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LEFT VENTRICULAR HYPERTROPHY AMONG HYPERTENSIVE PATIENTS: PREVALENCE AND DETERMINANTS IN A CROSS-SECTIONAL STUDY

Original Research

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ABSTRACT

Objective: This study aimed to assess the prevalence of left ventricular hypertrophy (LVH) and determine its clinical and demographic determinants in patients with hypertension.

Methods: This was a cross-sectional study including 200 hypertensive patients visiting a primary care clinic during January 2024 and June 2025. Demographic and clinical information, such as age, sex, body mass index (BMI), duration of hypertension, blood pressure (BP) control, and comorbidities were noted. Every respondent received an echocardiography to determine left ventricular mass (LVM) and left ventricular mass index (LVMI). Pearson correlation and multivariate logistic regression were used to establish relationships with LVH. A p-value of less than 0.05 was taken as a significant statistical value.

Results: Mean age of the participants was 57.8 ± 12.1 years and 50% of the respondents were male. The incidence of LVH was 34% (68/200), and concentric hypertrophy was the most widespread pattern (58.8%). LVMI revealed positive correlations with systolic BP ($r = 0.53$, $p < 0.001$) and duration of hypertension ($r = 0.45$, $p < 0.001$), and also BMI ($r = 0.36$, $p < 0.001$). The multivariate analysis indicated that longer hypertension duration (OR 2.8, 95% CI 1.6–4.9), uncontrolled BP (OR 3.2, 95% CI 1.85-5.7), greater BMI (OR 1.9 per 5 kg/m^2 increase, 95% CI 1.2-3.0) and male sex (OR 1.7, 95% CI 1.0-2.9) had an independent association with LVH.

Conclusion: LVH occurs in about one-third of hypertensive patients and is independently related to longer duration of the disease, uncontrolled BP, increased BMI and male gender. Timely echocardiographic screening and regulation of modifiable risk factors could help mitigate the cardiovascular morbidity of LVH.

Keywords: Echocardiography, Structural Heart Disease, Cardiovascular Risk, Hypertension, Left Ventricular Hypertrophy.

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INTRODUCTION

Hypertension is one of the major health challenges across the world and one of the key contributors to cardiovascular morbidity and mortality. [1] One of its most important heart complications is left ventricular hypertrophy (LVH), which is a structural adaptation of the myocardium to chronic overload of pressure. [2] LVH is an important indicator of target organ damage and an independent predictor of poor outcomes including heart failure, arrhythmias, myocardial infarction and sudden cardiac death. [3] Despite the high availability of antihypertensive treatment, the burden of LVH in hypertensive patients remains high and this underscores the need for early detection and intervention of the condition. [4]

The pathogenesis of LVH in hypertension is multifactorial and is caused by chronic hemodynamic stress, neurohormonal stimulation and metabolic factors. [5] Lifestyle factors such as diet and physical inactivity have been reported to have an impact on cardiovascular risk and may have an indirect influence on the development of LVH through the impact on lipid profile or general metabolic health. [6] Additionally, obesity has been implicated as a modifiable risk factor with studies exploring the correlation between obesity, metabolic characteristics and cardiovascular risk markers in different populations. [7,8] Other determinants like prolonged duration of hypertension, poor blood pressure (BP) control, male sex and coexisting diabetes mellitus have also been determined although the strength and consistency of these associations may differ among populations. [9,10]

Echocardiography is a reliable and non-invasive method for the evaluation of cardiac structure and function, including assessment of left ventricular mass (LVM) and geometry. [11] Prevalence and determinants of LVH in hypertensive populations are important to improve cardiovascular risk stratification and directing management approaches, as well. [12] Data from primary care settings are of special interest, because this is the first point of contact for the majority of hypertensive patients, but such data are limited in many regions. [13]

This study was designed to show the prevalence of LVH in hypertensive patients attending a primary care clinic, and to establish its important clinical and demographic determinants, such as age, sex, body mass index (BMI), duration of hypertension, BP control and diabetes mellitus. [14,15] Results of this study can be used in targeted screening and intervention to reduce the cardiovascular complications of LVH.

