Hypertensive Cardiac Myocyte Hypertrophy: a cardinal attribute in the sequence of biological events manifesting Echocardiographic left ventricular Hypertrophy.

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ABSTRACT

Background: Ecocardiography has been widely used as a realtime, reliable tool in the early detection and differentiation of cardiac geometry and function. In Hypertension and prehypertension this tool has become an asset in early assessment and detection of Left ventricular hypertrophy (LVH) and its morphological categorization. Therefore, it helps in the process of prevention detection and prompt management of hypertensive LVH. Prehypertension has now become the criteria of management of raised blood pressure in intial stages so that the levels of BP should not go above to produce full fledged hypertension. By detecting so the irreversible damage can be curtailed and morbidity and mortality can be restricted and minimized since most of the patients are asymptomatic in early days.

Methods design: Study design includes 120 Hypertensive patients as cases who attended OPD in the Department of Medicine at Sri Ram Murti Smarak Institute of Medical Sciences, and Rohilkhand hospital, Bareilly, (U.P.). Equal number of normal subjects were taken as controls who attended the OPD for their routine health checkups. Study subjects were sged between 25-70 years of both the sexes. ECG, 2D-Echocardiography and color Doppler was later performed after thorough clinical history taking, clinical examination and demographic assessment. Echocardiographic parameters were recorded in accordance with American Society of Echocardiography (ASE) Convention. LVH was calculated and categorized by LVMI and RWT relationship with the given formula.

Result: It was seen that the hyper dynamic circulation present in hypertension is demarcated by raised Systolic and Diastolic BP >140/90 mmHg according to WHO-JNC-7 Criteria., Echocardiographic parameters like Left Ventricular Internal Dimention in Diastole (LVIDd). Left

Ventricular Internal Dimention in Systole (LVIDs). Interventricular Septal Thickness (IVST), Posterior wall Thickness (PWT), Left Ventricular mass (LVM) and Left Ventricular Mass Index (LVMI) were significantly deranges due to the hyper dynamic circulation and sympathetic overstimulation. Relative wall Thickness (RWT) and Left Ventricular Mass (LVM) were calculated by given formulae. LVMI has a great role in determining the Left ventricular hypertrophy (LVH) and its classification as Normal geometry, concentric hypertrophy, concentric remodeling and eccentric hypertrophy. End systolic stress (ESS), End isovolumetric stress (EISS) & total peripheral resistance (TPR) were also found to be increased in the present study. Systolic function parameters like Ejection fraction (EF%) & Fractional Fiber Shortening (FFS%) were also raised significantly in the present study.

Conclusion: Study indicated a prompt action to be undertaken for early detection of asymptomatic prehypertension in the initial stages which will help in reduction of the morbidity and mortality. Early echocardiographic assessment will benefit the patient to undertake necessary preventive steps for the management of the disease. Therefore it is advocated to include routine echocardiography after 40 years of age even in normotensives to rule out early myocardial remodeling.

INTRODUCTION

It is widely known that high arterial blood pressure (BP) may lead to variety of organic derangements. Hypertension and prehypertension (as categorized in the JNC-7 criteria) has a significant systemic and cardiac impact and lead to the production of structural remodeling and functional failure. Cardiac geometry is severely hampered in raised blood pressure and in long duration will lead to left ventricular hypertrophy (LVH). Many cardiac parameters are their which may reflect the deteriorating condition of the heart in early stages of hypertension and can be detected early by 2d-echocardiography and colour Doppler. The hypertensive patients are initially asymptomatic, it is therefore said that hypertension and prehypertension both are the silent killers of the mankind. Despite of the cause of raised blood pressure it cannot be ignored and should be detected early so as to reduce the morbidity and mortality. The brunt of attack mainly falls on the left side of the heart due to raised pressures as compared to right side. Cardiac myocyte elongation is the hallmark of hypertensive LVH. The hypertrophy may or may not be limited to left ventricle but may progress to the other chambers of the heart too. LVH has several forms which are categorized as Normal geometry, Concentric remodeling, Concentric

hypertrophy and Eccentric hypertrophy. These categorizations are dependent on the dimensions of the cardiac wall parameters like RWT and LVMI (1). It is therefore validated that hypertension imposes remodeling changes in cardiac chambers for procuring LVH (2). LVM has a direct relationship with height, weight and body size (BMI) BSA (3,4) genetic configuration (5,6), sex (7,8), age (6,9), race (10,11), and sedentary lifestyle (12) etc.

