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# Prevalence of Left Ventricular Hypertrophy in Hypertensive Patients in A Community Survey in Bhagalpur, Bihar

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**ABSTRACT**

*Hypertension is one of the leading causes of the global burden of disease. Hypertension doubles the risk of cardiovascular diseases including coronary artery diseases and congestive heart failure. It leads to left ventricular hypertrophy (LVH) which add to the ischemic injury to heart. LVH affects the heart in many ways - impaired coronary vasodilator reserve, increased myocardial cell diameter without proportional proliferation of capillary vessels results in decreased capillary density and increased diffusion distance, decreased high energy phosphate content, impaired fatty acid oxidation, reduced myocardial glucose transport into the cell. In our study we look into the prevalence of LVH in patients with hypertension.*

**KEYWORDS : – LVH, Hypertension, LVMI, Myocyte**

**INTRODUCTION**: Left ventricular hypertrophy (LVH) may reflect physiological adaptation to an increased work load of the heart following intense physical training. However, LV hypertrophy often represents a pathophysiologic condition, and can develop due to intrinsic stimuli (cardiomyopathy), or secondary to extrinsic stimuli, such as pressure or volume overload accompanying hypertension and valvular disease. Myocardial hypertrophy is also part of the remodelling process following an acute myocardial infarction, and is a common finding in patients with congestive heart failure caused by systolic and/or diastolic LV dysfunction. Various studies in medical literature have reported a prevalence of LVH in Hypertensive patients in a range of 15-45%.

**AIMS AND OBJECTIVES**: Aim of the study is to determine the prevalence of LVH in patients with hypertension in Bhagalpur, Bihar.

**METHODS:** The study was undertaken in patients attending OPD and Indoor Department of Medicine, JLN Medical College, Bhagalpur. A total of 300 hypertensive patients were included in the study. After informed consent from each one of them, they were subjected to 2D Echocardiography for evaluation of their left ventricular hypertrophy. Left ventricular hypertrophy diagnosis was established using the American Society Of Echocardiography criteria of left ventricular mass index.

LVMI>110 g/m^2 for Female

LVMI>135 g/m^2 for Male

**EXCLUSION CRITERIA:** Patients having valvular heart diseases, Diabetes mellitus and Cardiomyopathy were excluded from the study.

**RESULTS:**

***TABLE 1. PATIENT CHARACTERISTICS***

| PATIENT CHARACTERISTICS OF THE STUDY POPULATION | |
|---|---|
| AGE | RANGE:45-75 Years, MEAN: 63 Years |
| SEX | MALE:224/300, FEMALE:76/300 |

**TABLE 2. FINDINGS IN HYPERTENSIVE PATIENTS**

| RESULTS: HYPERTENSIVE PATIENTS | | | |
|---|---|---|---|
| | TOTAL POPULATION | MALE | FEMALE |
| HYPERTENSION | 300 | 224 | 76 |
| LVH IN HYPERTENSION | 68 | 49 | 19 |
| PREVALENCE OF LVH IN HYPERTENSIVE PATIENTS: 68/300 (**22.67%**) | | | |

From the above results we see that prevalence of LVH in patients with hypertension in 22.67%. While if we divide it in to sex wise distribution we see that about 25% of the female hypertensive patients are having LVH which is higher than the average.

**DISCUSSION:**

The myocardium has three distinct morphological units-the muscular part, interstitial part & avascular part. The muscular part consists of the myocytes & it incorporates 70% of the cardiac tissue volume. The interstitial part consists of fibroblasts & collagen while the vascular part is rich in smooth muscle and endothelial cells. An increase in LV wall stress—for example, caused by hypertension induced increase in afterload—will stimulate myocyte hypertrophy, collagen formation and fibroblasts, and thus remodelling of the myocardium with a disproportionate increase in fibrous tissue. These changes will subsequently reduce LV compliance, leading to diastolic dysfunction. Structural changes of the coronary arteries and the increase in both interstitial myocardial fibrosis and in myocardial mass contribute to reduce the vascular coronary flow reserve. In addition, myocardial ischaemic episodes cause transient diastolic dysfunction

An increase in LV wall stress is the principal mechanical factor in the development of LV hypertrophy, and blood pressure the most powerful determinant of LV mass. However, some additional haemodynamic factors play important roles in the development and maintenance of LV hypertrophy. Thus, volume overload also contributes importantly to the development of cardiac hypertrophy. Although the exact mechanism by which sodium intake influences LV mass is unclear, a high salt intake could expand intravascular volume and increase LV preload. Hypertrophy of the arterial resistance vessels with an increased peripheral vascular resistance is present in established hypertension.

LVH affects the heart in many ways - impaired coronary vasodilator reserve, increased myocardial cell diameter without proportional proliferation of capillary vessels results in decreased capillary density and increased diffusion distance, decreased high energy phosphate content, impaired fatty acid oxidation, reduced myocardial glucose transport into the cell. Now we consider how hypertension affects the heart. Apart from LVH, hypertension promotes development of ischaemic injury to myocardium with decreased tolerability to both ischemia and reperfusion because of accelerated coronary atherosclerosis causing stenotic lesions & increased afterload increasing workload and myocardial oxygen demand.

Echocardiographic assessment of LV hypertrophy has a high specificity and sensitivity (both $\geqslant$ 80%). Echocardiography can also provide important additional information, such as other reasons for LV hypertrophy (for example valvular disease, hypertrophic cardiomyopathy),

LV geometric pattern, and information on systolic and diastolic function. Recent development in echocardiographic techniques such as tissue velocity imaging appear to be more sensitive than traditional Doppler echocardiography to detect early signs of hypertensive heart disease such as systolic or diastolic dysfunction. New three dimensional imaging techniques, including magnetic resonance imaging, advanced computed tomography techniques and three dimensional echocardiography, can measure myocardial mass more accurately than conventional echocardiographic techniques. This may offer advantages, especially in relation to mechanistic studies, but may also give additional information on myocardial fibrosis and other structural alterations.

The presence of LV hypertrophy is a strong independent risk factor for future cardiac events and all cause mortality. The risk for sudden cardiac death is increased in subjects with LV hypertrophy, independent of the aetiology, and this event occurs also in individuals with no or only mild prior symptoms related to cardiovascular disease.

Thus from the above study and discussion we see that hypertension is the main cause for development of LVH and LVH can affect the health of the heart in various ways. Sudden cardiac death is also increased in patients with LVH and can also cause ischemic injury to the heart. In our study we find a significant number of hypertensive subjects having left ventricular hypertrophy and thus the risk of various cardiac ailments including sudden cardiac death.

**CONCLUSION**: Hypertension is one of the leading causes of the global burden of disease. Hypertension doubles the risk of cardiovascular diseases including coronary artery diseases and congestive heart failure. It leads to left ventricular hypertrophy which add to the ischemic injury to heart. So timely identification of left ventricular hypertrophy and its aggressive management is the key. Aggressive control of hypertension can reverse left ventricular hypertrophy and reduce the risk of cardiovascular disease.

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