LEFT VENTRICULAR MASS INDEX: A PREDICTOR OF MORBIDITY AND MORTALITY IN ESSENTIAL HYPERTENSION

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ABSTRACT: BACKGROUND: Hypertension is one of the leading causes of the global burden of disease and is the most important health problem met by general physicians. The development of left ventricular hypertrophy increases with the severity and duration of hypertension and presence of increased left ventricular mass is associated with increased incidence of other target organ damage. Early detection and appropriate treatment decreases the development of increased left ventricular mass index and reduces the mortality and morbidity. **AIM OF STUDY:** To establish the correlation of increased left ventricular mass index with target organ involvement in hypertensive patients. **METHOD:** 50 patients of both sexes with essential hypertension attending the outpatient clinic as well as those admitted in medical wards at Sri Adichunchanagiri Institute of Medical Science and Research Centre, B.G. Nagara from November 2010 to April 2012 were involved in the study. 2D echocardiography was performed for all subjects and left ventricular mass index was calculated for each. **STATISTICAL TESTS:** 1. Kappa measurement of agreement 2. Chi-square test. **RESULTS:** Out of 50 patients, 22 had increased left ventricular mass index, by echocardiogram. Target organ damage in the form of Retinopathy was found in 90.9% ($p < 0.01$), nephropathy in 13.6%, TIA/stroke in 22.7% and cardiac failure in 22.7% of patients with increased left ventricular mass index. Whereas among patients with normal left ventricular mass index, 46.4% had retinopathy, 7.1% cardiac failure, 14.3% stroke/TIA and 3.6% nephropathy. **CONCLUSION:** Target organ damage in the form of retinopathy, cardiac failure, TIA/stroke, nephropathy were found more in patients with increased left ventricular mass index than in patients with normal left ventricular mass index. Hence increased left ventricular mass index in essential hypertension is an important predictor of target organ damage which results in significant morbidity and mortality.

KEY WORDS: Left ventricular mass index, Left ventricular hypertrophy

INTRODUCTION: Affecting 1 billion people worldwide, systemic hypertension remains the leading cause of death worldwide and one of the world's great public health problems¹. It is usually asymptomatic, readily detectable, usually easily treatable, and often leads to lethal complications if left untreated.

Left ventricular hypertrophy (LVH) is one of the most important complications of hypertension. The prevalence of left ventricular hypertrophy increases with the severity of hypertension and presence of increased left ventricular (LV) mass is associated with increased incidence of myocardial infarction, congestive cardiac failure, stroke and other target organ damage.

An increase in left ventricular mass even if not of a substantial degree is associated with cardiovascular risk. In the Framingham heart Study, in 1990, it was shown that for each 50 g/m increase in LV mass (corrected for height) there was a relative risk for mortality of 1.73, even in subjects free of clinically apparent cardiovascular disease². It was also demonstrated that this risk

was statistically independent of blood pressure, age, antihypertensive treatment, and other cardiovascular risk factors.

Left ventricular hypertrophy is accurately recognized with M-mode echocardiography through the estimation of left ventricular mass. By echocardiography, left ventricular mass is shown to progressively increase with increases in blood pressure. Left ventricular mass is greater in those whose pressure does not fall during sleep because of a more persistent pressure load³. An increase in left ventricular mass predicts higher incidence of clinical events including death attributable to cardiovascular disease².

Accurate measurements of left ventricular mass by echocardiography have been used to show that LVH is more prevalent, more heterogeneous in its anatomic and pathophysiologic patterns and more important as a determinant of prognosis in hypertensive patients than previously thought. The importance of early detection of increased left ventricular mass by echocardiography is therefore obvious. The left ventricular mass reduction during antihypertensive treatment is associated with reduced rate of complications of essential hypertension. The development or regression of LVH during antihypertensive treatment may be more closely linked to prognosis than are changes in clinic blood pressure⁴.

Devereux and Reichek in 1981 were the first to co-relate echocardiographic LV mass estimates with LV specimens of same hearts at biopsy⁵. They tested various geometric formulas and different methods of measuring wall thickness and left ventricular internal diameter and found that anatomic LV mass correlated best with LV mass measurements by Penn convention ($r=0.96$) using the following empirical equation:

$$\text{LV mass} = 1.04[(\text{IVST} + \text{LVID} + \text{PWT})^3 - \text{LVID}^3] - 13.6\text{g}.$$

Using the American Society of Echocardiography method of measuring septal and left ventricular wall thickness, Devereux and Woythaler et al., found a good correlation between echocardiographic and anatomic LV mass ($r=0.90$ and 0.81 respectively)⁶.

