PREVALENCE OF LEFT VENTRICULAR HYPERTROPHY IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS

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ABSTRACT

Objective: To detect frequency of left ventricular hypertrophy in patients with newly diagnosed hypertension.
Study Design: Cross sectional study.
Place and Duration of Study: Out patient and inpatient, Department of Medicine of Combined Military Hospital Nowshera, from Sep 2018 to Sep 2019.
Methodology: One hundred and twenty patients were included in this study and they were classified into stage 1 and 2 hypertension according to American college of cardiology guidelines. Electrocardiography of all patients was done and Sokolow-Lyon and Cornell voltage criteria were used to detect left ventricular hypertrophy (LVH). 2-D echocardiography was also done to confirm the presence or absence of left ventricular hypertrophy.
Results: Amongst 120 patients 71 (59.16%) were males and 49 (40.83%) were females with mean age 47.79 ± 0.79 years. There was no significant gender based relationship. In this study 41 (34.16%) patients had stage 1 hypertension whereas 79 (65.83%) had stage 2 hypertension at time of diagnosis. Sokolow-Lyon criteria showed 21 (17.5%) patients had left ventricular hypertrophy, 17 (14.6%) patients had left ventricular hypertrophy as per Cornell Voltage criteria with an overlap of 9 (7.5%). Echocardiography revealed left ventricular hypertrophy in 28 (23.33%) patients amongst which 24 had Stage 2 hypertension and 4 had Stage 1 hypertension (p<0.05).
Conclusion: Significant number of patients with newly diagnosed hypertension already had left ventricular hypertrophy due to lack of screening and education.

Keywords: Cornell voltage criteria, Echocardiography, Hypertension, Left ventricular hypertrophy, Sokolow-Lyon criteria.

INTRODUCTION

Hypertension is a significant global public health problem due to its high prevalence. It is estimated that worldwide hypertension accounts for almost 7.5 million or 12.8% of total annual deaths. By the year 2025, it is estimated that 1.56 billion adults will be affected by hypertension. Hypertension is defined as abnormally raised arterial blood pressure[1]. Many environmental factors including sedentary life style, obesity, smoking, diabetes, excessive salt and alcohol consumption have been identified to accelerate the prevalence of hypertension[2]. It is a main risk factor for stroke, heart failure, coronary heart disease, peripheral vascular disease, renal impairment, visual impairment and retinal hemorrhage[1].

According to Joint National Committee 8 (JNC-8), blood pressure goal in general population aged ≥60 years is systolic blood pressure (SBP) <150 mm Hg and diastolic blood pressure (DBP) <90 mmHg and in population aged <60 years goal SBP is <140 mmHg and goal DBP is <90 mmHg[3].

Left ventricle is the major pumping chamber of heart, which pumps oxygenated blood to aorta that transports blood to other tissues of body. Muscular hypertrophy and thickening of left ventricle occurs when it has to work too hard. This condition is known as left ventricular hypertrophy (LVH). Blood supply to the muscle may become insufficient due to this thickness which can cause cardiac ischemia[4].

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Left ventricular hypertrophy (LVH) is a familiar risk factor in hypertensive patients[5]. LVH is a type of preclinical heart disease which either is induced by pressure or volume overload, genetic factors and various other stimuli are also implicated. Volume or pressure overload initiates cardiac myocyte growth which subsequently causes increase in connective tissue without remarkable disarrangement in interstitial architecture. Combination of volume and pressure overload in hypertensive patients results in a mixture of elongated myocyte, required to accommodate a higher ventricular chamber volume and thickened myocyte, activated due to greater afterload[6]. LVH is a common risk factor for the development of cardiac arrhythmia in hypertensive patients. Impact of different blood pressure components on the development of LVH is variable but many studies have shown a positive correlation between pulse pressure and left ventricular mass[5]. Either wall thickening or chamber dilation can lead to increase in left ventricular mass (LVM). Commonly in response to pressure overload wall thickening occurs and due to volume overload chamber dilates. In order to categorize these two types, echocardiography is used to measure relative wall thickness i.e. ratio between LV wall thickness and diastolic diameter. When the relative wall thickness is increased, it is classified as concentric LVH and if left ventricular wall is not thickened, this condition is known as eccentric LVH. There is another pattern known as concentric remodeling, in which relative wall thickness occurs but LV mass is not increased. Echocardiography studies have shown that any of these LV geometric patterns can be present in hypertensive patients[7].

Multiple factors such as gender, age, body size, diabetes and blood pressure are also involved in etiology of LVH. Left ventricular hypertrophy due to hypertension is also implicated as a risk factor for insulin resistance and raised levels of insulin. There is significant correlation between insulin like growth factor-1 (IGF-1), insulin and left ventricular mass. In Framingham heart study, left ventricular mass heritability was analyzed, which showed that genes determined 30% of left ventricular mass variance[8]. Other than mechanical stress due to raised blood pressure, growth factors, cytokines and neurohormones are also involved in development of hypertrophy[7].

