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FREQUENCY AND DETERMINANTS OF LEFT VENTRICULAR HYPERTROPHY IDENTIFIED ON ECHOCARDIOGRAPHY IN HYPERTENSIVE PATIENTS TO CARDIOLOGY DEPARTMENT OF DHQ TEACHING HOSPITAL MIRPUR AJK

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Abstract

Objective: *To observe the Frequency and Determinants of Left Ventricular Hypertrophy identified on Echocardiography in Hypertensive Patients presenting to Cardiology Department of DHQ Teaching Hospital Mirpur AJK Study*
Design: *Cross Sectional Observational Study*
Study Duration: *06 months from*
Study Place: *Divisional Head Quarter Hospital Mirpur Azad Kashmir*
Methods: *Total 100 patients were observed in this study after taking informed written consent and ethical approval from hospital ethical review committee. BMI, blood pressure, sugar levels, age and gender of all the participants were recorded. In our study, 36 patients were male and 64 were females. All patients underwent 2D echocardiography to check for left ventricular hypertrophy, which was categorized as mild, moderate and severe. 2D echo was performed by same operator to remove biases.*
Results: *In our study, we observed 100 participants, among them 64 were females and 36 were males. Only those patients were included who have hypertension. In our study, the mean age was 55±5 years, average random sugar levels were 170±10 and BMI was in the range of 25-33. The blood pressures were SBP 140±10mmHg-95±7mmHg. Our study revealed that out of 100 patients 83 (83%) had LVH, among those 83, 39 (46.9%) had mild LVH, 27 (32.5%) had moderate LVH and 17 (20.4%) had severe LVH. The most significant factor leading to LVH was uncontrolled hypertension (96.3%), second was obesity (44.5%) followed by age (36.1%).*
Conclusion: *From our study, we can conclude that left ventricular hypertrophy has strong association with hypertension. Furthermore, we can also conclude that obesity and age in the setting of hypertension also have relationship with LVH.*

INTRODUCTION

High blood pressure, along with its associated risks for heart and kidney diseases, represents a significant global public health issue. Cardiovascular diseases (CVDs) are among the leading causes of death and disability globally.[1,2] High blood pressure is a major risk factor for cardiovascular diseases, significantly contributing to disability. It accounts for 57% of stroke-related deaths and 24% of deaths caused by coronary heart disease.[3]

Left ventricular hypertrophy (LVH) is one of the earliest signs of organ damage in individuals with hypertension and serves as a strong, independent indicator of cardiovascular risk, including both mortality and morbidity. [4] Numerous studies have demonstrated that the prevalence of left ventricular hypertrophy (LVH) rises with factors such as advancing age, obesity, gender, and elevated blood pressure.[5] The Framingham Heart Study has established a correlation between increased left ventricular (LV) mass and obesity, indicating that higher body weight contributes to LV hypertrophy (LVH). Early detection of LVH, along with appropriate risk assessment and proactive treatment, is crucial to reduce the risk of cardiovascular diseases and associated mortality.[6] Studies indicate that the evaluation of cardiovascular risks in hypertensive patients is often insufficient, with limited data available on left ventricular hypertrophy (LVH) in this population, particularly in this region. Therefore, this study was undertaken to examine the prevalence and patterns of LVH and identify the factors associated with its development in hypertensive patients.

Methodology:

Total of 100 patients were included in our study after taking informed written consent from patients and ethical approval by institution review board. Age and gender of all the patients was noted along with there weight and area of residence. Patients were also asked about co-morbid conditions, there sugar levels and blood pressure were also checked. There BMI was also written down. Every patient included in the study underwent 2D- echocardiography to check for left ventricular hypertrophy and septal thickness. In our study 2D echo was done by single operator so as to remove biasness in the study. 2D echo was done in left lateral decubitus position. The mode taken was M-mode as per guidelines of American Society of Echocardiography.

Inclusion criteria included:

1. Patients with hypertension whether primary or secondary
2. Patients age greator than 25 years
3. Either gender

Exclusion criteria included:

1. Unwilling for studies
2. Patients with morbid obesity, BMI>40
3. Prior heart failure
4. Patients with congenital heart disease

The data was analysed using SPSS version 23. Normally distributed variables were presented as mean and standard deviation. A p value of less than .05 was considered as statistical significant. Multiple Regression Model and Mann- Whitney equation was also used.

