

# A Study of Left ventricular hypertrophy in hypertension

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**Abstract- Introduction:** Increased arterial pressure is one of the most important public health problem in developed as well as in developing countries. It is common, asymptomatic, readily detectable, easily treatable. Burden of hypertension increases with age among individuals aged above 60 years, the prevalence is 65.4%. Left ventricular hypertrophy has been shown to be a common and surprisingly early finding in hypertension even in patients without the evidence of coronary artery disease. Studies have also shown that aggressive control of hypertension not only reduces the hypertrophy but also reduces the long term cardiovascular morbidity and mortality. **Objectives:** this study was undertaken to find out the incidence of left ventricular hypertrophy in hypertension. **Materials and Methods:** After careful exclusion of patients with secondary hypertension, valvular heart disease, Diabetes mellitus, gross congestive heart failure and ischemic heart disease, 85 patients were considered for the study. Data was collected by history, clinical examination, ECG, X-ray and echocardiography. Coronary angiography was done in selected patients to rule out ischemic heart disease. **Statistical analysis:** Analysis was done using Chi square test. Descriptive data are presented as mean standard deviation and range values. Pearson's correlation coefficient was used to measure the relationship between the measurements. P value of 0.05 was considered for statistical significance. **Results: Of the 85 patients, 60 were males and 25 were females. 18 patients showed the ECG evidence of LVH, 40 patients had echocardiographic evidence of LVH. All had concentric hypertrophy. LV mass index in patients with LVH was  $226.22 \pm 51.18/m^2$  and is statistically significant ( $P < 0.001$ ). 54% showed increased LV mass index. LV relative wall thickness was  $0.61 \pm 0.09$  which is again statistically significant ( $P < 0.001$ ). **Conclusions:** In the present study the incidence of LVH was 47%, all had concentric hypertrophy. **LV mass index was determined by Pen's formula. The LV mass index in patients with hypertrophy is  $226.22 \pm 51.58 gms/m^2$  and  $118.43 \pm 22.72$  in patients without hypertrophy and this marked increase in LV mass is statistically significant ( $P < 0.001$ ). Echocardiography can detect LVH even before patients develop ECG evidence of LVH. Studies show that early and aggressive treatment of hypertension with drugs like angiotensin converting enzyme inhibitors and angiotensin receptor blockers not only reduces hypertension but also reduce left ventricular hypertrophy thereby reduces the morbidity and mortality.****

**Index Terms-** Hypertension, left ventricle, hypertrophy, LV mass index

## I. INTRODUCTION

Increased arterial pressure is one of the most important public health problem in developed as well as in developing countries. It is common, asymptomatic, readily detectable, easily treatable (1). In spite of increasing awareness in public and rapidly expanding array of antihypertensive drugs, hypertension remains one of the major causes of cardiovascular morbidity and mortality (2). Burden of hypertension increases with age among individuals aged above 60 years, the prevalence is 65.4% (19). Incidence of hypertension is 8-18% of adult population in developed countries and in India it is estimated to be between 1-5% in rural and 3-15% in urban population (3). Heart was traditionally regarded as a target organ adapting to the increased pressure load by hypertrophy and eventually failing if the load continues unremitting or was suddenly aggravated particularly if complicated by the advent of coronary disease (4).

Most patients with hypertension, the heart becomes secondarily involved in response to the progressively increasing LV afterload. The heart does this through two mechanisms, i.e. by increasing contractile function and second by undergoing structural adaptation. This structural adaptation is primarily achieved through Left ventricular hypertrophy (5).

Left ventricular hypertrophy has been shown to be a common and surprisingly early finding in hypertension even in patients without the evidence of coronary artery disease. It has been described not only in adolescents with borderline hypertension but also in the offspring of hypertensive patients (4).

Prevalence of hypertrophy increases with the duration of hypertension and is also influenced by gender, obesity and age factors (6,7). Echocardiography is the most sensitive technique that can identify left ventricular hypertrophy at early stage. LVH is both a target organ response to hypertension as well as a factor responsible for other cardiovascular events (8,9,10).

Studies have also shown that aggressive control of hypertension not only reduces the hypertrophy but also reduces the long term cardiovascular morbidity and mortality.

## II. OBJECTIVES

This study was undertaken to find out the incidence of left ventricular hypertrophy in hypertension.

## III. MATERIALS AND METHODS

After careful exclusion of patients with secondary hypertension, valvular heart disease, Diabetes mellitus, gross congestive heart failure and ischemic heart disease, 85 patients

were considered for the study. Data was collected by history, clinical examination, ECG, X-ray and echocardiography. Coronary angiography was done in selected patients to rule out ischemic heart disease.

