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Prevalence of Left Ventricular Hypertrophy and Its Association with Blood Pressure Control in Hypertensive Patients at Vajira Hospital, Navamindradhiraj University

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ABSTRACT

OBJECTIVES: This study aimed to determine the prevalence of left ventricular hypertrophy (LVH) diagnosed via echocardiography and the relationship between blood pressure (BP) control and LVH and identify factors associated with LVH among patients with hypertension in Thailand.

METHODS: This cross-sectional study included 107 patients with hypertension who visited a cardiology clinic between March 2024 and August 2024. The baseline characteristics, office BP, and morning and evening home BP measurements of the participants were obtained. Echocardiographic criteria for LVH diagnosis are left ventricular mass index > 95 g/m^2 in women and > 115 g/m^2 in men. The primary outcomes were to determine the prevalence of LVH and assess the relationship between BP control and LVH.

RESULTS: The prevalence of LVH was 32.70%, with all the patients diagnosed with LVH exhibiting a concentric hypertrophy phenotype. Among the patients, 59.80% had controlled home BP, whereas 42% had controlled office BP. The prevalence of LVH was 22.50% among patients with both controlled office and home BP, 44.70% among those with both uncontrolled office and home BP, 20% in the group with controlled office but uncontrolled home BP, and 33.30% in the group with uncontrolled office BP but controlled home BP. Multivariate analysis showed that the number of antihypertensive drugs use was the only significant associated factor.

CONCLUSION: The prevalence of LVH is high among patients with hypertension, particularly those with uncontrolled office and home BP. This indicates the need for effective hypertension management strategies to prevent hypertension-mediated organ damage associated with LVH.

KEYWORDS: hypertension, hypertensive heart disease, left ventricular hypertrophy

INTRODUCTION

Cardiovascular disease remains a leading cause of death and disability, with hypertension as a major contributing factor[1,2]. Prolonged hypertension affects the left ventricle, leading to left ventricular hypertrophy (LVH) due to increased blood pressure (BP) and neurohormonal activation[3]. LVH is an early indication of cardiac damage, classified as hypertension-mediated organ damage, and is associated with cardiovascular events from conditions such as heart failure, diastolic dysfunction, stroke, congestive heart failure, coronary artery disease (CAD), ventricular arrhythmia, and sudden cardiac death[4-8].

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BP control is key to LVH management, reducing its incidence and improving prognosis[9,10]. Early detection of LVH is crucial for risk stratification and appropriate intervention.

Echocardiography is used to assess LVH; it offers greater sensitivity and accuracy than electrocardiography[11,12]. Research reveals that LVH identified through echocardiography can be a predictor of cardiovascular mortality[13-15]. LVH is a well-established consequence of long-standing uncontrolled hypertension. The prevalence of LVH among patients with hypertension varies from 24% to 72.20%[16-18], whereas studies in Thailand report prevalence rates between 28% and 62%, depending on diagnostic criteria used[19]. Key factors linked to LVH include male sex, advanced age, obesity, and increased BP[14,16,18-20]. However, the manifestation and risk profile of LVH can vary across ethnic groups due to genetic predisposition, environmental exposures, lifestyle behaviors (e.g., diet, salt sensitivity), and healthcare access. While international data provide useful insights, Thai-specific data are limited. Given the unique demographic and clinical characteristics of Thai patients, including differences in obesity patterns, dietary sodium intake, and hypertension control rates, studying LVH in this population is essential for more accurate risk stratification and targeted interventions.

Out-of-office BP measurement, such as ambulatory BP monitoring and home BP monitoring (HBPM), have shown a stronger correlation with LVH than in-office measurement[21,22]. Despite these known associations, data on LVH and its relationship with BP control in Thai hypertensive patients remain scarce.

The current study aimed to determine the prevalence of LVH diagnosed using echocardiography and investigate its relationship with office BP and home BP control in patients with hypertension admitted at Vajira Hospital, Navamindradhiraj University.