METHODOLOGY

This was a cross-sectional study aimed at determining the prevalence of LVH and the clinical and demographic determinants of LVH in hypertensive patients (Ref: 2024/10421BS) between January 2024 and June 2025. The institutional review board gave ethical approval and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

A total of 200 patients with a definite diagnosis of primary hypertension attending the outpatient clinics of the Department of Medicine were consecutively recruited. The inclusion criteria included adults (aged ≥30 years) who were diagnosed with hypertension according to the guidelines issued by Joint National Committee (JNC-8) (systolic BP ≥140 mmHg and/or diastolic BP ≥ 90 mmHg or antihypertensive drug use). Patients having secondary hypertension, valvular or congenital heart disease, ischemic cardiomyopathy or chronic kidney disease (stage 4 or higher) were excluded. Consecutive sampling was used to prevent selection bias.

A clinical assessment was conducted on each of the participants that was structured to record their age, sex, BMI, history of smoking and alcohol, hypertension duration, treatment, BP management. Standardized mercury sphygmanometer was used to measure the BP, with the mean of two measurements taken five minutes apart after rest.

The transthoracic echocardiography was done with a transducer of 2.5–3.5 MHz to evaluate the left ventricular wall thickness and dimensions as per the American Society of Echocardiography (ASE) guidelines. LVM was determined by the cube formula of Devereux et al. and the left ventricular mass index (LVMI) was obtained by dividing LVM by body surface area (g/m^2). . LVH was an LVMI of >115 g/m^2 in men and >95g/m^2 in women. Echocardiographic readings were blinded by cardiologists.

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Statistical analysis of data was done with the help of IBM SPSS version 27.0. Continuous variables were reported as mean ± SD and categorical variables were reported as percentages. Pearson correlation and multivariate logistic regression were applied to find the determinants of LVH ($p < 0.05$).

RESULTS

A total of 200 patients with hypertension were included. The average age was 57.8 ± 12.1 years with male and female representation evenly split (50% of each). The mean BMI was 27.6 ± 4.3 kg/m² and the average duration of hypertension was 8.4 ± 5.6 years. Uncontrolled BP was present in 39% of the patients, and 31% had concomitant diabetes mellitus (Table 1).

Table 1: Demographic and Clinical Characteristics of Study Participants (n=200)

Parameter	LVH (n = 68)	Non-LVH (n = 132)	Test	Test value	p-value
Age (years)	59.3 ± 11.7	57.0 ± 12.3	t-test	1.28	0.201
Gender					
Male, n (%)	40 (58.8%)	60 (45.5%)	Chi-square	3.27	0.071
Female, n (%)	28 (41.2%)	72 (54.5%)			
BMI (kg/m²)	29.3 ± 4.8	26.7 ± 4.1	t-test	3.16	0.002
Duration of Hypertension (years)	11.2 ± 7.3	6.8 ± 5.1	t-test	5.18	<0.001
Uncontrolled BP, n (%)	45 (66.2%)	33 (25.0%)	Chi-square	33.7	<0.001
Diabetes Mellitus, n (%)	24 (35.3%)	38 (28.8%)	Chi-square	0.85	0.356

Significance at $p < 0.05$. BMI = Body Mass Index; BP = Blood Pressure; LVH = Left Ventricular Hypertrophy.

Echocardiographic evaluation showed the presence of a mean LVM of 198.5 ± 42.7 g and a mean LVMI of 104.3 ± 22.5 g/m^2. The overall prevalence of LVH was 34% (68/200), and concentric hypertrophy was the predominant pattern (58.8%) (Table 2).