Results:

In our study, total 100 patients were included in the study. Among the 100 patients, 64 were females and 36 were males. (table 1.1) The mean age of participants was 55±5 years.

The average random sugar levels were 170±10 and BMI was in the range of 25-33. The blood pressures were SBP 140±10mmHg-95±7mmHg. Our study revealed that out of 100 patients 83 (83%) had left Ventricular Hypertrophy (LVH).

Patients with LVH were further divided into mild, moderate and severe left ventricular hypertrophy.

39 (46.9%) patients had mild LVH, 27 (32.55) had moderate LVH and severe LVH was present in 17 (20.4%) participants. (Table 1.2, and Chart 1.1). Among the patient affected with LVH, the most number of patients were from female population, the number being 49 (59%).

Left Ventricular Mass (LVM) was also determined in our study and it was divided into concentric and eccentric based on left ventricular wall thickness. Among 83 subjects who had LVH, they were further divided into bases of LVM into 04 groups. 14 (16.8%) had eccentric non dilated LVM, 25 (30.1%) had eccentric dilated LVM, 32 (38.5%) had concentric

dilated and 12 (14.4%) had concentric dilated LVM. (Chart 1.2)

The most significant factor leading to LVH was uncontrolled hypertension (96.3%), second was obesity (44.5%) followed by age (36.1%), depicted in pie chart 1.3

Gender	
Female n= 64	Male n= 36

Table 1.1

Severity of LVH		
Mild LVH	Moderate LVH	Severe LVH
39	27	17

Table 1.2

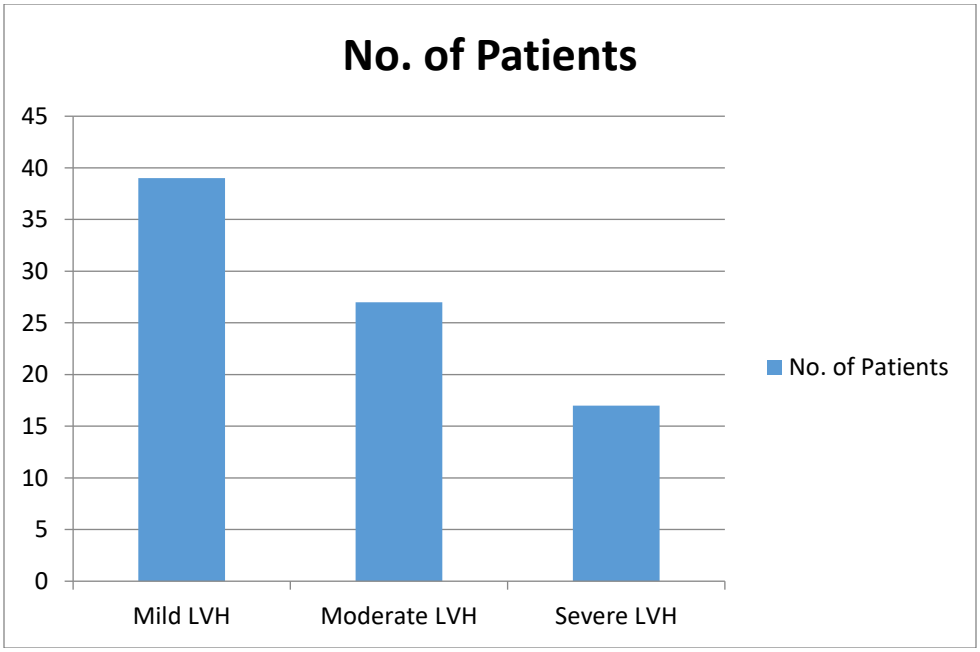


Chart 1.1 (Severity Of LVH)