The primary objectives of this study were to confirm the prevalence of LVH in hypertensive patients at Vajira Hospital and examine the relationship between BP control and presence of LVH using echocardiography in Thai patients with hypertension and investigate its relationship with office BP and home BP control. By focusing on a Thai cohort, we sought to provide region-specific insights that may differ from those reported in other populations and inform clinical management. The secondary objective was to identify other factors associated with LVH in these patients.

METHODS

This single-center cross-sectional study was approved by the Ethical Committee of the Faculty of Medicine Vajira Hospital, Navamindradhiraj University (certificate of approval 044/2567 protocol 017-67). The study population included patients diagnosed with hypertension who visited the cardiology clinic of the Faculty of Medicine Vajira Hospital, Navamindradhiraj University between March 01, 2024, and August 31, 2024. The inclusion criteria were patients aged ≥ 18 years and those who had their own BP monitoring apparatus and can perform home BP measurement. Patients with a clinical diagnosis of hypertension were eligible regardless of whether they were treated with antihypertensive medications or managed with lifestyle modification alone. No changes to antihypertensive therapy were made before enrollment, and all participants were enrolled during routine clinical follow-up. In contrast, the exclusion criteria were patients with a poor echocardiographic window, patients with moderate or severe valvular heart disease, and patients with comorbidities including atrial fibrillation, secondary hypertension, other myocardial diseases/cardiomyopathies, left ventricular dysfunction (left ventricular ejection fraction of less than 40%), and congenital heart disease.

Electronic medical records and patient interviews for baseline characteristics, including age, sex, body mass index (BMI), smoking status, duration of hypertension, hypertension treatment status, number of antihypertensive drug classes prescribed, and comorbidities, were studied. Smoking history was defined as current or former smoking of ≥ 100 cigarettes in a lifetime (quantified in pack-years). Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate < 60 mL/min/1.73 m^2 for ≥ 3 months (CKD- exocrine pancreatic insufficiency (EPI) equation). CAD was defined by prior myocardial infarction, history of percutaneous coronary intervention or coronary artery bypass grafting, or angiographic evidence of ≥ 50% stenosis in a major coronary artery.

Office BP was measured with the patient in a seated position, using the arm for measurement. Systolic BP (SBP) and diastolic BP (DBP) was taken twice, 5 minutes apart. The average of these two readings was considered the office SBP and DBP. If patients had previous treatment records in their medical history with BP measurements taken within the past 6 months, the values were averaged to verify whether the patient's hypertension was controlled or uncontrolled. An SBP < 140 mmHg and DBP < 90 mmHg were considered controlled office BP.

The patients were advised to record their home BP in the morning and evening for 1 month with a semiautomatic BP apparatus of any brand or model. All home BP values were recorded within one month before echocardiography, and no changes to antihypertensive medications were made during this monitoring period. They were instructed to measure their BP at home twice daily—once in the morning (between 7:00 AM and 10:00 AM) and once in the evening (between 5:00 PM and 8:00 PM), at a consistent time each day. Each measurement consisted of two consecutive readings, and the average of these two readings was recorded. These time windows were chosen based on standard recommendations that morning BP should be measured within 1 hour of waking and before medication or meals, and evening BP should be measured before bedtime, consistent with major hypertension guidelines[23,24]. Moreover, they were asked to record the measurements for 1 month in a BP logbook provided by the researcher. The average of the morning and evening readings were used to obtain the home SBP and DBP. An SBP < 130 mmHg and DBP < 80 mmHg were considered controlled home BP. Patients were then categorized into four groups based on BP control status: (1) controlled both office and home BP, (2) uncontrolled both office and home BP, (3) uncontrolled office BP but controlled home BP, and (4) controlled office BP but uncontrolled home BP.

