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Original Article

Physiology Section

Impact of Prehypertension on Left Ventricular Structure, Function and Geometry

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ABSTRACT

Background: Awareness of prevalence, determinants, and prognosis of asymptomatic untreated prehypertension is still lacking especially in India and subcontinent. The present study was to assess the effects of prehypertension on structure, function and geometrical pattern of left ventricle on the basis of left ventricular mass (LVM), left ventricular mass indexed to height (LVMI/Ht), and relative wall thickness (RWT) recorded by echocardiography based on the American society of echocardiography (ASE) convention.

Methods: The study population included prehypertensives (n 61; 31 M, 30 F) and normotensives (n 38; 19 M, 19 F) between age 25 and 65 years, and were assessed by echocardiography.

Results: It was observed that the stroke volume (SV), cardiac output (CO), cardiac index (CI), body mass index (BMI), body surface area (BSA), were found to be little elevated but was not significant in hypertensive females compared to normotensives. Systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), pulse pressure (PP), mean arterial pressure (MAP), end systolic stress (ESS), and end isovolumetric systolic stress (EISS) were significantly elevated ($p<0.001$) in female prehypertensives compared to normotensives. Left ventricular mass (LVM) was significantly ($p< 0.05$) elevated, indicating alterations in cardiac morphology and functions even during prehypertensive stage. However, in prehypertensive males, SBP, DBP, HR, PP, MAP, ESS, and EISS were significantly (<0.001) raised; ejection fraction (EF%) and fractional fibre shortening (FS%) were noted to be within normal range in both sexes. Prehypertensive males showed changes in left ventricular geometry in the form of concentric remodeling (CR-3.44%), eccentric hypertrophy (EH-3.44%) and concentric hypertrophy (CH-13.79%). Prehypertensive females showed (CR-6.45%), (EH-3.22%) and (CH-6.4%).

Conclusion: Such findings carry prognostic implication and require further population survey involving a larger group. Early diagnosis of prehypertension will help to take necessary preventive measures to reduce mainly the future cardiovascular complications. The care of prehypertensive subjects should include, to reduce the afterload in order to improve the left ventricular contractile state as early as possible. So it is advisable to do routine echocardiography after the age of 40 years.

Keywords: Prehypertension, Left ventricular mass, Relative wall thickness

INTRODUCTION

Hypertension is supposed to be the “silent killer of the mankind” because the patient may or may not be aware of the presence of hypertension [1,2]. WHO and other organizations define hypertension as SBP>140 mmHg and DBP>90mmHg, not taking any antihypertensive medications [3,4]. However, Seventh Report of the Joint National Committee (JNC-7) provides a newer guideline on classification of hypertension [5,6]; SBP between 120-139 mmHg or DBP between 80-89 mmHg fall into prehypertension [7] and possess higher cardiovascular and renal risk in future [8,9]. Prehypertension is commonly prevalent in the general population [10] but there are only a few studies on the prevalence and risk factors available in Indians [11-13].

Prolonged elevation of BP commonly lead to a variety of changes in myocardial structure, coronary vasculature, and conduction system of heart, creeping into development of left ventricular hypertrophy (LVH), coronary artery disease (CAD), angina, myocardial infarction (MI), cardiac arrhythmias, congestive heart failure (CHF) [14-16]. Increase in LVM might be physiological or pathological. Several factors which are associated with increased LVM include age, diet [17], salt intake [18], gender, genetic, stress, blood pressure, body size, physical activity, blood viscosity [19] ageing and obesity [20]. Obesity, Hypertension (HT) and diabetes have been implicated as still more important determinants of increased LVM [21,22].

There is a greater age-related increase in LV wall thickness, LV mass and LV hypertrophy [23], and such changes even during early stage can be detected by echocardiography. Although the prehypertensives do not have symptoms in day-to-day life, because of adaptive changes yet, it is essential to diagnose the prehypertensives and to assess the consequent structural alterations in cardiovascular system with passage of time. In the present study, such investigation was conducted in both the groups i.e. prehypertensives and normotensives, who came for routine health check-up.

AIMS AND OBJECTIVES

To study the impact of prehypertension on the structure, function and geometry of left ventricle in asymptomatic untreated males and females.

MATERIALS AND METHODS

The present study was carried out at SRMS-IMS Hospital, Bareilly, Uttar Pradesh state, India, after obtaining approval from the ethical committee of the institute, and informed written consents were obtained from subjects.