Patients who fulfilled the inclusion criteria were subjected to ECG and X-ray and later to 2-D, M-mode and Doppler echocardiography. LV internal dimensions were obtained by M-mode echocardiography, End diastolic volume(EDV), end systolic volume(ESV) and ejection fraction were taken from parasternal short axis view by 2-D echocardiography. LV internal dimension can also be obtained by 2-D echo from apical as well as parasternal windows. LV mass index and relative LV wall thickness were calculated using the Penn's formula.

LV mass index=  $1.04[(LVIDD+IVS+LVPWd)3-LVIDd^3]-13.6$   
in gms/sq.m

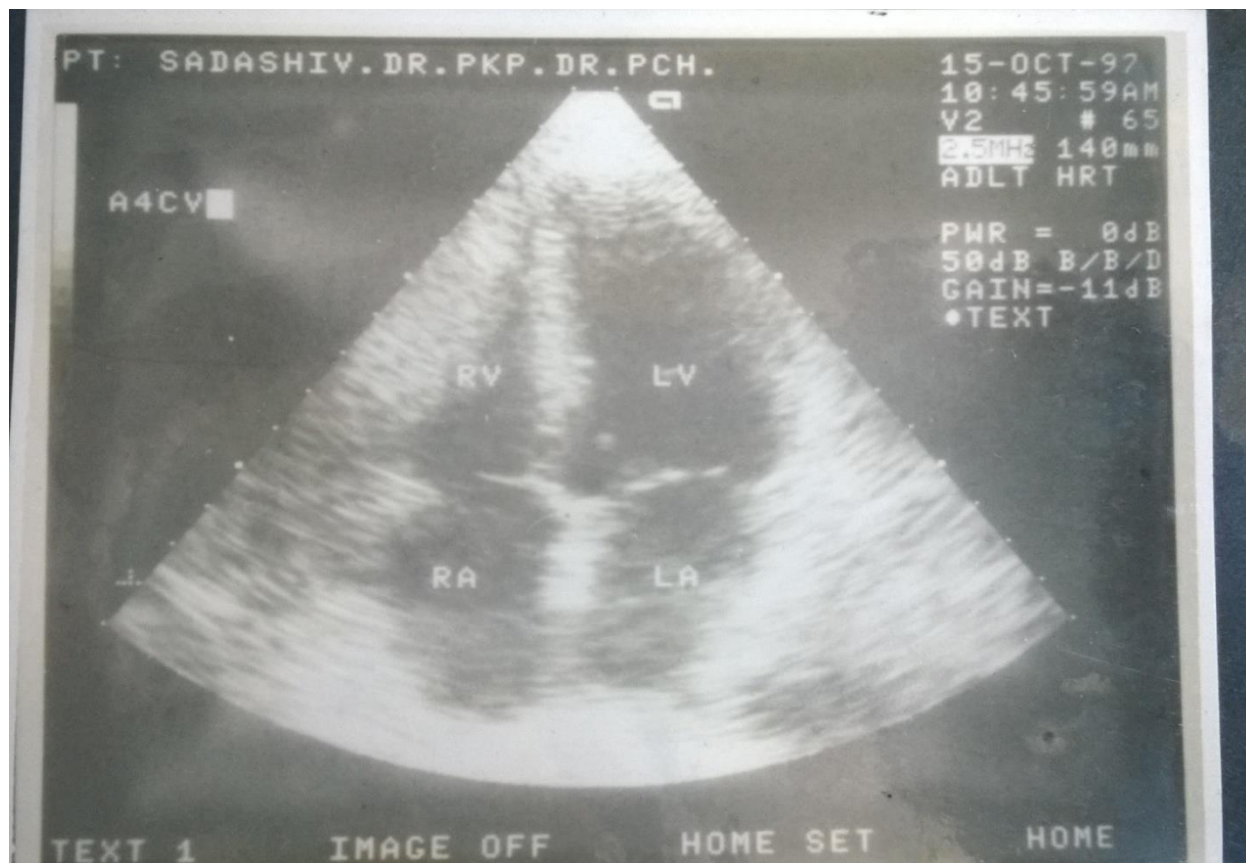
Relative wall thickness= $\frac{2 \times LV Pwd}{LVIDd}$

#### IV. STATISTICAL ANALYSIS

Analysis was done using Chi square test. descriptive data are presented as mean standard deviation and range values. Pearson's correlation coefficient was used to measure the relationship between the measurements. P value of 0.05 was considered for statistical significance.