Two-dimensional transthoracic echocardiography was performed in all participants using a Philips EPIQ CVx machine after completing the one-month of BP recording. Echocardiographic variables included interventricular septal diameter in diastole (IVSd), left ventricular diameter in diastole (LVDd), left ventricular posterior wall thickness in diastole (LVPWd), and relative wall thickness (RWT). LVH by echocardiography was defined according to the criteria of the American Society of Echocardiography: LV mass index (LVMI) > 115 g/m^2 for men and > 95 g/m^2 for women, measured using the 2D-linear measurement method. The LV mass is calculated using 0.8 x 1.04 x [(IVSd + LVDd + LVPWd)^3 – LVDd^3]+ 0.6 grams[25]. LV mass was then indexed to body surface area to obtain the LVMI, expressed in g/m^2. Furthermore, RWT was calculated using the formula RWT = ((2 × LVPWd)/LVDd). The types of LVH were classified by the geometric patterns into concentric hypertrophy (RWT > 0.42) and eccentric hypertrophy (RWT ≤ 0.42). Concentric remodeling was defined as LVMI not meeting the criteria for LVH and RWT > 0.42. Image acquisition was performed by a cardiology fellow in training, using standardized parasternal long-axis views. Each echocardiographic study took approximately 10–20 minutes.

Measurements of IVSd, LVDd, LVPWd, LV mass, and RWT were independently obtained by the acquisition operator. All measurements were then reviewed by a board-certified cardiologist with expertise in echocardiography, who was blinded to the patients' BP status.

The prevalence of LVH in patients was presented in numbers and percentages. Continuous variables with a normal distribution were demonstrated as mean and standard deviation (SD) (mean ± SD). Continuous variables with skew distribution were shown as the median and interquartile range (IQR) (median ± IQR).

The sample size for this study was calculated based on two primary objectives. For the first objective—determining the prevalence of LVH in patients with hypertension—the sample size was estimated using the formula for a single proportion. Based on a previous study reporting an LVH prevalence of 36%[17], with a 95% confidence level (Z = 1.96) and a margin of error of 10%, the required sample size was 89 participants. After accounting for an estimated 10% rate of incomplete data, the adjusted sample size was 98 participants. For the second objective—assessing the association between BP control (office and home BP) and the presence of LVH—the sample size was calculated using the formula for comparing two proportions. Based on reported LVH prevalences of 32% in patients with controlled BP and 17% in those with uncontrolled BP[21], with a power of 80% ($Z\beta$ = 0.84) and a significance level of 0.05 ($Z\alpha$ = 1.96), the required sample size was 126 participants per group. After adjusting for 10% data incompleteness, the total required sample size was 277 participants. Therefore, we used 277 as the final sample size for this study.

Analysis of the correlation between BP control (i.e., office BP measurement and home BP) and LVH employed regression analysis. We used multinomial logistic regression to assess the relationship between the number of antihypertensive drug classes and BP control categories. Univariate and multivariate logistic regression analyses were used to determine the predictive factors for LVH. Data were analyzed using the statistical software STATA version MP17.

RESULTS

This study enrolled 107 hypertensive patients who visited the cardiology clinic between March 1, 2024, and August 31, 2024. The mean age of the patients was 68.80 ± 10.20 years, and 73% were females. The mean BMI of the patients was 25.46 ± 4.56 kg/m^2. Smoking history was found in 19.60% of the patients. The most common comorbidities included diabetes mellitus (38.30%), dyslipidemia (86.90%) and coronary artery disease (36.40%). The mean duration of hypertension was 13.54 ± 8.55 years. Moreover, the mean number of antihypertensive drug groups used was 2.50 ± 1.20. Regarding echocardiographic parameters, the patients' mean LVMI was 98.80 ± 32.20 g/m^2. The mean IVSd was 1.07 ± 0.23 cm. The mean LVPWd was 1.08 ± 0.23. Additionally, the mean LVDd was 4.28 ± 0.64 cm, and the mean RWT was 0.51 ± 0.16.

The baseline characteristics, including various clinical, demographic, and echocardiographic variables, were grouped into four according to patterns of LVH: all patients, normal geometry thickness, concentric remodeling, and concentric hypertrophy (Table 1). No patient met the definition of eccentric hypertrophy. The concentric hypertrophy group had the highest mean age (72.30 years), BMI (26.42 kg/m^2), duration of hypertension (17.50 years), and average use of antihypertensive drugs (3.20 kinds).