Structural Material: The study population included 61 pre-hypertensive (31 M; 30 F) and 38 normotensive (19 M; 19 F) subjects between age 25 and 65 years. A detailed medical record including history of hypertension with or without medications, diabetes mellitus, non-essential habits like smoking, alcohol consumption, chewing tobacco, physical activity including past and family history were noted. Clinical examination included the

record of their height, weight, blood pressure (SBP, DBP), and resting heart rate (HR).

Inclusion Criteria: Both male and female subjects who visited to OPD for their routine health check up without any cardiorespiratory symptoms.

Exclusion Criteria: Those with any history of recent surgeries, diabetes mellitus, congenital heart disease, rheumatic heart disease, unstable or stable angina, valvular heart disease, pericardial disease and hypertrophic cardiomyopathy which were based on the echocardiographic findings, congestive heart failure, respiratory disease, kidney disease and thyroid dysfunction.

Case Definition: Followed JNC-7 provided newer guidelines on the classification of hypertension [7].

Echocardiography: Two dimensional M-mode echocardiograms (Siemens Acuson P 300, Germany) of all participants were obtained by trained cardiologist assisted by technician. Left ventricular dimensions were obtained in parasternal short axis view, with measurement of interventricular septal thickness (IVST), LV internal dimension in diastole (LVIDd), LV internal dimension in systole (LVIDs) and LV posterior wall thickness (PWT) according to guidelines of American Society of Echocardiography [23,24,12].

The data were analyzed for each group between males and females by using Microsoft Excel 2010 software. Mean ± SD was calculated and unpaired student's t-test was applied. P-value of ≤0.05 was considered as statistically significant, a value of ≤0.01 as very significant and a value of ≤0.001 as highly significant.

RESULTS AND OBSERVATIONS

Among prehypertensives of both sexes, no cardiovascular abnormality was detected in 80-83%, whereas the normotensives the same varied between 89-94%. The prehypertensives of both sexes as shown in [Table/Fig-1,2]. The rest of the subjects showed some compensatory cardiac changes [Table/Fig-3].

	Males			Females		
	Normotensives (n 19)	Prehypertensives (n 31)	p-value	Normotensives (n 19)	Prehypertensives (n 30)	p-value
Age (years)	42.94±13.99	44.40±13.33	0.71	44.78±13.87	47.06 ±13.15	0.56
Height (m)	1.67±0.04	1.65±0.078	0.33	1.63±0.06	1.67 ±0.065	0.016
Weight (kg)	61.42±6.99	60.63±5.95	0.67	57.89±5.72	56.67 ± 5.00	0.67
BMI (kg/m2)	21.94±2.16	22.27±2.60	0.64	21.91±3.24	23.02 ±1.86	0.72
BSA (m^2)	1.69±0.10	1.66±0.10	0.45	1.62±0.08	1.73±0.09	0.51
SBP (mmHg)	109.78±7.48	121.93±3.94	<0.001	108.73±4.67	131±3.09	<0.001
DBP (mmHg)	66±4.65	75.2±5.76	<0.001	66.73±4.43	79.22±4.73	<0.001
HR (beats/min)	77.73±7.22	85.8±5.13	<0.001	78.42±9.21	83.03±5.55	0.031
PP (mmHg)	43.78±5.36	56.73±6.63	<0.001	42±5.07	52.25±5.54	<0.001
MAP (mmHg)	80.60±5.17	90.77±4.19	<0.001	80.79±3.83	96.65±3.36	<0.001

[Table/Fig-1]: Demographic characteristics of the study subjects
Ht: height, Wt: weight, BMI: body mass index, BSA: body surface area, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, PP: pulse pressure, MAP: mean arterial pressure

