography. Their analysis revealed a notable association between poor control of nocturnal SBP and an increased risk of LVH. Intriguingly, in contrast the clear association between nocturnal BP and LVH, daytime SBP did not display a substantial association with the risk of LVH in this cohort [28].

Furthermore, the study by Andrikou et al. [29] involving 305 hypertensives patients followed up for an average duration of 42 months, demonstrated that baseline nocturnal SBP was a strong predictor for the development or persistence of LVH. Whereas baseline daytime SBP did not exhibit such predictive capability. Additionally, a decrease in nocturnal SBP was associated with a nearly threefold increase in the likelihood of left ventricular mass index (LVMI) reduction, regardless of daytime BP changes [29]. This reinforces the importance of nocturnal BP in predicting LVH outcomes, which is consistent with our finding that nocturnal SBP and DBP are significantly higher in patients with LVH.

Furthermore, findings from a substantial cohort within the Italian general populace also highlighted the role of nocturnal blood pressure as a strong, independent predictor for the onset of LVH among individuals with initially normal left ventricular mass [30]. These results confirmed our results that nocturnal BP measurements can serve as an important marker for LVH in hypertensive patients.

This study exhibits notable strengths, including comprehensive data collection encompassing clinical, biochemical, and echocardiographic parameters, facilitating a thorough examination of factors influencing cardiovascular outcomes. Employing ambulatory blood pressure monitoring over a 48-hour period enhances the accuracy and detail of measurements, augmenting the reliability of the findings. Moreover, including an echocardiographic evaluation by a cardiologist enhances the credibility and precision of the results. However, several limitations should be considered. The small sample size may restrict the generalizability of findings and diminish statistical power for detecting significant associations. Additionally, being a single-center study conducted at a hospital, the research's findings may lack representativeness of the broader population, potentially compromising the external validity of the results. Moreover, the cross-sectional design precludes establishing causal relationships between nocturnal blood pressure and left ventricular hypertrophy, emphasizing the need for future prospective studies to elucidate causal pathways.

Conclusion

In conclusion, our study highlights a significant association between nocturnal BP patterns and the presence of LVH in hypertensive individuals. The nocturnal SBP and the DBP emerged as independent risk factors for LVH. However, further investigation is warranted, particularly regarding the timing of drug administration in hypertensive patients. While our findings underscore the importance of monitoring nocturnal blood pressure in this population, further research is needed to confirm causal relationships and expand generalizability. Despite limitations such as sample size and study design, our study contributes valuable insights into potential mechanisms underlying cardiovascular risk in hypertensive patients with LVH, informing future clinical practice and research efforts.

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Informed written consent was obtained from all participants included in this study.

Disclosure of conflict of interest

None.

Abbreviations

CVD, Cardiovascular Disease; HTN, Hypertension; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; LVH, Left Ventricular Hypertrophy; LV, Left Ventricle; ABPM, Ambulatory Blood Pressure Monitoring; ECG, Electrocardiogram; BMI, Body Mass Index; TG, Triglyceride; TC, Total Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; HDL-C, High-Density Lipoprotein Cholesterol; IVST, Interventricular Septal Thickness; LVESD, Left Ventricular End-Systolic Dimension; LVEDD, Left Ventricular End-Diastolic Dimension; PWT, Posterior Wall Thickness; FS, Fractional Shortening; EF, Ejection Fraction; LVMI, Left Ventricular Mass Index; NSBP, Nocturnal Systolic Blood Pressure; NDBP, Nocturnal Diastolic Blood Pressure; RAAS, Renin-Angiotensin-Aldosterone System.

Address correspondence to: Elham Keikha, Department of Internal Medicine, Shahid Labbafinejad Medical Center, Shahid Beheshti University of Medical Science, Boostan 9th Street, Pasdaran Avenue, Tehran 1666663111, Iran. E-mail: Keykha6454@yahoo.com

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