Table 1 Patient baseline characteristics and echocardiographic values (n = 107)

	All (n = 107)	Normal geometry (n = 24)	Concentric remodeling (n = 48)	Concentric hypertrophy (n = 35)	P-value
Age (mean ± SD)	68.77 ± 10.22	63.91 ± 10.57	68.62 ± 9.01	72.31 ± 10.39	0.007
BMI (mean ± SD)	25.46 ± 4.56	23.78 ± 3.97	26.42 ± 4.55	25.09 ± 4.69	5.829
Male (%)	29 (27.10%)	5 (20.83%)	20 (41.67%)	4 (11.43%)	0.007
Smoking history (%)	21 (19.63%)	5 (20.83%)	13 (27.08%)	3 (8.57%)	0.109
Duration HT (year)	13.54 ± 8.55	8.75 ± 7.91	13.00 ± 8.00	17.50 ± 8.00	< 0.001
anti-HT Drug (number)	2.50 ± 1.20	1.83 ± 1.09	2.45 ± 1.03	3.25 ± 1.17	< 0.001
Diabetes mellitus (%)	41 (38.32%)	6 (25.00%)	19 (39.58%)	16 (45.71%)	0.267
Dyslipidemia (%)	93 (86.92%)	20 (83.33%)	41 (85.42%)	32 (91.43%)	0.609
Stroke (%)	17 (15.89%)	4 (16.67%)	9 (18.75%)	4 (11.43%)	0.662
CAD (%)	39 (36.45%)	11 (45.83%)	12 (25.00%)	16 (45.71%)	0.085
CKD (%)	25 (23.30%)	1 (4.17%)	12 (25.00%)	12 (34.29%)	0.025
LV mass index (g/m^2)	98.80 ± 32.20	80.70 ± 13.40	85.30 ± 15.30	129.80 ± 36.20	
Men	99.56 ± 22.73	79.56 ± 21.96	95.93 ± 10.98	142.75 ± 13.25	< 0.001
Women	98.51 ± 35.21	80.96 ± 11.01	77.64 ± 13.25	128.11 ± 38.00	< 0.001
IVSd (cm)	1.07 ± 0.23	0.88 ± 0.12	1.06 ± 0.17	1.21 ± 0.26	< 0.001
LVPWd (cm)	1.08 ± 0.23	0.81 ± 0.09	1.11 ± 0.19	1.21 ± 0.19	< 0.001
LVIDd (cm)	4.28 ± 0.64	4.55 ± 0.49	4.01 ± 0.56	4.47 ± 0.71	< 0.001
RWT	0.51 ± 0.16	0.35 ± 0.39	0.57 ± 0.14	0.56 ± 0.17	< 0.001

Abbreviations: BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; cm, centimeter; g/m^2, grams per square meter; HT, hypertension; IVSd, interventricular septal diameter in diastole; LV, left ventricle; LVIDd, left ventricular diameter in diastole; LVPWd, left ventricular posterior wall thickness in diastole; n, number; RWT, relative wall thickness; SD, standard deviation
Data are presented as n (%) of row total.

The prevalence of echocardiography-diagnosed LVH was 32.70% (24 of 107 patients), which is consistent with hypertensive heart disease. All patients with LVH met the geometric pattern of concentric hypertrophy. The remaining 83 (67.30%) patients manifested no LVH on echocardiography. Of these patients, 48 (44.90% of total population) met the concentric remodeling criteria.

The percentage of patients achieving target office BP and home BP were 42.10% and 59.80%, respectively. These results can be further classified into the following categories. A 37.40% had both controlled office and home BP, 35.50% had both uncontrolled office and home BP, 4.70% had controlled office BP but uncontrolled home BP, and 22.40% had uncontrolled office BP but controlled home BP. When stratified by BP control patterns, LVH was present in 9 of 40 patients (22.50%) with both controlled office and home BP, 17 of 38 patients (44.70%) with both uncontrolled office and home BP, 1 of 5 patients (20.00%) with controlled office BP but uncontrolled home BP, and 8 of 24 patients (33.30%) with uncontrolled office BP but controlled home BP. Table 2 demonstrates BP control and the prevalence of LVH in each BP control category.