An increase in left ventricular (LV) mass primarily from increase in LV wall thickness, sufficient to be defined as LVH, is associated with clinically important abnormalities of diastolic, electrophysiologic and systolic function⁷⁻⁹. Hence this study has been carried out to highlight the importance of recognizing increased left ventricular mass in patients with essential hypertension because of its association with adverse events. Early institution of therapy to control blood pressure and reduce left ventricular mass can prevent the development and progression of complications including target organ damage in hypertensives.

METHODS: The study was carried out at Sri Adichunchanagiri Institute of Medical Science and Research Centre from November 2010 to April 2012.

The study group consisted of 50 patients of both sexes with essential hypertension attending the outpatient clinic as well as those admitted in medical wards.

The study group consisted of patients above the age of 40 years. All freshly detected and old cases of essential hypertension, irrespective of duration of hypertension and type of treatment receiving were taken into the study. The exclusion criteria were all cases of secondary hypertension, patients with previous ischemic heart disease myocardial infarction or ischemic cardiomyopathy, congenital heart disease and patients with valvular heart disease, patients with diabetes mellitus.

History, physical examination, chest X-ray, standard 12 lead ECG and two dimensional echocardiography were done for all patients.

The following clinical information was obtained, apart from investigations.

1. Body surface area for calculating left ventricular mass index (LVMI)
2. Cardiovascular examination:
 - ☐ Site and character of apical impulse
 - ☐ Character of heart sounds
 - ☐ Presence of abnormal heart sounds and murmurs.
3. History of stroke or transient ischaemic attack (TIA).
4. Ophthalmic examination for any evidence of hypertensive retinopathy.
5. Investigations:
 - Fasting Blood Sugar, blood urea, serum creatinine, fasting lipid profile, urine for albuminuria.
6. Chest X-ray: PA view- to measure cardiothoracic ratio.

Electrocardiogram: Standard 12 lead ECG was obtained in all patients. Presence of left ventricular hypertrophy was assessed using Sokolov-Lyon index and Romhilt –Estes point score system.

Echocardiographic Studies: Combined M-mode and 2-dimensional echocardiographic studies were performed in all study subjects. The subjects were positioned in a 30° left decubitus position with slight elevation of the head. Comprehensive 2-D tomographic planes were employed with multiple parasternal views of left ventricle in long and short axis, apical four chamber and long axis view and subcostal four chamber and short axis views. After positioning of the cursor through interventricular septum and posterior wall, at the level of chordae tendinae, simultaneous M-mode and two dimensional recording were obtained from the parasternal transducer position in both long and short axis views of the left ventricle.

Measurement: The left ventricular posterior wall and interventricular septum were measured at the time of atrial depolarization before the onset of notch. The left ventricle internal dimension was measured at the level of chordae tendinae, as the distance between the left side of interventricular septum and the posterior left ventricle. M-mode measurements were taken by the leading edge to leading edge technique as recommended by the American Society of echocardiography. All measurements were averaged to the closest 1 mm from three good quality cardiac cycle.

The left ventricular mass index (LVMI) was calculated by using: Penn's convention formula:

Left ventricular mass (LVM) = $1.04 [(LVIDd + PWT + IVST)^3 - LVIDd^3] - 14 \text{ gm}$.

As this closely correlated with necropsy LV mass and sensitivity and specificity were 93% and 95% respectively, this was chosen over other methods to calculate LV mass¹⁰.

Left ventricular mass index (LVMI) = Left ventricular mass/Body surface area

[LVIDd = left ventricle internal dimension in end diastole; PWT = posterior wall thickness; IVST = interventricular septal thickness]

The normal left ventricular mass index for the Indian population¹¹ is:

1. Males = 121g/m²
2. Females = 110g/m²

Any value more than this was considered as left ventricular hypertrophy.

ORIGINAL ARTICLE

Patients were divided into two groups based on their left ventricular mass index. Group I with normal left ventricular mass index and Group II with increased left ventricular mass index.

STATISTICAL ANALYSIS: Chi-square test/Fisher exact test were used to analyse the association between increased left ventricular mass index and various parameters such as age, sex, duration of hypertension and target organ damage.

RESULTS: In this study 50 patients were included and were divided into two groups. Group – I with normal Left ventricular mass index (LVMI) and group II with increased left ventricular mass index (LVMI).

Out of 50 patients 22 patients had increased LVMI and 28 patients had normal LVMI.

SEX	Patients-Normal LVMI(Group I)	Patients – Increased LVMI(Group II)
MALES	19(67.9%)	8(36.4%)
FEMALES	9(32.1%)	14(63.6%)
TOTAL	28	22

Table 1: Correlation of sex with increased LVMI

$p < 0.05 = S$

More number of females i.e. 63.6% (14) were found to have increased LVMI as compared to males 36.4 % (8). $p < 0.05$, so statistically significant.