The Framingham Heart Failure Risk Score (FHFRS) was evolved to recognize persons who are at greater risk of heart failure. Left ventricular hypertrophy (LVH) is one of the constituent of FHFRS. Either electrocardiography (ECG) /echocardiography (echo) can be used for assessment of LVH. It has been observed that ECG has less sensitivity for LVH detection, though it is an inexpensive modality, which is easily available and interpreted[9]. In order to assess structure and function of heart echocardiography (ECHO) is most relevant and noninvasive investigation. However it is an expensive method and requirements are strict for examiners, it is not easily available in rural areas as compared to ECG[10]. It is presently not clear that whether echo-LVH or ECG-LVH have same predictive ability for assessment of heart failure and whether mode of detection for LVH changes the FHFRS predictive ability[9]. Due to left ventricular hypertrophy, risk of non fatal complications and death is raised to 2 to 4 fold irrespective of gender, age and other risk factors[6]. The purpose of our study was to detect left ventricular hypertrophy in newly diagnosed hypertensive patients.

METHODOLOGY

It was a cross sectional study conducted in out-patient and inpatient and emergency department of medicine, Combined Military Hospital, Nowshera during the period of thirteen months from September 2018 to September 2019. A total of 120 newly diagnosed hypertensive patients reported during period of study fulfilling inclusion criteria were enrolled. Study was approved by ethical review committee of the hospital vide certificate no. Ethical Committee/DME/09 dated September 2018. Written informed consent was taken from patients involved in the study.

Diagnosis of hypertension was made on the basis of serial blood pressure monitoring. Mercury sphygmomanometer was used to measure blood pressure in sitting posture after taking rest for 5-10 minutes as per standard protocol. Individuals who had average systolic blood pressure more than 130mmHg and diastolic blood pressure more than 80mmHg on three different hospital visits with the gap of one week were declared as hypertensive for this study. These newly diagnosed hypertensive patients were further classified into two groups i.e., Stage 1 (BP 130-139/80-89 mm Hg) or Stage 2 (BP >140/90 mmHg) as per American College of Cardiology and American Heart Association (ACC/AHA) 2017 guidelines. While some patients presented for the first time with either hypertensive urgency or hypertensive emergency. In this study these patients were not taking anti hypertensive treatment at the time of enrolment.

Table-I: Left ventricular hypertrophy prevalence by various criteria.

Variable	Sokolow-lyon+/Cornell voltage -	Sokolow-lyon-/Cornell voltage +	Sokolow-lyon+/Cornell voltage +	Echo+	Sokolow-lyon+/Cornell voltage+/Echo+
Male	7 (5.8%)	6 (5%)	5 (4.2%)	17 (14.2%)	4 (3.3%)
Female	5 (4.2%)	2 (1.6%)	4 (3.3%)	11 (9.2%)	4 (3.3%)
Hypertension stage 1	3 (2.5%)	-	-	4 (3.3%)	-
Hypertension stage 2	18 (15%)	17 (14.2%)	9 (7.5%)	24 (20%)	8 (6.7%)

Table-II: Prevalence of left ventricular hypertrophy by sokolow-lyon criterion.

			Patients Gender		*p*-value
			Female	Male	
Sokolow –Lyon left ventricular hypertrophy	No	Count	40	59	0.835
		% within patients gender	81.6%	83.1%	
	Yes	Count	9	12	
		% within patients gender	18.4%	16.9%	

Table-III: Prevalence of left ventricular hypertrophy by cornell voltage criterion.

			Patients gender		*p*-value
			Female	Male	
Cornell Voltage left ventricular hypertrophy	No	Count	43	60	0.616
		% within patients gender	87.8%	84.5%	
	Yes	Count	6	11	
		% within patients gender	12.2%	15.5%	

Table-IV: Prevalence of left ventricular hypertrophy by echocardiography.

			Patients gender		*p*-value
			Female	Male	
Echo left ventricular hypertrophy	No	Count	38	54	0.849
		% within patients gender	77.6%	76.1%	
	Yes	Count	11	17	
		% within patients gender	22.4%	23.9%	

Table-V: Relationship between hypertension stage and left ventricular hypertrophy.

			Hypertension		*p*-value
			Stage 1	Stage 2	
	left ventricular hypertrophy	Count	4	24	0.012
		%	14.3%	85.7%	
	No left ventricular hypertrophy	Count	37	55	
		%	40.2%	59.8%	

Newly diagnosed patients with hypertension of all age groups, genders and urban and rural population were included in this study. Whereas patients with other co morbid such as ischemic heart disease, diabetes mellitus, moderate to severe obesity, congestive cardiac failure, atrial fibrillation, chronic kidney disease (CKD), endocrine disorders and pregnant women were excluded from the study.

ECG of all newly diagnosed hypertensive patients was done by "Nihon Kohden Cardiofax" and interpreted for presence of left ventricular hypertrophy (LVH) changes on the basis of Sokolow-Lyon and Cornell voltage criteria. 2D-Echo of all patients was also performed by "Aloka Prosound SSD 4000SV" to diagnose LVH.