These structural changes may be attributed due to long standing hyperdynamic circulation. There are evidences that variety of hypertrophic changes also exist in long older age groups. The declining aortic compliance and LA enlargement is also seen in prehypertensive cases (9,13). Other metabolic diseases also impart detrimental effect on the cardiac structure and function like Obesity and metabolic syndrome (14), diabetes mellitus(15,16), menopause etc. Chronic stress has a strong correlation with LVH (17,18), high salt intake, increased blood viscosity (19), impaired GTT (20), hyperinsulinemia (21), smoking and alcohol (19) hypercholesterolemia (22), chronic sympathetic stimulation (23) also potentiates LV hypertrophy due to excess release of angiotensin-II (5). These changes are potentiating hypertrophy and hyperplasia of myocardial cell (24).

This comparative study design explores about the correlation and implications of raised hemodynamic circulation and sympathetic overstimulation in hypertensives and normal subjects by echocardiography.

MATERIAL AND METHODS

This study includes 120 hypertensive patients as cases, of both the sexes between 25-70 years of age, who attended cardiac OPD of Medicine Department at SRMS-IMS hospital and Rohilkhand hospital, Bareilly, (U.P). Same number of controls were taken as normal patients who attended the OPD for their routine health checkups.

Study subjects were informed about the study design and a formal consent was obtained to participate in the study. A detailed clinical history and clinical examination was performed to rule out other associated illnesses. Demographic parameters like age, sex, height, weight, was recorded and BSA, and BMI was calculated by the given formula. Later, ECG and echocardiography with colour Doppler was performed and the data was recorded in accordance

with the American society of echocardiography (ASE) convention. Hypertension was classified in accordance with WHO-JNC-7 criteria of classifying blood pressure.

Inclusion criteria:

Male and females subjects between 25 to 70 years of age of both the sexes having blood pressure more than 140/90 mmHg were taken as cases others who had normal BP and attended the OPD for routine health checkup were considered as controls.

Exclusion criteria: Study subjects who belonged to any known medical diseases or on medications like Diabetes Mellitus, Hypertension, Hypo or Hyperthyroidism, Cardiac disorders, Obesity etc were excluded. Also those who have undergone any previous surgeries or cardiac surgery like CABG were excluded.

Case definition: WHO-JNC-7 criteria was applied to classify hypertension and the study subjects were not taking any antihypertensive medications (25).

Echocardiography: 2-D Eechocardiography and color Doppler machine (GE by PHILIPS) was deployed and parameters were recorded by the trained echo technician under the supervision of the cardiologist. Parasternal transthoracic views were sequentially taken to record LV parameters like LVIDd, LVIDs, IVST, RWT, PWT, LVM LVMI etc. Functional parameters like ESS, EISS, EF%, FFS% etc were recorded to assess the systolic and diastolic function of the LV. LVMI indexed to Ht and LVMI indexed to the allometric power 2.7 were calculated according to the ASE convension by the given formulas (27).

Statistical analysis:

Data analyses was performed with SPSS software version 11.0.1 (SPSS Inc., Chicago, IL). 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.

OBSERVATIONS AND RESULTS

TABLE-1: GENERAL PHYSICAL PARAMETERS OF STUDY SUBJECTS.

PARAMETRS	FEMALES (n=60)			MALES (n=60)		
Variables	**CONTROLS (n=30)**	**CASES (n=30)**	**p-value**	**CONTROLS (n=30)**	**CASES (n=30)**	**P-**
Age (yrs)	48.40 ±10.65	49.83 ± 7.31	0.212	48.36 ± 11.20	49.84 ± 11050	0.0533
Height (m)	1.53 ± 0.08	1.33 ± 0.02	0.1822	1.90 ± 0.03	1.90 ± 0.05	1.0031
Weight (kg)	59.52 ± 4.14	60.95 ± 6.52	0.0741	59.33 ± 7.83	69.50 ± 7.99	0.0265
BMI (kg /m^2)	21.88 ± 1.76	21.97 ± 2.80	0.8922	20.71 ± 2.18	25.63 ± 2.66	0.0011
BSA (kg /m^2)	1.44 ± 0.07	1.61 ± 0.09	0.0211	1.93 ± 0.11	1.99 ± 0.07	0.0087

BMI- Body mass index, BSA- Body surface area, SBP- Systolic blood pressure, DBP- Diastolic blood pressure, PP- Pulse pressure, MAP- Mean arterial pressure.

In this study it was noted that Age, Height, and Weight was not significantly altered in the study subjects but BMI, and BSA showed were recorded to be significantly altered both in males and in females (Table-1)

TABLE-2: HAEMODYNAMIC PROFILE OF THE STUDY SUBJECTS.