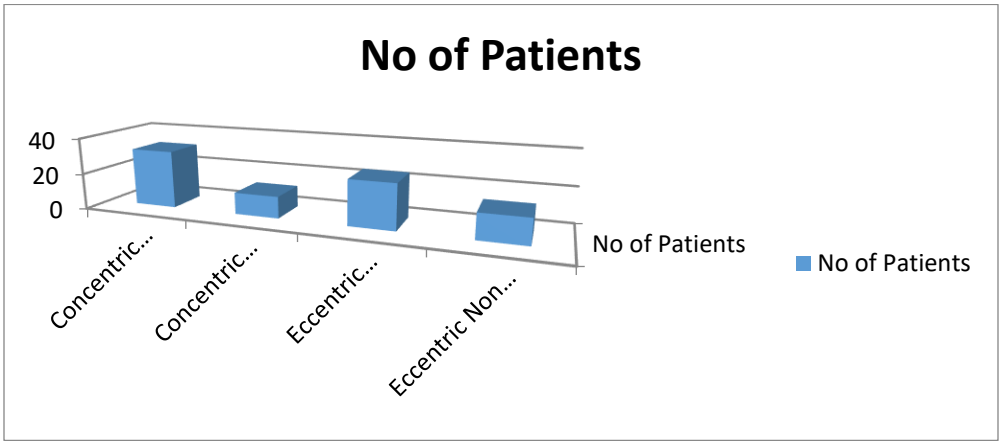
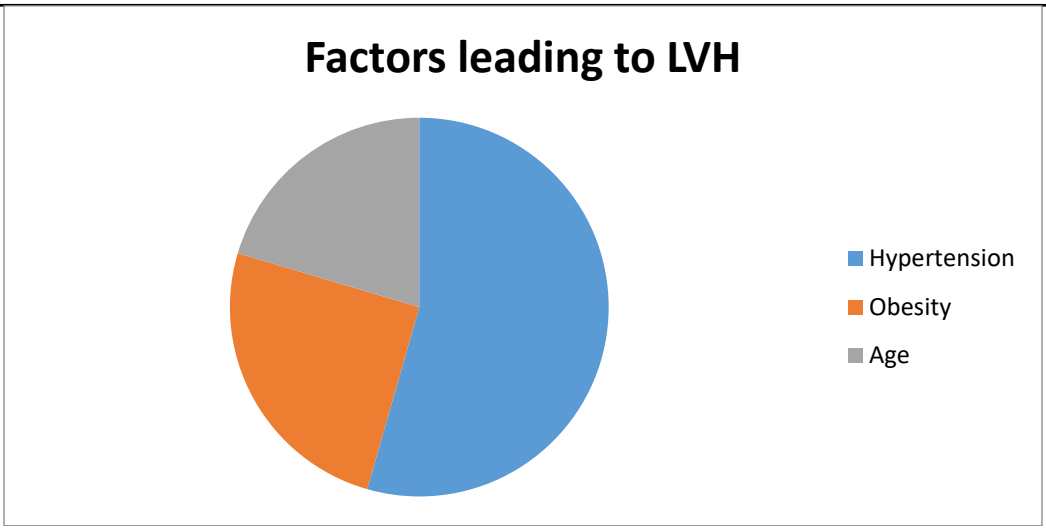


Chart 1.2 (No of patients showing LVM)



Pie Chart 1.3 (factors leading to LVH)

Discussion:

The study found that one in four hypertensive patients had LVH, even though 41.5% had achieved their target blood pressure. This prevalence was similar to the 23-26% reported in primary care settings in New York and Rome [7,8].

The prevalence observed in this study was higher than that reported in primary care studies from Japan (15%) and the United States (19%). This study aims to highlight the growing concern over the increasing cases of hypertension in Mirpur, AJK, particularly in the context of the rising incidence of hypertension across South Asia, driven by aging populations and lifestyle changes. [8,9] Previous studies have shown that obesity significantly increases the risk of developing LVH. In this study, obesity was identified as a key predictor of LVH among hypertensive patients. Obese patients were found to have twice the odds of developing LVH compared to those with normal weight. Additionally, the odds of developing LVH were 1.4 times higher in obese individuals compared to overweight individuals, although this difference was not statistically significant.[10] Similar findings were reported by Kathrotia et al., who highlighted the association between obesity and LVH, potentially linked to obesity cardiomyopathy, which is characterized by the presence of LVH.11

This study also found that systolic blood pressure (SBP) and diastolic blood pressure (DBP) were independent determinants of LVH, suggesting that lowering either SBP or DBP could significantly reduce LVH. This observation aligns with findings from various studies, emphasizing the importance of blood pressure management targeting both SBP and DBP to prevent the progression of LVH.

Additionally, left atrial enlargement, which serves as a marker of overall cardiovascular risk, was frequently observed in hypertensive patients, as reported by Cuspidi et al. 12 This abnormality showed a strong association with LVH. In this study, LVH was found to be 1.4 times more likely with each unit increase in left atrial diameter (p=0.088). Given its significance, the European Society of Cardiology strongly recommends measuring left atrial size during echocardiographic evaluations.