ORIGINAL ARTICLE

Duration of HTN in years	Patients N-LVMI (group I)	Patients Increased LVMI (group II)
1-5 Years	13(46.4%)	4(18.2%)
6-10 Years	11(39.3%)	9(40.9%)
>10 Years	4(14.3%)	9(40.9%)
Mean $\pm$ SD	7.32 $\pm$ 5.39	10.50 $\pm$ 5.23

Table 2: Correlation of duration of Hypertension with increased LVMI

p<0.05

More number of patients in Group I i.e. 46.4%(13) were found to have a duration of HTN between 1-5. More number of patients in Group II were found to have a duration of HTN of ≥ 6 years i.e. 40.95%(9) between 6-10 years and 40.9%(9) >10 years. p < 0.05, so statistically significant.

Target organ damage	Patients N- LVMI (group I)	Patients Increased LVMI (group II)	P value
Retinopathy	13(46.4%)	20(90.9%)	0.001
Cardiac failure	2(7.1%)	5(22.7%)	0.217
Stroke/TIA	4(14.3%)	5(22.7%)	0.481
Nephropathy	1(3.6%)	3(13.6%)	0.308

Table 3: Target organ damage associated with increased LVMI

The target organ damage including retinopathy, cardiac failure, nephropathy and TIA/Stroke were more in patients with increased LVMI when compared to patients with normal LVMI, as shown above. Statistically strong significance (p<0.01) was obtained for retinopathy.

DISCUSSION: Systemic arterial hypertension impacts constant hemodynamic burden on the heart. Left ventricular hypertrophy with consequent increase in left ventricular mass is the end result of the same. It is an adaptation method of the myocardium to systemic arterial hypertension. A number of studies have identified LVH, most accurately represented by increased left ventricular mass index as a major and independent risk factor for development of sudden death, acute myocardial infarction, congestive cardiac failure and other cardiovascular morbidity and mortality¹².

A significantly higher percentage (63. 6%) of females than males (36. 4%) had increased left ventricular mass index in the present study ($p < 0. 05$). This finding is consistent with that reported by Gerdtts et al¹³, who found a higher prevalence of increased left ventricular mass index in females (80%) as compared to males (70%). The duration of hypertension in patients with increased LVMI was found to be more than in patients with normal LVMI ($p < 0. 05$). Ross et al¹⁴ reported duration of hypertension as a significant factor in the development of increased LVMI. Glasser SP et al¹⁵, also showed that duration of hypertension added significantly in predicting an elevated LVMI.

Other baseline characteristics like age, blood pressure and body surface area were similar in the two groups. Target organ damage was found to be higher among patients with increased LVMI as compared to those with normal LVMI, with $p < 0.01$ for retinopathy. Ding Y et al¹⁶ showed that there is extremely high prevalence of retinopathy in patients with increased left ventricular mass index ($p < 0. 05$). This study also showed retinopathy changes in 90.9% (20) of patients with increased LVMI ($p < 0. 01$).

Ding Y et al¹⁶ showed increased serum creatinine levels in patients with increased LVMI ($p < 0. 01$). This study showed more number of patients (13. 6%) in group II, with nephropathy than in group I (3. 6%). Y Shigematsu et al¹⁷ also found that hypertensives with increased LVMI had the most advanced fundusoscopic abnormalities and the greatest renal involvement, and hypertensive patient with normal LVMI had the least extracardiac target organ damage.

Kannel WB¹⁸ showed that the risk of congestive heart failure was proportional to the degree of increase in left ventricular mass. This study also showed that congestive heart failure was more common in patients with increased LVMI (22. 7%) than patients with normal LVMI (7. 1%). This study found a higher prevalence of stroke/TIA in patients with increased LVMI (22. 7%) than patients with normal LVMI (14. 3%). This finding is consistent with that of the Atherosclerosis Risk

in Communities (ARIC) Study by Ervin R. Fox et al¹⁹, in which the occurrence of ischaemic stroke was higher in patients with increased LVMI(62. 6%) than in those with normal LVMI(38. 6%). The ARIC study also showed that echocardiographic LVMI is an independent predictor of ischemic stroke.

CONCLUSION: This study shows that increased left ventricular mass index is associated with target organ damage in the form of retinopathy, nephropathy, cardiac failure and stroke/TIA in hypertensives. Although the prevalence of all forms of target organ damage was more in patients with increased LVMI, statistically significant association was found only for retinopathy. Nevertheless increased LVMI is a cause for morbidity and mortality in hypertensives. Hence routine estimation of LVMI in hypertensives and appropriate treatment for reversal of increased LVMI can prevent target organ damage in these patients.

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ORIGINAL ARTICLE

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