#### V. OBSERVATIONS

Of the 85 patients, 60 were males and 25 were females. The age range was 25 to 75 years with a mean age of 54.42+/-10.96 years. 41 out of 85 patients had mild symptoms at time of entry into the study. 10 had exertional dyspnea, 5 had fatigue and 31 had chest pain. It means 52% were asymptomatic, only 4 patients had clinically detectable cardiomegaly. The average blood pressure of the subjects were 155.83+/-17.81mmhg systolic and 94.8+/-6.74mmhg diastolic, the range was 140/90 to 210/120mmhg. Duration of hypertension ranged from 1 month to 20 years. 18 patients showed the ECG evidence of LVH, 40 patients had echocardiographic evidence of LVH. All had concentric hypertrophy, 62 patients had diastolic dysfunction.



2-D Echo showing concentric LVH

**Table I**

Table showing distribution of LV hypertrophy in different age group.

Age Group	Male		Female		Total	
	Total	Cases	Total	Cases	Male+ Female	Cases
35-45	18	10	5	1	23	11
46-55	13	7	10	2	23	9
56-65	19	10	6	1	25	11
66-75	10	7	4	2	14	9
<b>Total</b>					<b>85</b>	<b>40</b>

average interventricular septal thickness was 1.18+/-0.14cms in patients with LVH, posterior wall thickness was 1.30+/-0.13cms, EDV was 4.23+/-0.48cms, LV mass index in patients with LVH was 226.22+/-51.18/m<sup>2</sup> and is statistically significant (P<0.001). 54% showed increased LV mass index. LV relative wall thickness was 0.61+/-0.09 which is again statistically significant (P<0.001). The range of 0.45-0.50 was considered normal.

**VI. CONCLUSIONS**

Since in the introduction of non-invasive methods such as radionuclide ventriculogram and doppler echocardiography, these techniques have become the modalities of choice for the assessment of left ventricular diastolic dysfunction. These have advantages of ease of performance and repeatability (14). Presence of ischemic heart disease, valvular lesions, hypertrophic cardiomyopathy and pericardial disease by 2-D and doppler echocardiography and other parameters such as ECG excluded patients from the study. 30 patients had chest

pain. 18 out of 30 of these patients had ECG evidence of ischemia and they were subjected to coronary angiogram and angiogram was normal, rest 12 were considered noncardiac chest pain. In the present study the incidence of LVH was 47%, all had concentric hypertrophy. **LV mass index was determined by Pen's formula. The LV mass index in patients with hypertrophy is 226.22+/-51.58gms/m<sup>2</sup> and 118.43+/-22.72 in patients without hypertrophy and this marked increase in LV mass is statistically significant (P<0.001)**

Echocardiography can detect LVH even before patients develop ECG evidence of LVH. According to the literature the incidence of LVH is 20-60% by echocardiography in referral centres (13). Savage D, Daniel et al (22) who assessed the prevalence of anatomic functional abnormalities of the heart in hypertension concluded that 61% of asymptomatic subjects had LVH in the form of increased interventricular and posterior wall thickness. LVH delays the active relaxation of the myocardium and also alters the chamber compliance. This results in altered flow velocity across mitral valve. Jian Fang Ren et al (20) had showed in their study that the incidence of LVH with abnormal

filling was 54%. Frank R Bauwens et al (21) conducted a study to know the influence of the arterial pressure and left ventricular hypertrophy. They conducted a study on untreated hypertensive patients with mild to moderate hypertension, a 24 hour ambulatory BP monitoring and determination of LV mass index was done. LV mass index did not correlate well with the office systolic or diastolic blood pressure, but there was a statistically significant correlation between 24 hour systolic blood pressure and LV mass. In a similar study, Devereux B. Richard et al (14) concluded that hypertensive LVH is poorly related to clinical/home measurement of blood pressure but a substantial relationship exists between LVH and blood pressure during recurring stress at work.

The present study also showed that 36 patients with LVH had hypertensive eye changes. Literature shows that by the time of echocardiographic evidence of LVH, most patients will have grade II hypertensive changes in eyes (23).

To conclude the incidence of LVH is 47% and all of them had concentric hypertrophy. LVH can manifest as angina due to impaired coronary oxygen reserve. With the increase in LV mass myocardial oxygen demand also increases.

Echocardiography is an easily available noninvasive technique and can be used for early detection of LVH and thus treat these patients with specific medications so as to arrest or reverse the pathological changes.

Recent studies show that early and aggressive treatment of hypertension with drugs like angiotensin converting enzyme inhibitors and angiotensin receptor blockers not only reduces hypertension but also reduce left ventricular hypertrophy thereby reducing the overall morbidity and mortality (19).

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