	FEMALES (n=60)		MALES (n=60)	

PARAMETER	CONTROLS (n=30)	CASES (n=30)	P-value	CONTROLS (n=30)	CASES (n=30)	P- value
SBP (mmHg)	132.50 ± 8.22	144.66 ± 8.84	0.0001	127.73 ±7.79	147.87 ± 13.13	0.0001
DBP (mmHg)	73.80 ± 6.99	98.16 ± 9.97	0.0003	77.46 ± 6.99	99.95 ± 7.45	0.0001
PP (mm Hg)	48.70 ± 6.98	51.50 ± 9.13	0.3472	51.26 ± 7.74	55.91 ± 11.01	0.0831
MAP (mmHg)	89.70 ± 7.71	110.66 ± 9.14	0.0001	99.22 ± 5.98	119.92 ± 8.16	0.0001
Heart rate /min)	81.42 ± 5.90	87.41 ± 5.77	0.0411	87.30 ± 6.47	89.69 ± 4.99	0.2201

SBP-Systolic Blood Pressure , DBP-Diastolic blood pressure, PP-Pulse pressure, MAP-Mean arterial Pressure, HR-Heart rate.

Other parameters like SBP, DBP, MAP were significantly raised in prehypertensive cases and therefore they reflected about characteristic impact on the LV structure and function also they indicated of hyperdynamic circulation as a result of sympathetic overstimulation. (Table-2)

TABLE-3: LV STRUCTURAL AND FUNCTIONAL PARAMETERS IN THE STUDY POPULATION.

	FEMALES			MALES		
PARAMETER	CONTROLS (n=30)	CASES (n=30)	p-value	CONTROLS (n=30)	CASES (n=30)	P-value

LVIDd (mm)	48.55 ± 1.90	41.00 ± 2.65	0.2834	47.76 ± 2.88	46.19 ± 1.88	0.3445
LVIDs (mm)	28.92 ± 0.90	27.99 ± 1.66	0.3054	28.10 ± 0.90	28.26 ± 2.30	0.5643
PWT (mm)	14.07 ± 0.55	15.70 ± 1.89	0.0021	16.13 ± 0.88	6.26 ± 2.98	0.0001
IVST (mm)	12.12 ± 0.57	13.87 ± 1.65	0.0017	12.13 ± 0.55	14.47 ± 2.30	0.0001
LVM (gm)	166.55 ± 7.60	170.44 ± 30.81	0.0091	160.14 ± 19.31	234.41 ± 19.08	0.0001
LVMI $^{2.7}$)	55.14 ± 7.31	47.58 ± 2.13	0.5177	40.42 ± 4.98	53.54 ± 17.55	0.0001
RWT	0.48 ±0.08	0.49 ±0.060	0.0151	0.47 ±0.08	0.58 ±0.07	0.0001

LVIDd- Left ventricular internal dimension in diastole, LVIDs- Left ventricular internal dimension in systole, PWT- Posterior wall thickness, IVST- Interventricular septal thickness, LVM- Left ventricular mass, LVMI- Left ventricular mass index index, RWT- Relative wall thickness.

Echocardiographic parameters like LVIDD, LVIDS were not significantly alterd in both the males and females. But PWT, IVST, LVM, LVMI, RWT were severely and significantly affected procuring left ventricular hypertrophy (Table-3).

TABLE-4: FUNCTIONAL ECHOCARDIOGRAPHIC PARAMETERS OF LEFT VENTRICLE.

	FEMALES (n=60)			**MALES (n=60)**		
VARIABLE	**CONTROLS (n=30)**	**CASES (n=30)**	**P-VALUE**	**CONTROLS (n=30)**	**CASES (n 30)**	**P- VALUE**

SV (ml)	78.21 ± 6.62	71.79 ± 11.93	0.3051	77.44 ± 10.10	78.47 ± 8.50	0.3523
CO L/min)	7.11 ± 0.78	7.12 ± 1.04	0.9623	7.34 ± 0.88	6.65 ± 0.80	0.1612
CI (L/m^2)	4.72 ± 0.55	4.61 ± 0.69	0.4645	3.77 ± 0.58	3.77 ± 0.56	0.7578
EF%	77.17 ± 8.34	70.18 ± 12.45	0.2998	76.46 ± 11.02	77.22 ±11.79	0.3398
FS%	0.48 ± 0.01	0.47 ± 0.01	1.0032	0.46 ± 0.20	0.44 ± 0.02	1.0021
ESS s/$cm^{2)}$	1.00 ± 0.17	1.33 ± 0.12	0.0023	1.22 ± 0.07	1.06 ± 0.10	0.7134
EISS s/$cm^{2)}$	0.66 ± 0.06	0.80 ± 0.11	0.0001	0.66 ± 0.03	0.70 ± 0.07	0.1054

SV-Stroke volume, CO- Cardiac output, CI- Cardiac index, EF- Ejection fraction, FS- Fractional fibre shortening, ESS- End systolic stress, EISS-End isovolumetric systolic stress.

