

# Prevalence Of Left Ventricular Hypertrophy On Transthoracic Echocardiogram, Irrespective Of The Cause; Its Age And Gender Distribution; An Observational Study

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## Citation

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## Abstract

**Introduction:** Left ventricular hypertrophy is defined as an increase in the mass of the left ventricle either secondary to an increase in wall thickness or an increase in cavity size or both. The abnormal increase in mass is caused by a chronically increased workload on the heart most commonly from hypertension, although there is also a genetic component. LVH has been associated with increased risk of cardiac events such as myocardial infarction, heart failure, and sudden cardiac death by a factor of three to five-folds.

**Objective:** To determine the prevalence of Left Ventricular Hypertrophy irrespective of the cause and its association with age and gender, via Transthoracic Echocardiographic approach.

**Study Design:** Descriptive Observational study consisted of 418 patients of all ages and both genders went through the Transthoracic Echo, 2-D/M-mode Echo findings for LVH were noted and data analyzed in SPSS.

**Results:** Out of 418, 49 % were males and 61 % were females. Mean age 56.1 years. 38.75 % of total patients were found with Left Ventricular Hypertrophy, of which 16.74 % were males and 22 % were females. 59.2 % of LVH patients were belongs to age group of 51 – 70 years.

**Conclusions:** From this study it is acknowledged that in our community, females are more frequently referred for the Echocardiography than males while males are more prone to LVH than females, irrespective of the cause. Also it is noticed that Left Ventricular Hypertrophy is mostly found after the fifth decade of life in both genders, either because of delayed diagnosis as most of patients avoid to come to a Physician or it is the principle time to develop hypertrophy in myocardial cells after a prolonged and continuous LV stress. Ideally, all hypertensive patients should be screened with echocardiography to look for LVH. Once diagnosed, patients with LVH should undergo proper management according to the cause as remarkable recovery has been confirmed form multiple studies. Further studies required to evaluate and differentiate the causes of LVH in our community.

## INTRODUCTION

It has been well recognized that the presence of left ventricular hypertrophy (LVH) is an adverse feature in hypertension, with such patients having a substantially higher risk of cardiovascular events, including mortality and morbidity from heart failure, atrial fibrillation, and sudden death.<sup>1</sup> Indeed, LVH is probably the most visible manifestation of hypertensive target organ damage.

When the heart faces a hemodynamic burden, it can do the following to compensate: (1) use the Frank-Starling mechanism to increase cross bridge formation; (2) augment

muscle mass to bear the extra load; and (3) recruit neuro hormonal mechanisms to increase contractility. The first mechanism is limited in its scope, and the third is deleterious as a chronic adjustment. Thus, increasing mass assumes a key role in the compensation for hemodynamic overload. This increase in mass is due to the hypertrophy of existing myocytes rather than hyperplasia, because cardiomyocytes become terminally differentiated soon after birth. In response to pressure overload in conditions such as aortic stenosis or hypertension, the parallel addition of sarcomeres causes an increase in myocyte width, which in turn increases wall thickness. This remodeling results in concentric

hypertrophy (increase in ratio of wall thickness/chamber dimension).<sup>2</sup>

While volume overload in conditions such as chronic aortic regurgitation, mitral regurgitation, or anemia engenders myocyte lengthening by sarcomere replication in series and an increase in ventricular volume. This pattern of eccentric hypertrophy (cavity dilatation with a decrease in ratio of wall thickness/chamber dimension) is also initially compensatory, such that the heart can meet the demand to sustain a high stroke volume. However, chronic hypertrophy may be deleterious because it increases the risk for the development of heart failure and premature death<sup>2</sup>. Both forms of hypertrophy are usually accompanied by complex changes in gene reprogramming.<sup>3</sup>

Treatment of LVH and the resulting regression in the hypertrophy has shown to have positive effects on the health and prognosis of the disease<sup>4</sup>. A Meta Analysis study, conducted in Italy, showed that reduction of left ventricular mass reduces the risk of cardiovascular events by more than one half. Another important observation was that compared to subjects with regression of LVH, those with persistently normal LV mass showed a similar risk of subsequent events<sup>5</sup>. Another study conducted in Italy showed with regression of LVH, the risk of stroke decrease as well<sup>6</sup>

Our strategy is to estimate the burden of LVH in our community so as to propose the screening plans to the concerned authority and so trying to reduce the morbidity and mortality secondary to Left ventricular hypertrophy.

