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Original Article

Internal Medicine Section

Factors Contributing to Development and Reversal of LVH: A Pilot Study

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ABSTRACT

Introduction: Left Ventricular Hypertrophy (LVH) often reflects as a physiological adaptation to chronic pressure overload. It has been identified as a strong independent risk factor of all-cause mortality and adverse cardiac events. Since not all subjects with hypertension develop LVH, understanding the clinical factors contributing to the development of LVH and the appropriate diagnostic and treatment strategies may help clinicians in conducting more definitive evaluation and managing the disease effectively.

Aim: To assess the incidence of LVH in hypertensive subjects and the factors influencing its development and reversal. The study also evaluated the most effective diagnostic technique and therapy that could improve the disease symptoms and prognosis.

Materials and Methods: The prospective study, conducted at Jagadguru Sri Shivarathreeshwara (JSS) Medical College JSS University, Mysore, India, included 50 patients with hypertension. Detailed history of the recruited subjects was collected from patient records and through physical examination. Demographic and clinical characteristics such as age, gender, BMI, and stage of hypertension (stage I HTN and stage II HTN) were also obtained. Funduscopic examination was done for all patients for evidence of hypertensive retinopathy. Echocardiography (ECHO), electrocardiography (ECG), and chest X-Ray were used for detection of LVH. The patients were reviewed after six months and reassessment of LVH was carried out. Statistical analysis was conducted using SPSS software and R 3.2 package.

Results: Angiotensin-Converting Enzyme (ACE) inhibitors were found to be more effective in the treatment of LVH when compared to calcium channel blockers and beta blockers. ECHO was found to be the best method to diagnose LVH. In patients with stage I HTN, 47.1% had normal LVM. Around 53% of the subjects with stage I HTN and all with stage II HTN had abnormal LVM. Retinal changes were noted in 96.2% of abnormal LVM patients and 50% of normal LVM patients. A positive association between BMI and LVH (OR: 1.39) was also noted.

Conclusion: BMI may positively influence LVH regression. The presence of retinopathy, in addition to LVH, suggests an increased chance of regression with anti-hypertensive treatment.

Keywords: Hypertrophy, BMI, Retinopathy, LVM, Echocardiography

INTRODUCTION

Hypertension, which can cause target organ damage to kidney, retina, and heart has been identified as the fourth largest mortality risk factor in the world. Left Ventricular Hypertrophy (LVH) denotes a pathophysiologic condition that can arise due to intrinsic (cardiomyopathy), or secondary to extrinsic stimuli, such as elevated blood pressure or volume associated with hypertension and valvular disease [1]. It is associated with numerous adverse cardiac outcomes including atrial fibrillation, myocardial infarction, diastolic/systolic heart failure, and sudden death. In a clinical setting, LVH is defined by an increase in Left Ventricular Mass [LVM] [2]. Based on the ratio of LV wall thickness to cavity dimensions, hypertrophy is classified into two types: concentric and eccentric. The relative wall thickness is not increased in the eccentric type, while significant increase in wall thickness can be seen in concentric variety [3].

The molecular and pathological mechanisms of diastolic dysfunction in LVH are not clearly elucidated and are proposed to include vascular dysfunction, changes in extra cellular matrix, and variation in mechano-elastical properties of cardiomyocytes [4]. Many studies have confirmed high frequency of LVH in hypertensive subjects and the major factors contributing to this association include age, obesity, diabetes [5-8]. Literature evidence also substantiates the association between LVH and increased risk for cardiovascular disease morbidity/mortality [9]. The electrocardiogram (ECG), due to its easy functionality and universal availability, is one of the most commonly used diagnostic techniques for LVH. Echocardiography (ECHO) is the best diagnostic procedure of choice and radiographic cardiac examination (chest X-ray) assists in LVH detection [10-12]. The commonly prescribed Anti hypertensive agents like diuretics, calcium channel blockers, beta blockers, and Angiotensin-Converting Enzyme (ACE) inhibitors promote regression in LVH. Hence choice of treatment may be highly crucial in hypertensive patients with LVH [13]. ACE inhibitors have significant beneficial effects in left ventricular remodeling and regression of LVH [14].

AIM

The present pilot study was aimed at finding the incidence of LVH, and factors influencing its development and reversal in hypertensive subjects from South India. Secondary aim of the study was to find association of LVH with other co-existing factors.

MATERIALS AND METHODS

A single-center, prospective observational study was carried out at the Department of Medicine, Jagadguru Sri Shivarathreeshwara Medical College (JSSMC), JSS University, Mysore, India. Fifty patients who attended outpatient/inpatient department and detected to be hypertensive based on clinical symptoms and examination (>140/90 mmHg) were selected for the study. The inclusion criteria considered were a newly diagnosed case of hypertension or hypertensive patients receiving treatment for not more than two weeks. Hypertensive subjects with valvular, ischemic or primary myocardial diseases or secondary hypertension were excluded.