The haemodynamic parameters like CO, CI, EF%, FFS%, ESS, and EISS were not severely hampered in the study subjects as observed in the study but there are likely chances that in long standing cases the systolic function may be affected widely.

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 (FFS%) = (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) = $[(Ht (cm) × Wt (Kg) /3600]^{1/2}$, 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)

LV geometry was configured by LVMI/RWT. In Indian males and females remodeling as -118/0.50 and 107/0.47 resp. Normal geometry in males and females as -118/0.50 and 107/0.47 resp. Concentric remodeling in males and females as 118/ >0.50 and 107/ >0.47 resp. Eccentric hypertrophy in males and females as >118/ 0.50 and >107/ 0.47 resp., an Concentric hypertrophy in males and females as >118/ >0.50 and >107/ >0.47) (36,37).

TABLE 5: DISTRIBUTION OF LV GEOMETRICAL PATTERNS IN CONTROLS AND CASES.

PARAMETERS	MALES (n=60)		FEMALES (n=60)	
	CASES (n=30)	CONTROLS (n=30)	CASES (n=30)	CONTROLS (n=30)
NORMAL GEOMETRY	10	25	15	27
CONCENTRIC DELING	10	03	08	01
ECCENTRIC RTROPHY	05	01	04	01
CONCENTRIC RTROPHY	05	01	03	01

Left ventricular hypertrophy procured in both the groups and both the sexes were observed that in females the cases had better outcome than males where females had less number of concentric and eccentric hypertrophy. On the other hand females also had better outcome in terms of normal geometry cases the number was more.

DISCUSSION

Raised blood pressure and Hyperdynamic circulation hypertension remains a challenging task in early identification and detection because most of the patient are asymptomatic. Therefore our cause of concern remains to detect the sustained increased arterial blood pressure in early times through various modalities of investigation chiefly echocardiography. Early remodeling changes in LV which can be procured in the form of left ventricular hypertrophy. Various causes have been implicated in the procurement of LVH but the present study revealed a great role of hyperdynamic circulation, and sympathetic overstimulation. But the other factors like, diabetes mellitus,

hyperthyroidism, sedentary lifestyle, obesity and dietary changes chronic stress are also implicated in the causation of increased peripheral resistance as a result of vascular smooth muscle constriction and raised blood pressure. Metabolic syndrome and obesity are widely prevalent in todays times giving rise to increased blood pressure (28).

Hypertension is one of the major attributable and modifiable risk factor for the procurement of coronary heart disease (CAD), and congestive heart failure (CHF). This study revealed about the functional and structural outcomes as a result of sustained elevated blood pressure (29,30,31).

Present study also denoted that Echocardiographic LV structural parameters LVIDd, LVIDs, were not significantly increased but slightly altered because it may be dependent on the duration of the illness and prevailing compensatory mechanisms operating. The other parameters like IVST, PWT, RWT, LVM, LVMI were noted to be highly significant because the compensatory changes have occurred leading to such remodeling responses as a result of raised blood pressure implicating myocardial fibre lenghthening. Changes are also observed in the LVM, LVMI, RWT which demonstrates a strong correlation with the elevated blood pressure which was observed by several studies in the past (32) (Table2).

Increase in the arterial blood pressure leads to myocyte hypertrophy and the impact is more on the left side of the heart as compared to the right side preferably in the LV. Such findings indicated that the LV dynamics and geometry consequently caused the diminution of LV contractile performance along with hypertrophy (33) and thereby hinting for higher CVS morbidity and mortality in future (31). ESS, EISS and TPR changes also observed to be altered in hyperdynamic circulation which are the indicators of diastolic performance increased in females and were highly significant ($p<0.001$). But in males, only TPR was significantly increased ($p<0.001$) as a result of hyperdynamic circulation and increased afterload,

LVM was highly significant ($p <0.001$) in post menopausal age groups It may be due to the post menopausal changes leading to decrease in the estrogen hormone level and subsequent elevation of blood pressure and increase in BMI.

Concentric hypertrophy has been suggested to be associated with a higher risk of adverse cardiovascular events (34). This pattern of remodeling ultimately progresses to left ventricular dilatation and failure in hypertensives (35). The possible factors which are involved in this structural change are increased BP, increased renin-angiotensin-aldosterone and increased adrenergic activation in subjects who have higher BMI (31). In addition, increased left ventricular

filling due to volume or elevated venous return is responsible for increase in stroke volume and maintain the normal systolic function

These adaptation in the left ventricle will lead to variety of changes which includes geometrical reorientation in different planes producing concentric remodeling, concentric hypertrophy and eccentric hypertrophy.