	Males			Females		
	Normotensives (n 19)	Prehypertensives (n 31)	p-value	Normotensives (n 19)	Prehypertensives (n 30)	p-value
AoD (mm)	29.52±1.26	29.2±2.8	1.00	29.26±2.20	30.0±1.45	NS
LAD (mm)	31.63±1.80	31.6±2.37	1.00	32.10±1.04	32.8±2.11	NS
SV (ml)	74.83±6.64	76.16±1.04	0.36	71.28±11.05	75.04±7.86	<0.001
CO (L/min)	5.82±0.79	6.72±0.71	0.23	5.59±1.08	6.23±0.73	0.25
CI ($L/m^{2)}$	3.47±0.58	3.98±0.08	0.20	3.48±0.75	3.60±0.45	0.23
EF%	63.09±8.17	69.37±16.6	0.37	62.78±13.66	63.4±9.72	0.16
FS%	0.44±0.01	0.43±0.03	0.20	0.44±0.02	0.44±0.007	0.10
ESS (dynes/min)	0.91±0.06	0.98±0.08	<0.001	0.91±0.11	1.08±0.08	<0.001
EISS (dynes/min)	0.55±0.03	0.60±0.07	<0.001	0.56±0.08	0.65±0.06	<0.001
IVST (mm)	10.10±0.45	10.61±1.60	0.18	10.15±0.76	10.25±0.68	0.63
LVIDd (mm)	44.84±1.25	44.33±2.57	0.42	44.15±2.50	44.87±1.87	0.25
LVIDs (mm)	24.94±0.229	25.13±1.13	0.48	24.89±1.37	25±1.29	0.78
PWT (mm)	10.10±0.45	10.55±1.35	0.17	10±5.77	10.25±0.68	0.175
LVM (gms)	157.44±9.23	165.65±31.44	0.27	152.94±11.65	161±17.39	0.05
LVM /BSA	93.43±6.94	99.96±20.81	0.19	94.73±7.82	93.15±10.58	0.17
LVMI /$Ht^{2.7}$	39.36±3.22	43.46±11.87	0.15	41.25±6.25	40.10±5.63	0.57
LVMI /Ht	94.17±5.49	100.68±21.24	0.200	93.99±8.46	96.14±10.44	0.45
TPR	1130±200.36	1247.27±455.06	0.42	1222.06±399.81	1267±247.73	0.61
RWT (mm)	0.45±0.03	0.47±0.08	0.16	0.46±0.05	0.46±0.046	0.82

[Table/Fig-2]: LV structural and functional parameters in the study population
AoD: aortic dimension, LAD: left atrial dimension, SV: stroke volume, CO: cardiac output, CI: cardiac index, EF%: ejection fraction, FS%:fractional shortening, ESS: end systolic stress, EISS: end isovolumetric systolic stress. IVST: interventricular septal thickness, LVIDd: left ventricular internal dimention in diastole, LVIDs: left ventricular internal dimention in systole, PWT: post wall thickness, LVM: left ventricular mass, LVMI/BSA: left ventricular mass indexed to body surface area, LVMI/$Ht^{2.7}$: left ventricular mass indexed to height $^{2.7}$, LVMI/Ht: left ventricular mass indexed to height, TPR: total peripheral resistance, RWT: relative wall thickness

LV geometrical pattern	Males		Females	
	Prehypertensive (n 31)	Normotensives (n 19)	Prehypertensive (n 30)	Normotensives (n 19)
Normal geometry (n)	25 (80.64)	18 (94.73)	25 (83.33)	17 (89.47)
Concentric remodeling (n)	1 (3.22)	1 (5.26)	2 (6.66)	2 (10.52)
Eccentric hypertrophy (n)	1 (3.22)	0	1 (3.33)	0
Concentric hypertrophy (n)	4 (12.90)	0	2 (6.66)	0

[Table/Fig-3]: Distribution of LV geometry in Prehypertensive and normotensive study subjects.
Values in brackets indicate % wise distribution

DISCUSSION

In the early stages of prehypertension, there occurs elevation of adrenergic tone typically characterized by hyperkinetic status i.e. an increased HR, SV, CO, and CI, including TPR [25]. In hypertension, reduced CO is mainly the result of LV diastolic dysfunction in the course of LVH and decreased beta-receptor reactivity in the mode of "down-regulation".

In the present study, the haemodynamic profile in patients with LVH suggested that increased LV filling which was due to volume overload or elevated venous return, which was responsible for elevated SV but not disturbing normal systolic function. Such observations had also been reported by Ganau et al., & Garg et al., [26,12]. Left ventricular contractility in this study was assessed by left ventricular EF% and FS%, which was normal and reflected the dynamics of work done by the LV [27-29].

Further, elevated values of SV, CO & CI noted in prehypertensives of both sexes, may possibly be due to compensatory changes caused by pressure overload [Table/Fig-1,2]. Such mechanisms involved, were probably due to increased sympathetic activity (norepinephrine release) and involvement of rennin-angiotensin-aldosterone system [25]. Balci et al., [30] observed the similar results related to systolic function in essential hypertension. However, de Simone et al., [31] reported the opposite values in hypertensives with LVF, where CO has been diminished. In another study, it has been reported that there is absence of hypertrophy in diastolic dysfunction patients [32]. Further, concentric hypertrophy and global diastolic dysfunction had also been reported in prehypertensives [30].