All data were analyzed using SPSS v.16.0. Quantitative variables were presented as mean (SD) and qualitative data as frequencies and percentages. Chi Square test was used for analysis and *p*-value of ≤0.05 was considered significant.

RESULTS

Study included 120 newly diagnosed hypertensive patients amongst which 49 (40.83%) were females and 71 (59.16%) were males. Mean age at diagnosis was 47.79 ± 0.59. Mean systolic blood pressure at diagnosis was 148.17 ± 0.93 and mean diastolic blood pressure was 95.23 ± 0.74. Amongst patients included in study 41 (34.16%) patients had stage 1 hypertension while 79 (65.83%) had stage 2 hypertension at time of diagnosis. Echocardiography revealed LVH in 28 (23.33%) patients (*p*=0.849) (table-IV) amongst which 24 (85.71%) patients had stage 2 hypertension and only 4 (14.28%) patients had stage 1 hypertension (*p*=0.012) which showed significant relationship between LVH and stage 2 HTN (table-V). Nine patients demonstrated LVH according to both ECG criteria and amongst these 8 patients were confirmed to have LVH on echocardiography (table-I).

Sokolow-Lyon criterion showed 21 (17.5%) patients had LVH (*p*=0.835), among them 12 were males and 9 were females (table-II). In 17 (14.6%) patient LVH was diagnosed as per Cornell Voltage criteria (*p*=0.616) comprising 6 females and 11 males (table-III).

DISCUSSION

Hypertension is a main modifiable risk factor for cardiovascular disease (CVD) and the major cause of preventable death globally. There is clear evidence which shows that lowering blood pressure with help of anti hypertensive medication remarkably reduces mortality and morbidity due to CVD. In spite of this many hypertensive patients remain undiagnosed, or improperly treated[11]. Study showed that in Pakistan main issues in management of hypertension are lack of knowledge, non adherence to treatment, inadequate follow ups and financial limitations which need to be addressed immediately[12]. Due to these limitations, we observed in our study that at time of diagnosis, 79 (65.83%) patients had already progressed to Stage 2 hypertension.

Philippe *et al* reported that R wave measurement in lead aVL on ECG propounded the best relationship to LVM and presence of an echocardiographic LVH[13]. For diagnosis of LVH single ECG criteria has low sensitivity and high specificity. In urban community of Beijing, Jiang *et al* concluded that by combining Sokolow-Lyon voltage and Cornell product criteria sensitivity for LVH detection is increased and it also helped to improve further risk prediction of cardiovascular events and mortality in future[10]. Likewise in our study, amongst 9 patients who fulfilled both Sokolow-Lyon and cornell voltage criteria for LVH, 8 patients were confirmed to have LVH on echocardiography.

There have been contradicting results regarding prevalence of LVH in genders. Cuspidi *et al* concluded from their study that changes in cardiac architecture are present in both untreated and treated hypertensive patients, gender based studies showed similar prevalence in both males and females[14] which was also demonstrated in our study that 11 (22.4%) females and 17 (23.9%) males had LVH on echocardiography and no

significant gender based relationship was observed.

Our results were in congruence with study conducted in Belgarde by Koncarevic *et al* who concluded that 26.2% newly diagnosed hypertensive patients had LVH[15]. Whereas our results showed 23.3% patients had LVH, which proves that target organ damage has already been done in newly diagnosed hypertensive patients. Our findings were also supported by Rita Rani Maggon *et al* who demonstrated target organ damage in treatment naive hypertensive patients and showed significant relationship between microalbuminurea and LVH[16].

Katholi observed that various non pharmacologic and pharmacologic strategies have been implicated in management and prevention of LVH in hypertensive patients. Prevention and regression of LVH has been independently facilitated by reduced sodium intake and weight loss in addition to blood pressure control[17]. In order to treat a patient with left ventricular hypertrophy, anti hypertensive agents have significant role. Díez concluded that all groups of anti hypertensive drugs are involved in LVH regression. Angiotensin-1 receptor blockers were found to have most potent effect followed by calcium channel blockers and then ACE inhibitors. Diuretics and beta blockers also have mild to moderate effect on regression of LVH[18]. Dominguez *et al* studied the effects of carvedilol in hypertensive heart disease and reported that it increased left ventricular ejection fraction and helped in reduction of cardiac dimensions and remodeling, which subsequently reduced mortality[19].

In addition to increased overload, secretion of vasoactive peptides like endothelin-1, norepinephrine, angiotensin II, Rho and Ras proteins, heat shock proteins, calcineurin, some kinases and oxidative stress also contribute to hypertrophic process. Studies on animal models have shown that suppression of biochemical responses can prevent cardiac complications. Latest drugs derived from spironolactone, Cyclosporine-A, scutellarin and humoral factors could be used in antagonizing LVH[20].

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CONCLUSION

Significant number of patients with newly diagnosed hypertension already had left ventricular hypertrophy due to lack of screening and education.

CONFLICT OF INTEREST

This study has no conflict of interest to be declared by any author.

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