A detailed history of patients like age, sex, family history, mode of transmission, treatment history, and history of co-existing illness was taken. A thorough physical examination was conducted. The clinical variables evaluated include: BMI, hypertension stage I and II, complete blood count, urine routine, blood urea, serum creatinine, and lipid profile. Funduscopic examination was done for all patients for evidence of hypertensive retinopathy and grading was done according to Keith-Wagener-Barker classification [15]. The use of anti-hypertensive drugs namely ACE inhibitors, beta blockers, and calcium channel blockers, either as mono or combination therapy along with diuretics was also evaluated. The choice of these drugs was based on the physician's prescription and convenience of the patients. Chest X-ray, ECG, and ECHO were performed to diagnose LVH. All the patients were subjected to M-mode ECHO to assess LV dimensions. Left ventricular echograms were measured at or just below the tips of the mitral leaflets in areas of recording that showed largest left ventricular internal diameter (LVIDd), posterior wall thickness (LVPWTd) and interventricular septal thickness (IVSd]). LVM was calculated using Echocardiography by Penn-cube formula [16]. The presence of >0.5 cardiothoracic ratio in a chest X-ray (posterior-anterior view was taken as an evidence of cardiomegaly. Age and BMI were taken as continuous variables. The classification of recruited subjects according to Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) report was as follows: Normal:>120 mmHg/>80 mm Hg; Pre-hypertension: 120-139/80-89 mm Hg; Stage 1 hypertension: 140-159/90-99 mm Hg; and Stage 2 hypertension:>160/100 mmHg [17]. The patients were reassessed for LVH, blood pressure control, and other variables at the end of six months.

LVM-associated variables before and after treatment were compared separately. The groups were compared for demographic, clinical, and treatment characteristics. Chi-square test was used for categorical variables and t-test for continuous variables. Fisher's-exact test was used for variables with expected count >5. Mann-Whitney U test was used for continuous variables that were not normal. Univariate logistic regression was performed to measure the association between the dependent and independent variables. A p-value ≤0.2 was considered as the cut-off for inclusion of the variables in multivariate analysis. A multivariate logistic regression was performed to verify the factors associated with the LVH before treatment and LVM regression after treatment. The 95% confidence interval was noted and p-value <0.05 was considered significant. The reference groups considered for gender were female, stage 1 hypertension, abnormal retinopathy, not on ACE inhibitors, beta blockers, calcium channel blockers and combination therapy for treatment history and normal LVM. The LVM status of regression or hypertrophy was taken as dependent variable for evaluating treatment outcome. The factors associated with LVM were assessed by classifying patients into normal or abnormal based on the LVM before treatment. LVM>196g for men and >167g for women was considered as abnormal. The agreement of chest X-ray, ECG and ECHO before treatment of LVM was verified by Fleiss kappa test with statistical significance taken as p< 0.05. Statistical analysis was performed using the Statistical Package for Social Sciences version 22 (SPSS Inc. Chicago, IL, USA) and R 3.2 package.

RESULTS

Out of 50 recruited subjects, 40 patients were reassessed for LVH, blood pressure control and other variables at the end of six months. The mean±standard deviation (sd) age of the population was 47.53±11.92 with 21 males and 19 females. The BMI mean±sd was 23.68±2.43.

The assessment of before treatment LVM showed that ECHO detected maximum patients, followed by ECG and Chest X-ray. The techniques had low agreement (Fleiss kappa= 0.139, p= 0.129), but the test value was not significant, indicating the methods differ in assessment of LVM. ECHO could assess more number of patients with LVM abnormality. The treatment regimen of patients included ACE inhibitors, beta blockers, and calcium channel blockers either as mono or on combination therapy along with diuretics.

Comparison of the demographic and clinical characteristics of patients with abnormal/normal LVM before treatment is listed in [Table/Fig-1]. It showed that age, hypertension and retinopathy staging were statistically significant.

Median (range) age noted in patients with abnormal LVM was 52.5(22-71), whereas it was 35(31-56) in the normal LVM group. In patients with stage I HTN, 47.1% had normal LVM. Around 53% of the subjects with stage I HTN and all with stage II HTN had abnormal LVM around 96% of abnormal LVM patients and 50% of normal patients had cataract. No difference between the groups was noted with regard to gender and BMI. The comparison of treatment outcome between regression and hypertrophy groups considered the following variables: age, gender, BMI, hypertension staging, funduscopy of retinopathy staging, drugs used for treatment and LVM before treatment. None of them achieved significance at the 0.05 level [Table/Fig-2].