A study was conducted by Chinese Researchers in 2012 which was a community-based cross-sectional study, and it comprised 4270 hypertension patients with integrated clinical and echocardiographic data. Left ventricular mass was measured by transthoracic echocardiography. LVH was diagnosed by using the criteria of over 49.2 g/m(2.7) for men and 46.7 g/m(2.7) for women. LV geometric patterns (normal, concentric remodeling, concentric or eccentric hypertrophy) were calculated according to LVH and relative wall thickness. The prevalence of LVH was 42.7% in 4270 hypertensive patients, with 37.4% in males and 45.4% in females, respectively. Study concluded that female gender, body mass and systolic blood pressure are major factors contributing to the left ventricular hypertrophy.[13]

A study conducted in Ethiopia General hospital by Geri et ell showed that Left ventricular hypertrophy was found to be highly prevalent in hypertensive patients in Ethiopia. It was hospital based cross sectional study and it was conducted on 200 hypertensive patients on treatment in southwest Ethiopia. The study further correlated effects of ACE inhibitors on LVH and found positive correlation.[14]

Left ventricular hypertrophy [LVH, high left ventricular mass (LVM)] is traditionally classified as concentric or eccentric based on left ventricular relative wall thickness. Nine hundred thirty-nine participants in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) echocardiography substudy had measurable LVM at enrolment. Patients with LVH (LVM/body surface area ≥116 g/m in men and ≥96 g/m in women) were divided into four groups; eccentric nondilated, eccentric dilated concentric nondilated and concentric dilated. 12% had eccentric nondilated, 20% eccentric dilated, 29% concentric nondilated, and 14% concentric dilated LVH, with normal LVM in 25%.[15]

30% of hypertensives have been reported to have LVH. The estimated prevalence of structural remodelling is increased to 50-60% of the same group of subjects when 'low-SD' measurements such as wall thickness and the wall thickness: internal radius ratio are employed. The estimated prevalence of LVH and remodelling is still greater with multivariate discriminant function analysis, with which it is found in about 70% of hypertensives. Overall, the data suggest that prevalence of LVH in established hypertension is high.[16]

Left ventricular hypertrophy is an independent predictor of cardiovascular morbidity and mortality in adults. In children, the primary correlate of left ventricular mass (LVM) is lean body mass, but fat mass, gender and systolic blood pressure are also contributors. In children with sustained hypertension, 8-41 % have LVMI above the 95th percentile and in 10-15.5 % of these, LVMI is elevated above levels associated with increased mortality in adults. The presence of obesity is associated with higher LVMI than is found in children with hypertension alone.[17]

A total of 372 untreated hypertensive patients were studied by Ratti et ell. Four different patterns of LV hypertrophy (eccentric nondilated, eccentric dilated, concentric nondilated and concentric dilated hypertrophy) were identified by echocardiography. A modified National Cholesterol Education Program definition for Metabolic Syndrome was used, with body mass index replacing waist circumference. The overall prevalence of Metabolic Syndrome and LV hypertrophy (LVH) was 29% and 61%, respectively. Patients with MS showed a higher prevalence of LVH (P=0.0281) and dilated LV geometries, namely eccentric dilated and concentric dilated hypertrophy.[18]

In our study, we also found that 83 individuals suffered from left ventricular hypertrophy and among the participants, the most affected were females. In our study, we also found that the most important risk factor contributing to the LVH was hypertension, followed by obesity and age. It was further seen that, the higher the systolic blood pressure, more severe will be the LVH.

Limitation of Study:

Our study has a few limitations. First, the findings may not be generalizable to other settings, as the study was conducted in a single tertiary care hospital in Mirpur, Azad Jammu and Kashmir. Second, due to the cross-sectional design of the study, a causal relationship between the identified factors and the development of LVH cannot be established. Despite these limitations, the results of this study could still be valuable for formulating strategies to detect LVH in hypertensive patients.

Funding: Nil
Conflict: Nil

Conclusion:

Left ventricular hypertrophy is a significant cause of mortality and morbidity and it has strong correlation with uncontrolled systolic hypertension. Higher the blood pressure the more severe will be the LVH and LVM. Further factors that also contribute to LVH includes gender, obesity and age, they also influence in the development of LVH.

References:

Levey AS, Coresh J, Balk E. et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Ann Intern Med. 2003;139((2)):137–47. doi: 10.7326/0003-4819-139-2-200307150-00013.