Table 2: Echocardiographic Parameters and LVH Prevalence

Parameter	Overall (n = 200)	LVH (n = 68)	Non-LVH (n = 132)	Test used	Test value	p-value
LV Mass (g)	198.5 ± 42.7	220.1 ± 35.4	186.2 ± 30.8	t-test	t = 6.5	<0.001
LV Mass Index (g/m²)	104.3 ± 22.5	121.4 ± 18.6	95.2 ± 14.3	t-test	t = 7.3	<0.001
LVH prevalence, n (%)	68 (34%)	68 (100%)	0 (0%)	–	–	–
LVH Pattern	–	40 (58.8%) / 28(41.2%)	–	–	–	–

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Parameter	Overall (n = 200)	LVH (n = 68)	Non-LVH (n = 132)	Test used	Test value	p-value
(Concentric / Eccentric)						

Significance at p < 0.05. LV = Left Ventricle; LVH = Left Ventricular Hypertrophy; LV Mass Index = LVMI.

LVMI was significantly positively correlated with systolic BP ($r = 0.53$, $p < 0.001$), duration of hypertension ($r = 0.45$, $p < 0.001$) and BMI ($r = 0.36$, $p < 0.001$). Age was weakly correlated with the presence of LVH ($r = 0.21$, $p = 0.064$) (Table 3).

Table 3: Correlation of LVMI with Clinical Parameters

Variable	LVMI (g/m²)	p-value
Systolic BP (mmHg)	r = 0.53*	<0.001
Duration of Hypertension (years)	r = 0.45*	<0.001
BMI (kg/m²)	r = 0.36*	<0.001
Age (years)	r = 0.21	0.064

Significance at p < 0.05. LVMI = Left Ventricular Mass Index; BMI = Body Mass Index; BP = Blood Pressure. Pearson correlation coefficient (r) used.

Multivariate logistic regression identified longer duration of hypertension (OR 2.8, 95% CI 1.6–4.9, $p < 0.001$), uncontrolled BP (OR 3.2, 95% CI 1.8–5.7, $p < 0.001$), higher BMI per 5 kg/m² increase (OR 1.9, 95% CI 1.2–3.0, $p = 0.005$), and male sex (OR 1.7, 95% CI 1.0–2.9, $p = 0.042$) as independent predictors of LVH. Age and diabetes mellitus were not found to be independently associated with LVH (Table 4).

Table 4: Multivariate Logistic Regression for Predictors of LVH

Variable	OR	95% CI	p-value
Duration of Hypertension (per 5 years)	2.8	1.6–4.9	<0.001
Uncontrolled BP	3.2	1.8–5.7	<0.001
BMI (per 5 kg/m² increase)	1.9	1.2–3.0	0.005
Male Sex	1.7	1.0–2.9	0.042
Age	1.1	0.9–1.3	0.218
Diabetes Mellitus	1.3	0.8–2.2	0.287

Significance at p < 0.05. OR = Odds Ratio; CI = Confidence Interval; BMI = Body Mass Index; BP = Blood Pressure; Logistic regression model adjusted for all listed variables.

DISCUSSION

In this study, LVH was found in about one-third of hypertensive patients with concentric hypertrophy being the most frequent pattern. The results suggest that LVH is not only common among patients with high BP but also strongly affected by longer duration of hypertension, uncontrolled BP, higher BMI, and male gender. These findings underscore the ongoing burden of target organ damage in

hypertensive patients even in primary care areas, where routine management is accessible. [16] Moreover, metabolic processes including the disrupted regulation of glucose and insulin have also been reported to affect cardiac structure, and they may also be the cause of the LVH development in hypertensive groups. [17]

The positive relation between LVH and hypertension duration highlights the additive effects of chronic pressure overload on cardiac structure. [18] Patients with long-standing hypertension are subjected to long term hemodynamic stress, resulting in myocardial remodeling and hypertrophy [19]. In animal models, experimental evidence has demonstrated that cardiac remodeling can be aggravated by chronic metabolic and inflammatory stress, which is mechanistic evidence of the role played by prolonged hypertension in LVH. **[20]** In addition, uncontrolled BP was a significant determinant, further supporting the idea that insufficient BP control is known to increase the pace of cardiac structural changes. **S**tudies that have been conducted on chronic diseases populations indicated that metabolic imbalances and associated endocrine factors might also modulate this process. **[21]** The association between higher BMI and the presence of LVH emphasizes the role of metabolic factors, in which obesity leads to an extra hemodynamic burden and facilitates maladaptive cardiac remodeling. [22] The higher prevalence of LVH in males could be explained by sex differences in the myocardial response to pressure stress, which may be hormonally and genetically mediated. [23,24]