Variables	Groups/ descriptive	LVM		Total	p-value
		Normal	Abnormal		
Age (in years)	Median (Range)	35(31-56)	52.5(22-71)	48.5(22-71)	0.012
BMI	Mean±SD	22.36±2.24	24.01±2.40	23.68±2.43	0.086
Gender	Male	3(14.3)	18(85.7)	21	0.442
	Female	5(26.3)	14(73.7)	19	
Hypertension Stage	I stage	8(47.1)	9(52.9)	17	<0.001
	II stage	0(0)	23(100)	23	
Funduscopy retinopathy Staging	Normal	7(50)	7(50)	14	0.001
	Abnormal	1(3.8)	25(96.2)	26	
Total		8(20)	32(80)	40	

[Table/Fig-1]: Comparison of demographic and clinical characteristics of patients with normal/ abnormal LVM before treatment.

Variables	Groups	LVM status		p-value
		Hypertrophy	Regressed	
Age (in years)		52.57±7.89	46.45±12.45	0.222
BMI		23.17±2.80	23.79±2.38	0.548
Gender	Male	3(14.3)	18(85.7)	0.689
	Female	4(21.1)	15(78.9)	
Hypertension stage	I stage	3(17.6)	14(82.4)	1
	II stage	4(17.4)	19(82.6)	
Funduscopyretinopathy staging	Normal	3(21.4)	11(78.6)	0.679
	Abnormal	4(15.4)	22(84.6)	
ACE inhibitors	Not given	6(20)	24(80)	0.656
	Given	1(10)	9(90)	
Beta blockers	Not given	4(13.3)	26(86.7)	0.338
	Given	3(30)	7(70)	
Calcium channel blockers	Not given	5(16.7)	25(83.3)	1
	Given	2(20)	8(80)	
Combination drug	Not given	6(20)	24(80)	0.656
	Given	1(10)	9(90)	
LVM	Normal	2(25)	6(75)	0.611
	Abnormal	5(15.6)	27(84.4)	
Total		7(17.5)	33(82.5)	

[Table/Fig-2]: Assessment of treatment outcome based on LVM status of regression or hypertrophy.

Univariate analysis of LVM before treatment had revealed age, BMI, and funduscopy retinopathy staging were within the ≤0.2 cut-off p-value [Table/Fig-3]. Women were 2.14 times more likely than men to have abnormal LV mass, but the estimate did not reach p-value threshold of cut-off. All the abnormal LVM patients had stage II hypertension; hence, the stage of hypertension was not considered for univariate and multivariate logistic regression. The multivariate analysis was performed by including all above three variables. The likelihood of abnormal LVM in the abnormal retinopathy staging patients was 14.82. The age and BMI had OR of 1.102 and 1.586 respectively with a narrow 95% CI.

The results of univariate analysis of after treatment LVM status [Table/Fig-4] revealed that LVM regression was 1.6 times more in male patients than female. Patient with concurrent retinopathy were 1.5 times more likely to have regression than the normal stage. The likelihood to undergo regression was 2.25 times more in patients receiving ACE inhibitors as mono or combination therapy. Patients with abnormal LVM were 1.8 times more likely to undergo regression than those with normal LVM. The chance of regression was 64.1% lesser in patients receiving beta blockers than those who were not on the drugs. None of the variables were within the p≤0.2 threshold for their inclusion in the multivariate logistic analysis. Hence multivariate analysis could not be performed for after treatment LVM.

DISCUSSION

In concurrence with the previous findings, the present study has demonstrated that ECHO was the best detection method for LVH. The method has been found to be more sensitive and specific in diagnosing the LVH when compared to ECG and chest X-ray. ECG is the most cost effective tool for LVH detection. However, the method is less accurate compared to its other counterparts. Framingham heart study showed that unlike its counterpart ECG, ECHO determined LVH is a common finding [18]. However, studies have also shown for assessing the increased risk, both ECG and ECHO should be performed [19]. Considering the improved sensitivity, we used LVH detected by ECHO as the reference point for analysing the influencing factors.

Variables	Odds ratio	95% CI	p-value
Univariate logistic regression (LVM)			
Age (in years)	1.100	1.012-1.196	0.026
BMI	1.395	0.942-2.067	0.096
Gender(male)	2.143	0.436-10.536	0.348
Funduscopyretinopathy staging (abnormal)	25.000	2.617-238.787	0.005
Multivariate logistic regression (LVM)			
Age	1.102	0.980-1.239	0.103
BMI	1.586	0.891-2.822	0.117
Funduscopyretinopathy staging(abnormal)	14.819	1.273-172.498	0.031
Constant	0.000		0.070

[Table/Fig-3]: Factors associated with before treatment LVM assessed by univariate and multivariate logistic regression respectively.