In the multinomial model, each additional antihypertensive agent was associated with 1.83-fold higher odds of having both uncontrolled office and home BP (95% CI 1.20–2.78; p = 0.005), with no significant associations in the other BP categories. Accordingly, the number of anti-hypertensive agents was included in the LVH multivariate analysis.

Table 2 Blood pressure control and LV geometry in each blood pressure control category

Blood pressure categories	Normal geometry (n = 24)	Concentric remodeling (n = 48)	Concentric hypertrophy (n = 35)
Controlled office BP and controlled home BP, n (%)	19 (47.50)	12 (30.00)	9 (22.50)
Uncontrolled office BP and uncontrolled home BP, n (%)	1 (2.63)	20 (52.63)	17 (44.74)
Uncontrolled office BP but controlled home BP, n (%)	3 (12.50)	13 (54.17)	8 (33.33)
Controlled office BP but uncontrolled home BP, n (%)	1 (20.00)	3 (60.00)	1 (20.00)

Abbreviations: BP, blood pressure; HT, hypertension; LV, left ventricular; LVH, left ventricular hypertrophy; n, number
Data are presented as n (%) of row total.

Univariate analysis found that the number of antihypertensive drugs use and uncontrolled both office and home BP subgroup are predictive factors of LVH. However, after adjusting for other variables in the multivariate analysis, the number of anti-hypertensive agent use remained significantly associated with LVH (Table 3.)

Table 3 Univariate and multivariate analysis of factors associated with LV hypertrophy

Characteristic	Univariate analysis			Multivariate analysis		
	Odds ratio	95% CI	P-value	Odds ratio	95% CI	P-value
Age	1.02	0.96-1.08	0.581	1.02	0.97-1.08	0.417
BMI	0.96	0.85-1.09	0.551			
Male	0.57	0.13-2.54	0.464	0.48	0.13-2.55	0.476
Smoking	0.40	0.07-2.19	0.291	0.23	0.07-1.91	0.229
Duration of hypertension	1.05	0.98-1.13	0.194	1.04	0.97-1.11	0.243
Number of anti-hypertensive drug	1.90	1.14-3.15	0.013	1.88	1.14-3.09	0.013
Diabetes mellitus	0.99	0.33-3.02	0.997			
Dyslipidemia	0.96	0.17-5.44	0.964			
Stroke	0.56	0.13-2.45	0.440			
CAD	1.96	0.67-5.70	0.219	2.34	0.86-6.36	0.095
CKD	1.29	0.33- 4.88	0.730	1.22	0.37-4.05	0.744
Controlled office BP	0.44	0.04-5.50	0.526			
Controlled home BP	1.12	0.09-13.64	0.931			
BP control categories						
Controlled both office and home BP (reference)	1			1		
Uncontrolled both office and home BP	2.79	1.05-7.43	0.040	1.84	0.58-5.98	0.309
Uncontrol office BP but controlled home BP	1.72	0.56-5.32	0.345	1.13	0.30-4.23	0.860
Controlled office BP but uncontrolled home BP	0.86	0.09-8.71	0.899	0.80	0.07-9.32	0.857

Abbreviations: BMI, body mass index; BP, blood pressure; CAD, coronary artery disease; CI, confidence interval; CKD, chronic kidney disease; HT, hypertension; LV, left ventricular; n, number
Multivariate model: logistic regression including age, sex, smoking history, duration of hypertension, number of hypertensive drugs, CAD, CKD and blood pressure–control category.

We also performed a sensitivity analysis which applied more stringent control criteria defining controlled BP as < 130/80 mmHg for office BP and < 120/70 mmHg for home BP measurements and re-examined its association with LVH. We found only the "uncontrolled office but controlled home BP" category was associated with significantly higher odds of LVH (OR 9.17; 95% CI 1.15–73.24; p = 0.0037) in univariate analysis. However, after adjusting for other variables in the multivariate analysis, it does not show statistically significant (Table 4 and Table 5).