Objectives:

“To find out the Prevalence of the Left Ventricular Hypertrophy irrespective of the cause; its age and gender associations; using Transthoracic Echocardiographic criteria.”

## **PATIENTS AND METHODS**

This is a descriptive analytical study, consisted of 418 numbers of patients. Case recruitment done in the Echo department of Memon Medical Institute Hospital Karachi which is a high volume Tertiary care public hospital having all faculties including Cardiology, placed in the centre of the city, Karachi, Pakistan. Study period: consisted of 6 months (from Jan, 2011 to Aug, 2011). Inclusion criteria: Patients of both genders, including all age groups, diagnosed with Left Ventricular Hypertrophy on Transthoracic Echocardiography. Exclusion criteria: Patients with

congenital heart disease, Pheochromocytoma, Friedreich ataxia and Amyloidosis were excluded from the study. Questionnaires were filled out during an interview with patients and included these variables; age, gender and findings of Left ventricular Hypertrophy on 2-D Transthoracic Echocardiogram. In our laboratory, an LV dimension usually was measured from the systolic and diastolic frames of 2D parasternal long-axis, as recommended by the American Society of Echocardiography. Inter Ventricular Septal thickness greater than 12.0 mm in end diastole, taken in parasternal long axis view, was taken as LVH.

Data analysis was performed through SPSS version 10. No statistical test was applicable for this descriptive study.

Operational definitions

Transthoracic Echocardiography (TTE)

Transthoracic echocardiography (TTE) is the primary noninvasive ultrasonographic imaging modality for quantitative and qualitative evaluation of cardiac anatomy and function.

2D Echocardiography

Two-dimensional (2D) echocardiography is still the primary tool for chamber quantification and evaluation of left ventricular systolic function. Real-time 2D echocardiography provides high-resolution images of cardiac structures and their movements and allows quantitative measurements of cardiac dimensions, area, and volume and visualization of the endocardial border and thickening of the ventricular walls.

## **RESULTS**

The study consisted of 418 (N) numbers of patients, out of which 49 % were males and 61 % were females and Mean age was found to be 56.1 years (Table-1). Among total number of cases, 38.75 % of patients were found with Left Ventricular Hypertrophy, of which 16.74 % were males and 22 % were females (Graph-1). While among Males, about 42.94% diagnosed with LVH and among Females, about 36.07% were diagnosed with LVH.

We have divided the Age into five equal groups (Table-2). Majority of LVH patients (59.2%) are found in the 3rd age group of 51-70 years (Table-3), in case of both genders.

# Prevalence Of Left Ventricular Hypertrophy On Transthoracic Echocardiogram, Irrespective Of The Cause; Its Age And Gender Distribution; An Observational Study

**Table 1**

DESCRIPTIVE STATISTICS

	Numbers of Patients (N)	Percentage (%)	AGE ( in years)			
			Minimum (Years)	Maximum (years)	Mean (Years)	Std. Deviation
TOTAL	418	100%	11	96	56.10	16.80
MALE	163	49%	12	94	59.59	16.92
FEMALE	255	61%	11	96	53.87	16.36