PWT, IVST, LVM, & RWT were raised but not so significant in prehypertensive males & females may be due to early stage of hyperdynamic circulation & LV wall stress. RWT provides information regarding LV geometry independent of other calculations. ESS & EISS were significantly raised in early stage, TPR though raised but not so significant in prehypertensive males & females depicted increased afterload. Moreover increase in TPR depicts increase in arteriolar radius [Table/Fig-2].

In the present study, visual assessment of active (E) and passive (A) transmitral peak velocities and their ratio (E/A ratio) during echocardiography is used as a routine procedure and had shown a decrease indicating compensatory diastolic dysfunction in prehypertensive group [33,34]. The various types of observation in the above mentioned studies including the present study portray the dissimilar compensatory adaptations related to morphology and function especially involving cardiovascular system. On considering the influence of compliance factor in LVH, it has been reported that intense accumulation of collagen or ischemic fibrotic changes cause increased interventricular pressure causing mortality and morbidity with eccentric hypertrophy, and even more in patients with concentric remodeling [30].

Frank starling's law which is being followed by normal compliant cardiac muscles but altered even during prehypertensive phase, where afterload has been increased. So, preload (volume overload) and afterload (pressure overload) [Table/Fig-1] combined together gradually invite the vicious cycle causing hyperdynamic circulation, increase in HR and the cardiac morphological changes thereby did the concerted effort to maintain functional status of the body (compensatory mechanism).Prehypertensive stage when detected early, the preventive and curative aspects of treatment therefore, might be initiated to reduce the cardiovascular risk factors [35]. In such cases, the regular physical exercise, modification of diet, yoga and relaxation therapy, low salt intake and overall life style modification are essential and to be judiciously followed to avoid further cardiovascular and renal complications.

CONCLUSION

The overall left ventricular systolic functions were normal in the asymptomatic prehypertensive subjects. However, the contractility functions (EF% and FS%) were minimally impaired because the compensatory changes are maintaining the function by undergoing remodeling and LVH. Afterload was also significantly increased in prehypertensives. More public awareness programs are to be set up to educate the society about the early detection and control of blood pressure by various means for improvement of cardiac pathophysiology even when the prehypertension is in asymptomatic stage to reduce the future cardiac morbidity and mortality. In addition, it is even better to conduct routine echocardiography after the age of 40 years in all (i.e. normotensives, prehypertensives and hypertensives) for future healthful population.

APPENDIX A

Calculations: Stroke volume (SV) = $(LVIDd)^3$ - $(LVIDs)^3$, Cardiac output (CO) = SV × HR, Cardiac Index (CI) = CO / BSA, LVMI (indexed to BSA) = LVM/ BSA, LVMI (indexed to Height) = LVM/Ht (m), Ejection fraction (EF %) = $(LVIDd)^3$ – $(LVIDs)^3$ × 100 / $(LVIDd)^3$, Fractional shortening (FS%) = (LVIDd – LVIDs) × 100 / LVIDd, Total peripheral resistance (TPR) = (MBP × 80 /CO), Left ventricular mass (LVM) = 0.8 [1.04 (IVS+ LVIDd+ PWT)^3- $(LVIDd)^3$+0.6, Body surface area (BSA) = [(Ht (cm) × Wt (Kg) /3600] ½, Body mass index (BMI) = Wt (kg) /Ht $(m)^2$, Relative wall thickness (RWT) = 2 × PWT /LVIDs, End systolic stress (ESS) =0.334 X SBP X LVIDs /PWTX (1+PWT/ LVIDs), End isovolumetric systolic stress (EISS) =0.334 X DBP X LVIDs /PWT X (1+PWT /LVIDs)

APPENDIX B

The pattern of LV remodeling was determined using LVMI and RWT considering normal values of Indian Asian males -118/0.50 and Indian Asian females- 107/0.47 residing in U.K. Normal geometry (NG) has been considered when both values of LVMI and RWT are within normal limits (males -118/0.50; females-107/0.47), whereas concentric remodeling (CR)- normal LVMI and increased RWT (males -118/ >0.50; females- 107/ >0.47), eccentric hypertrophy (EH)- increased LVMI and normal RWT (males >118/ 0.50; females >107/ 0.47), whereas concentric hypertrophy (CH)- both LVMI and RWT are increased (males >118/ >0.50; females >107/ >0.47) [36,37].

*Appendix A & B: In case, the experts and editorial board feel that these calculations are better to include, then these may be added in Material and Methods.

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