DISCUSSION

The current study focuses on the prevalence of LVH diagnosed by echocardiography in patients with hypertension at Vajira Hospital, Navamindradhiraj University, which may represent an urban population. Moreover, the relationship between LVH and BP control status was assessed, and other factors associated with the echocardiographic evidence of LVH in this patient population were identified.

Table 4 Univariate sensitivity analysis using stricter BP thresholds (office BP < 130/80 mmHg, home BP < 120/70 mmHg)

BP control categories	odds ratio	95% CI	P-value
Controlled both office and home BP (reference)	1		
Uncontrolled both office and home BP	1.69	0.43-6.63	0.454
Uncontrol office BP but controlled home BP	9.17	1.15-73.2	0.037
Controlled office BP but uncontrolled home BP	1.63	0.29-9.26	0.582

Abbreviations: BP, blood pressure; CI, confidence interval

Table 5 Multivariate analysis of factors associated with LV hypertrophy using stricter BP thresholds (office BP < 130/80 mmHg, home BP < 120/70 mmHg)

Characteristic	Multivariate analysis		
	odds ratio	95% CI	P-value
Age	1.02	0.97-1.08	0.417
Male	0.63	0.13-2.92	0.476
Smoking	0.39	0.07-2.25	0.229
Duration of hypertension	1.03	0.96-1.10	0.243
Number of anti -hypertensive drug	2.18	1.29-3.69	0.013
CAD	1.99	0.72-5.51	0.095
CKD	1.20	0.35-4.13	0.744
BP control categories			
Controlled both office and home BP (reference)	1		
Uncontrolled both office and home BP	2.44	0.47-12.70	0.289
Uncontrol office BP but controlled home BP	12.78	0.97-167.86	0.052
Controlled office BP but uncontrolled home BP	2.90	0.36-23.12	0.314

Abbreviations: BMI, body mass index; BP, blood pressure; CAD, coronary artery disease; CI, confidence interval; CKD, chronic kidney disease; HT, hypertension; LV, left ventricular; n, number
Multivariate model: logistic regression including age, sex, smoking history, duration of hypertension, number of hypertensive drugs, CAD, CKD and blood pressure–control category.

This study revealed that approximately 32.70% of hypertensive patients have LVH, which is comparable to the results of a literature review by Cuspidi et al., reporting an LVH prevalence of 36%-41%, depending on the criteria used[17]. However, a recent study by Apitz et al., using the same echocardiographic LVH criteria, showed a relatively low LVH prevalence (20%) compared with our findings[26]. In contrast, a higher prevalence of LVH in patients with hypertension was demonstrated in studies by Behera et al. and Conrady et al., with rates of 66.50% and 55.20%-72.20%, respectively, despite using the higher threshold for LVH than those applied in our study[16,18]. The overall varying prevalence of hypertensive heart disease among global populations can be attributed to different echocardiographic criteria, diverse patient populations, and varying degrees of hypertension. Additionally, approximately 50% of our study patients was able to control their office BP or home BP, which could affect the degree of LV remodeling and thus contribute to the prevalence of LVH. Furthermore, this could indicate that urban patients, who are more educated and have access to HBPM, are more attentive to their health care. Notably, regarding the pattern of LVH, all the study patients demonstrated a concentric hypertrophy geometric pattern, which is consistent with findings from several studies[16,27-28]. However, ethnic-specific reference values for LVMI may affect the estimation of LVH prevalence in different populations. A prior Thai study by Wong et al[29]. (2008) reported lower normal LVMI values in healthy Thai adults compared to American Society of Echocardiography (ASE) guidelines. Therefore, applying ASE cut-offs (> 115 g/m² for men, > 95 g/m² for women) might underestimate hypertensive LVH in this population. Future research is warranted to validate the appropriateness of international reference thresholds in Thai cohorts and consider ethnicity-specific criteria for LVH diagnosis.