**Table 2**

DISTRIBUTIONS OF AGE GROUPS

AGE GROUPS ( YEARS )	NUMBER OF PATIENTS ( N )
11 - 30	40
31 - 50	84
51 - 70	202
71 - 90	88
>90	4
TOTAL	418

**Table 3**

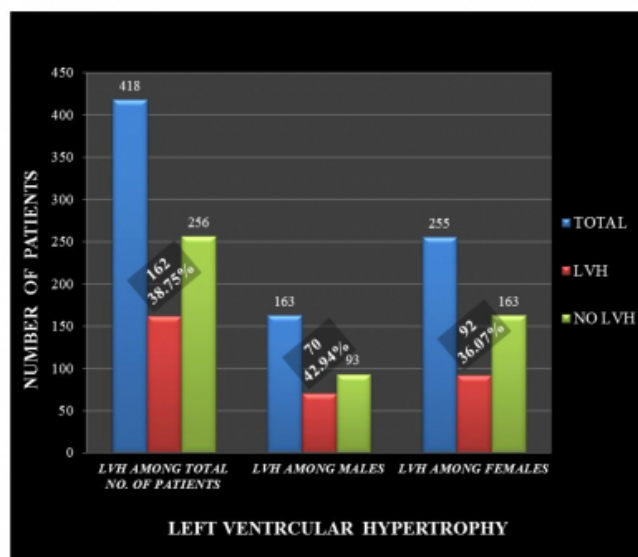
AGE DISTRIBUTION OF LEFT VENTRICULAR HYPERTROPHY

AGE GROUPS ( YEARS )	NO. OF LVH PATIENTS ( MALE )	NO. OF LVH PATIENTS ( FEMALE )	TOTAL NO OF LVH PATIENTS	% OF LVH ACCORDING TO AGE GROUP
11 - 30	0	4	4	5.7 %
31 - 50	8	14	22	13.5 %
51 - 70	42	54	96	59.2 %
71 - 90	19	17	36	22.2 %
>90	0	3	3	1.8 %
TOTAL	70	92	162	
% of LVH	16.74 %	22.00 %	38.75	

\*LVH = Left Ventricular Hypertrophy

**Graph 1**

GENDER DISTRIBUTION OF LEFT VENTRICULAR HYPERTROPHY (LVH)



## DISCUSSION

Due to the various risks associated with LVH, its early detection and treatment is vital for a better prognosis. Detection of LVH is done mainly by ECG, Echocardiography and MRI. MRI is considered to be the gold standard, but due to it being expensive and less available, Echocardiography is the test of choice.<sup>2</sup> Compared to ECG, Echocardiography is much more sensitive (though the specificity is not much different)<sup>7</sup>. Hence, Echocardiography must be used over ECG for a more accurate diagnosis.

Left Ventricular Hypertrophy may be associated with an absence of symptoms for many years before the development of congestive heart failure, or unexpected sudden death. Patients with LVH due to continuous pressure overload (systemic hypertension, aortic stenosis) or volume overload (mitral regurgitation) may remain in a compensatory phase with no symptoms and normal or near-normal exercise reserve for years.<sup>2</sup> Others have a transition to heart failure that may be due to diastolic dysfunction, systolic dysfunction, or both.

In chronic pressure overload and extreme volume overload, subendocardial ischemia due to reduced coronary flow reserve probably plays a role in limiting exercise reserve and promoting myocardial fibrosis.<sup>8</sup> Afterload excess due to inadequate hypertrophy to normalize wall stress itself reduces systolic ejection performance, independent of

intrinsic changes in contractility, and it accounts for the extremely rapid improvement in ejection fraction after valve replacement in some patients with aortic stenosis and aortic regurgitation.

In older patients with isolated systolic hypertension, concentric LVH is common.<sup>9</sup> Diastolic dysfunction, including the presence of a Doppler filling pattern of impaired relaxation, has been observed in >80% of older hypertensives.<sup>10</sup> On comparing younger (<60 years) and elderly (>65 years) patients with comparable severities of aortic stenosis showed that elderly patients with pressure overload were characterized by more severe hypertrophy and interstitial fibrosis, as well as more severe impairment of relaxation, myocardial stiffness, and filling indices. Ejection fraction and midwall shortening were similar in the 2 groups (younger and elderly patients).<sup>11</sup>

Gender also influences function in pressure-overload hypertrophy in humans. <sup>12, 13</sup>In men and women with aortic stenosis and similar aortic valve areas and gradients, men are more likely to have cavity enlargement, a lower ejection fraction, and increased diastolic myocardial stiffness associated with more severe changes in collagen architecture.<sup>14</sup> Sex-based differences in diastolic function are recapitulated in rodent aortic stenosis models in which female animals demonstrate more favorable changes in cardiac geometry, <sup>15, 16</sup> as well as better preservation of normal adult cardiac gene expression.<sup>17</sup>