Variables	Odds ratio	95% CI	p-value
Age (in years)	0.954	0.883-1.029	0.224
BMI	1.118	0.785-1.592	0.538
Gender (male)	1.600	0.308-8.301	0.576
Hypertension stage (II stage)	1.018	0.196-5.292	0.983
Funduscopyretinopathy staging (abnormal)	1.500	0.284-7.911	0.633
ACE inhibitors (given)	2.250	0.237-21.376	0.480
Beta blockers (given)	0.359	0.065-1.992	0.241
Calcium channel blockers (given)	0.800.	0.129-4.952	0.810
Combination drug(given)	2.250	0.237-21.376	0.480
LVM (abnormal)	1.800	0.279-11.600	0.536

[Table/Fig-4]: Univariate logistic regression analysis for factors associated with LVM status of patients after treatment.

Various studies have shown regression of LVM with improvement of LV diastolic dysfunction following treatment with anti-hypertensive agents, though there is variation in degree of regression. In the present study, compared to beta blockers and calcium channel blockers, ACE inhibitors were found to be more effective in facilitating regression of LV hypertrophy (OR: 2.250). Calcium channel blockers were less effective (OR: 0.800) than ACE inhibitors, and beta blockers (OR: 0.359) were the least. However, combination therapy was better in LVM reduction (OR: 2.250). The corresponding decrease in LVM index noted by Schmieder et al., with the treatment were 12% with ACE inhibitors, 11% with calcium channel blockers, 5% with β-blockers, and 8% with diuretics [20]. Gregory et al. have noted 13% LVM regression in patients treated with ACE inhibitors compared to 6% treated with β-blockers, 9% with calcium channel blockers, and 7% with diuretics [21]. The corresponding LVM reported by Dahlof et al., with calcium channel blockers, beta-blockers, and ACE inhibitors were 8.5%, 8% and 15% [22]. The results of meta-analysis and other population-based studies clearly indicate that reduction in LVH was better with ACE inhibitors followed by calcium channel blockers and beta-blockers. The present observation concurs with previous research showing correlation between hypertensive drugs and regression of LVH.

The present study revealed increased risk of retinopathy in patients with LVH. The prevalence of retinopathy was 92.1% in the study by Shirafkan et al., where all the recruited subjects had confirmed LVH [23]. The study by Cupsidi et al., has also concluded that retinopathy is significantly associated with LVH [24]. Kabedi et al., have observed that the risk for developing LVH increases significantly with the severity of hypertensive retinopathy [25].

Funduscopy of retinopathy staging is highly indicative of abnormal LVM. The present study indicates lack of improvement in the LVH in patients without retinal changes. This indicates that in patients with increased LVM without retinopathy, it is necessary to consider additional factors contributing to LVH in addition to hypertension. Such patients need to be evaluated further for other causes of LVH. The presence of retinal changes increased the probability of regression after introducing the anti-hypertensive agents.

Diabetes, hypertension, and obesity are some of the major independent risk factors for LVH. LVM of athletes with normal cardiac function is comparable to that of hypertensive patients [26]. The present study has indicated a positive association between BMI and LVH (OR: 1.39). The study by Wong et al., has reported similar findings [4]. Peterson et al., demonstrated that end-diastolic septal, posterior wall thickness, LVM, and relative wall thickness were higher in obese women than non-obese women [27]. Lavie et al., showed that left ventricular adaptation in obesity, regardless of arterial pressure level, consists of eccentric LVH. The 2004 Strong Heart Study has suggested that the presence of metabolic syndrome may positively influence the development of LVM [28]. Levy et al., has reported obesity as an independent predictor of LVH [29].

The study by Voyaki et al., demonstrated that LVH was present in about 46% of hypertensives with metabolic syndrome [30]. Hanevold et al., have found that increasing BMI is associated with a higher LVM index [31]. The study by Shashidharan et al., demonstrated that number of females with increased LVM index was more (63.6%) when compared to males 36.4 %, with higher organ damage in increased LVM index patients [32]. Meta-analysis by Cuspidi et al., showed that the probability of having LVH was much higher in obese cases.

The positive influence of sleep apnea in causing LVH has also been documented [33,34]. In addition, there is literature evidence

highlighting the effect of diabetes on LVH [5]. Genetic conditions may also contribute to LVH development [35].

LIMITATION

The study is limited to a small number of populations. Moreover, it lacks power to assess factors associated with regression of hypertrophy after treatment.

CONCLUSION

ECHO is the method of choice for diagnosing LVH and ACE inhibitors is more effective than other therapeutic agents for improving LVH. BMI positively influence the LVH. The presence of retinopathy, in addition to LVH, suggests an increased chance of regression with anti-hypertensive treatment.

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