According to current hypertension guidelines, BP control should be assessed using out-of-office measurements, such as HBPM or ambulatory BP monitoring, due to their stronger association with target organ damage. Thus, patients with elevated office BP but controlled home BP—often labeled as having white coat hypertension—are classified as having controlled BP. Conversely, patients with normal office BP but elevated home BP—defined as having masked hypertension—are considered uncontrolled and at higher cardiovascular risk. This study showed the highest prevalence of LVH in patients who could not control both their office and home BP to the target. Interestingly, patients with controlled office BP but uncontrolled home BP, indicating marked hypertension, had the lowest prevalence of LVH than the other subgroups. Our result may be partly due to the relatively small number of patients in this subgroup or possible misclassification caused by short-term BP variability or incorrect home BP technique. Additionally, it is possible that the duration or severity of elevated home BP in these patients was insufficient to produce measurable structural cardiac changes such as LVH. Future studies with longitudinal data and larger subgroup samples are needed to better understand these findings. The present study showed a much lower prevalence than a study by Cuspidi et al., which identified individuals who have masked hypertension with normal office BP and increased ambulatory BP or home BP or both[30]. Moreover, even patients with controlled office BP and home BP can still develop LV hypertrophy. This highlights the need for focusing on both home BP control and office control in patients with hypertension.

The current study found that the number of antihypertensive drugs was the only predictive factor for LVH after multivariate analysis, differing from previous studies that identified male sex, advanced age, obesity, and elevated BP as significant factors[14,16,18-20]. This difference may reflect variations in study populations,

definitions of LVH, and BP measurement methods. Importantly, the number of antihypertensive agents likely reflects treatment resistance or disease severity rather than being an independent causal factor. We addressed this by adjusting for it in our model, following a multinomial logistic regression that linked higher drug use with poor BP control. Nonetheless, this variable should be interpreted cautiously in regression analyses.

Although patients with both uncontrolled office and home BP are at high risk for LVH, no significant association was observed in our study. This may be due to the limited number of participants in this subgroup, reducing the power to detect a meaningful difference. Larger studies are needed to confirm this finding. Moreover, some predictors in the multivariate analysis showed wide confidence intervals and lacked statistical significance. This is likely attributable to small subgroup sample sizes, which resulted in less precise estimates and insufficient power to detect meaningful associations. Larger studies are warranted to clarify the impact of these factors on LVH. Nevertheless, our sensitivity analysis using lower BP cut-off threshold showed that patients with uncontrolled office, but controlled home BP had significantly higher odds of LVH. This suggests that episodic clinic BP elevations may drive ventricular remodeling despite acceptable home readings in this subgroup. Given the small subgroup size and wide CI, these findings should be confirmed in larger, prospective studies.

This study has several limitations. First, as an observational study, causality between BP control and LVH cannot be established. Second, as a single-center study with a relatively small sample size, the findings may not be generalizable to broader populations. Although the sample size was adequate for addressing the primary objectives, it may have limited the statistical power for secondary analyses, increasing the risk of false-negative results. Third, the absence of ambulatory BP monitoring and limited data on factors such as antihypertensive drug classes, salt intake, and physical activity may affect the robustness of the associations observed. Fourth, home BP was assessed over a short period, which may not capture long-term control and could be influenced by the Hawthorne effect. Fifth, while validated devices were recommended, there was no independent calibration of home BP monitors, introducing potential variability. Sixth, we did not assess several important factors that may influence LVH, such as antihypertensive drug classes, high salt intake, and physical activity. Lastly, although all echocardiographic studies were reviewed by a board-certified cardiologist, the use of a single operator may introduce intra-observer variability, which could affect the consistency of LVH assessment. However, this approach also reduced inter-observer variability and ensured procedural consistency throughout the study. Despite these limitations, we believe that the study results are beneficial for daily clinical practice and in developing strategies to identify LVH and consider more hypertension control in these patient populations. Future studies should focus on the clinical outcomes in these patient populations to gain clearer insight into the long-term implications of LVH and its management.

CONCLUSION

The prevalence of LVH is high among patients with hypertension, particularly those with uncontrolled office and home BP. This emphasizes the need for effective hypertension management strategies to prevent hypertension-mediated organ damage associated with LVH.

CONFLICT OF INTEREST

The authors have no financial interest in any of the products mentioned in this article.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to restrictions.

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