These results are generally in agreement with those reported in prior studies in heterogeneous populations. [25] Several cross-sectional and cohort studies have described similar prevalence of LVH in hypertensive patients with concentric remodeling as the predominant pattern. [26] The additional context on fluctuation in the prevalence and expression of risk factors of LVH and variability in nutritional status, anemia, and systemic stress factors can be offered by broader population-based studies. **[27,28]** In addition, the relationships with more prolonged duration of disease, uncontrolled hypertension, and obesity have been clearly described in the literature, strengthening the global applicability of these risk factors. In addition, other studies have shown that males are more susceptible than females to hypertensive cardiac remodeling, but this effect of sex seems also to vary across ethnicities and age groups. The finding of no independent relationship between diabetes and LVH in our cohort is in contrast to some studies that reported a synergistic effect of hyperglycemia on myocardial hypertrophy. *Mechanistic studies, including experimental models of diabetes, suggest that hyperglycemia and oxidative stress can contribute to myocardial hypertrophy, although this effect may not always be observed in human populations.* [29,30]

The biological mechanisms of these associations are complex hemodynamic, neurohormonal, and molecular pathways. [31] Chronic pressure overload causes an increase in the stress of the myocardial wall, which stimulates myocyte hypertrophy and deposition of extracellular matrix. [32] Neurohormonal activation, such as that involving the renin-angiotensin-aldosterone system, further advances hypertrophic signaling, fibrosis and adverse remodeling. [33] Obesity is involved by increased cardiac output, systemic inflammation and metabolic dysregulation, which exaggerate hypertrophic response. [34] Sex differences in myocardial structure and function, mediated, in part, by sex hormones and receptor signaling may account for the predisposition among males. [35]

These findings have important clinical implications for the early diagnosis and follow-up of LVH in hypertensive patients. [36] Echocardiography is still a useful tool for identifying structural abnormalities before the occurrence of overt cardiovascular events. [37] It is important that BP control, lifestyle modification and weight control are achieved for the purpose of preventing progression to hypertrophy and associated complications such as heart failure, arrhythmias and ischemic heart disease. [38] In addition, the identification of subgroups of individuals at high risk of adverse cardiovascular events such as males and patients with long-standing hypertension may guide personalized preventive interventions. [39,40]

This study has a number of limitations. The cross-sectional design restricts the possibility of inferring any causal effects, and results may not be generalizable to a population other than the primary care setting. Echocardiographic measurements were done at a single time point, which did not allow analysis of longitudinal changes. Prospective follow-up studies are needed to investigate the progression of LVH, the effect of specific interventions and possible interactions with metabolic and genetic factors in a variety of populations.

CONCLUSION

LVH occurs in one third of hypertensive patients and is most often of the concentric type. Its development is strongly associated with longer and duration of hypertension, uncontrolled BP, higher BMI and male sex. Early detection by echocardiographic screening and strict management of the modifiable risk factors including BP control and weight reduction is essential in order to reduce the risk of adverse cardiovascular outcomes in this population.

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ABBREVIATIONS:

ASE – American Society of Echocardiography
BMI – Body Mass Index
BP – Blood Pressure
CI – Confidence Interval
DBP – Diastolic Blood Pressure
DM – Diabetes Mellitus
HF – Heart Failure
JNC-8 – Eighth Joint National Committee
LV – Left Ventricle
LVM – Left Ventricular Mass
LVH – Left Ventricular Hypertrophy
LVMI – Left Ventricular Mass Index
OR – Odds Ratio
SBP – Systolic Blood Pressure
SD – Standard Deviation
SPSS – Statistical Package for the Social Sciences
USA – United States of America

AUTHOR CONTRIBUTIONS

Author	Contribution
Fazeelat Anwar	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Muhammad Ahmed	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Muhammad Akram*	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published

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