In severe cases of chronic LVH associated with diastolic dysfunction and preserved ejection fraction, both male and female patients may experience episodic severe congestive heart failure and hospitalization. Although heart failure patients with normal LV ejection fractions had a lower mortality risk than those with reduced ejection fractions, heart failure patients with normal ejection fractions had an annual mortality of 18.9% versus 4.1% for matched control subjects during the 6-year study period, indicating a >4-fold mortality risk.<sup>18</sup> Atrial fibrillation also modifies diastolic dysfunction in patients with LVH. The increased reliance on atrial contraction to fill the stiff left ventricle means that atrial fibrillation is usually very poorly tolerated. In addition, pressure overload from hypertension is responsible for more atrial fibrillation in the population ( $\approx$ 14% of cases) than any other risk factor, and atrial fibrillation confers a 4- to 5-fold increase in the risk of stroke.<sup>19</sup>

The population-based evidence suggests that therapies to

limit and reverse LVH in patients are desirable, even in the absence of symptoms of heart failure. We already know that a regression of severe LVH can be achieved in some patients. Major insights regarding the regression of hypertrophy in patients with hemodynamic overload can be drawn from the extraordinary collection of studies from one team in Zurich. <sup>20, 21</sup> This team performed serial LV hemodynamic and biopsy analyses before and after valve replacement in patients with valvular aortic stenosis and aortic insufficiency. These patients were characterized by massive LVH, severe collagen deposition, diastolic dysfunction and, in some instances, depression of systolic ejection indices. In brief, these observations demonstrated that near-normalization of systolic load causes a rapid reduction in myocyte hypertrophy and LV mass ( $\approx$ 35% reduction) within a few weeks after valve replacement.<sup>22</sup>

In this early phase of the rapid regression of myocyte hypertrophy but little change in collagen and matrix, myocardial relaxation improves; however, the fraction of collagen in the myocardium actually increases. This increase is accompanied by a worsening of diastolic indices of myocardial stiffness. Astonishingly, during continued reduction of load many months to a few years after valve replacement, regression of interstitial fibrosis and further regression of LVH occurs, resulting in near-normalization of both muscle mass and fibrous tissue content.<sup>22</sup> This initial rapid regression of hypertrophy and later regression of fibrosis is accompanied by a reversal of diastolic dysfunction, an improvement in systolic dysfunction (when present), and an improvement in exercise reserve.

ACE inhibitors reduced LV mass by  $\approx$ 15%. Lesser reduction was achieved with diuretics (11%),  $\beta$ -blockers (8%), and calcium-channel blockers (8.5%). Overall, LV mass was reduced by only 11.9%, which is far less than the magnitude of early regression and the late near-normalization of mass observed after valve replacement. <sup>23</sup> The relatively disappointing magnitude of regression observed in pharmacological trials in hypertensive patients is likely related to an incomplete reduction of hypertension itself rather than to inadequate targeting of downstream signal cascades. In addition to a more effective implementation of available antihypertensive agents to achieve current consensus treatment guidelines, new antihypertensive agents with potent effects on systolic hypertension raise the potential for more complete long-term regression of hypertrophy in hypertensive patients, similar to that which

can be achieved with valve replacement in aortic stenosis and regurgitation.<sup>24</sup> Treatment-induced regression of LVH decreases adverse cardiovascular events and improves overall survival. When modifying medications in hypertensive patients, it is important to remember that the treatment of LVH is not synonymous with blood pressure control.

## CONCLUSION

It is also acknowledged from our study that in our community, females are more frequently referred for the Echocardiography than males while males are more prone to LVH than females irrespective of the cause. Also it is noticed that Left Ventricular Hypertrophy is mostly found after the fifth decade of life either because of delayed diagnosis as most of patients avoid to come to a Physician or it is the principle time to develop hypertrophy in myocardial cells after a prolonged and continuous LV stress as discussed above.

Pharmacological treatment induced regression of LVH improves cardiovascular outcomes independent of blood pressure. Furthermore, surgical intervention for the treatment of valvular heart disease is the treatment of choice in case of valvular cause of LVH with proven after benefits.

LVH is a common and potentially modifiable cardiovascular risk factor often overlooked in clinical practice. Ideally, all hypertensive patients should be screened with echocardiography to look for LVH. It is to bring the attention of the concerned authority through our article that they should take a prompt action regarding the screening program for Echocardiographic detection of LVH especially in age group of 50-70 years, so that it could be a valuable step in reducing the morbidity and mortality risk associated with LVH patients.

Further study is necessary to examine the causes of LVH in our community.

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