Ezzati M, Lopez AD, Rodgers A. et al. Selected major risk factors and global and regional burden of disease. Lancet. 2002;360((9343)):1347–60. doi: 10.1016/S0140-6736(02)11403-6.

Gupta R. Trends in hypertension epidemiology in India. Hum Hypertens 2004;18:73-8. Available from: http://www.nature.com/doifinder/10.1038/sj.jhh.1001633.

Dahlof B, Devereux BR, Kjeldsen SE. Cardiovascular morbidity and mortality in the losartan intervention for end point reduction in hypertension study (LIFE): A randomised trial against atenolol. ACC Curr J Rev 2002;11:26. Available from: http://www.linkinghub.elsevier.com/retrieve/pii/S1062145802007778

Lozano JV, Redon J, Cea-Calvo L, Fernandez-Perez C, Navarro J, Bonet A, et al. Left ventricular hypertrophy in the Spanish hypertensive population. The ERIC-HTA study. Rev Esp Cardiol 2006;59:136-42

Ching SM, Chia YC, Wan Azman WA. Prevalence and determinants of left ventricular hypertrophy in hypertensive patients at a primary. Care Clin 2012;7:2-9.

Fragola PV, De Nardo D, Calo L. et al. Use of the signal-averaged QRS duration for diagnosing left ventricular hypertrophy in hypertensive patients. Int J Cardiol. 1994;44((3)):261–70. doi: 10.1016/0167-5273(94)90290-9.

Daniel P, Juni P, Egger M, Bachmann LM.. et al. Accuracy of electrocardiography in diagnosis of left ventricular hypertrophy in arterial hypertension: systematic review. British Medical Journal. 2007;335((7622)) doi: 10.1136/bmj.39276.636354.AE

Fragola PV, De Nardo D, Calo L. et al. Use of the signal-averaged QRS duration for diagnosing left ventricular hypertrophy in hypertensive patients. Int J Cardiol. 1994;44((3)):261–70. doi: 10.1016/0167-5273(94)90290-9

Wong CY, O'Moore-Sullivan T, Leano R, Byrne N, Beller E Marwick TH, et al. Alterations of left ventricular myocardial characteristics associated with obesity. Circulation 2004;110:3081-7

Kathrotia RG, Paralikar SJ, Rao PV, Oommen ER. Impact of different grades of body mass index on left ventricular structure and function. Indian J Physiol Pharmacol 2010;54:149-56.

Cuspidi C, Sala C, Negri F, Mancia G, Morganti A. Prevalence of left-ventricular hypertrophy in hypertension: An updated review of echocardiographic studies. Hum Hypertens 2012;26:343-49. Available from: http://www.nature.com/doifinder/10.1038/jhh.2011.104.

Wang SX, Xue H, Zou YB, Sun K, Fu CY, Wang H, Hui RT. Prevalence and risk factors for left ventricular hypertrophy and left ventricular geometric abnormality in the patients with hypertension among Han Chinese. Chin Med J (Engl). 2012 Jan;125(1):21-6.

Jaleta GN, Gudina EK, Getinet W. Left ventricular hypertrophy among black hypertensive patients: focusing on the efficacy of angiotensin converting enzyme inhibitors. BMC Res Notes. 2014 Jan 20;7:45. doi: 10.1186/1756-0500-7-45.

Bang CN, Gerdts E, Aurigemma GP, Boman K, Dahlöf B, Roman MJ, Køber L, Wachtell K, Devereux RB. Systolic left ventricular function according to left ventricular concentricity and dilatation in hypertensive patients: the Losartan Intervention For Endpoint reduction in hypertension study. J Hypertens. 2013 Oct;31(10):2060-8.

Korner PI, Jennings GL. Assessment of prevalence of left ventricular hypertrophy in hypertension. J Hypertens. 2009 Jun;16(6):715-23.

Kavey RE. Left ventricular hypertrophy in hypertensive children and adolescents: predictors and prevalence. Curr Hypertens Rep. 2013 Oct;15(5):453-7.

Ratto E, Viazzi F, Verzola D, Bonino B, Gonnella A, Parodi EL, Bezante GP, Leoncini G, Pontremoli R. Metabolic syndrome is associated with left ventricular dilatation in primary hypertension. J Hum Hypertens. 2016 Mar;30(3):158-63.