CONCLUSION

Although there are several alterations in the LV geometry and function in hypertension but remember that these changes are revertible changes which can be curtailed by some premonitory steps. These steps could be early detection, prevention and management of LVH by means of echocardiography and which could substantially decrease the morbidity and mortality relating to the early heart failure.

REFERENCES

1. Buckaleu V, Gruber K. Natriuretic hormone. Annu Rev Physiol.1984; 46:343-358.
2. Chockalingham, A. Recommendations of the Canadian consensus conference on non-pharmacological approaches to the management of highblood pressure, Mar21-23,1989, Halifax, Nova Scotia. Can Med Assoc.J. 1990; 142:1397-1409.
3. Strasser, T. Non-pharmacological treatment. J.Hum.Hypertens.1990; 4 (supplement 1):39-42.
4. Fodor, J.G. and A. Chockalingham. The Canadian consensus report on non-Pharmacological approached to the management of high blood pressure. Clin Exp Hypertens. 1990; A 12(5):729-743.
5. World Health Organization, Arterial hypertension. Report of a WHO expert committee. World Health Organization Technical Report Series No.628. 1978; 1-61.

6. Horan, M. and C. Lenfant. Epidemiology of blood pressure and predictors of hypertension. Hypertension. 1990; 15(suppl.1):20-24.
7. Whitcomb, B. and R. Byyny. Perspective on hypertension in the the elderly. West.J.Med.1990; 512:392- 400.
8. Williams L, Lowenthal D. Hypertension in the elderly. Cardiovascular Clin.1992; 22(2):49-61.
9. Nachtigall, L. Protecting older women from their growing risk of cardiac disease. Geriatrics.1990; 45(5):24- 34.
10. Sannerstedt, R. and Skinner, J. Hypertension. In: Exercise testing and exercise prescription for special cases. Philadelphia, PA: Lea and Febiger. 1987, pp.225-240.
11. Blumenthal, J., W. Seigel and M. Appelbaum. Failure of exercise to reduce blood pressure in patients with mild hypertension. JAMA.1990; 266(15):2098-2104.
12. Naugueh M.D, Middleton K.J, Kopelen et al. Doppler tissue imaging: A noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures, Journal of the American College of Cardiology. 1997; 30(6):1527-33.
13. Sung JK, Kim JY. Obesity and Preclinical Changes of Cardiac Geometry and Function. *Korean Circ J.* 2010;40: 55-61
14. Egan, B. and R. Schmouder. The importance of hemodynamic considerations in essential hypertension. Am Heart J. 1988;116:594-599.
15. Skelton TN, Andrew ME, Arnett DK, et al. Echocardiographic left ventricular mass in African-Americans: the Jackson cohort of the Atherosclerosis Risk in Communities Study. *Echocardiography.* 2003; 20: 111-20.
16. Devereux RB, Roman MJ, Paranicas M, et al. Impact of diabetes on cardiac structure and function: the strong heart study. *Circulation.* 2000; 101: 2271-76.
17. Lund-Johansen, P. The hemodynamics of the aging cardiovascular system. J.Cardiovasc.Pharmacol.1988; 12(suppl.8):S20-S30.
18. E. Braunwald. Heart disease. A textbook of cardiovascular medicine .W.B. Saunders Co.1992. pp. 857.
19. Manolio TA, Levy D. Relation of alcohol intake to left ventricular mass: The Framingham Study. *J Am Coll Cardiol.* 1991; 17:717-21.
20. Ilercil A, Devereux RB, Roman MJ, et al. Relationship of impaired glucose tolerance to left ventricular structure and function: The Strong Heart Study. *Am Heart J.* 2001; 141: 992-8.

21. Ilercil A, Devereux RB, Roman MJ, et al. Associations of insulin levels with left ventricular structure and function in American Indians: the strong heart study. *Diabetes*. 2002; 51: 1543-47.
22. Bella JN, Devereux RB, Roman MJ, et al. Relations of left ventricular mass to fat free and adipose body mass: the strong heart study. The Strong Heart Study Investigators. *Circulation.* 1998;98: 2538-44
23. Lips DJ, deWindt LJ, van Kraaij DJ, Doevendans PA. Molecular determinants of myocardial hypertrophy and failure: alternative pathways for beneficial and maladaptive hypertrophy. *Eur Heart J.* 2003;24: 883-96.
24. Vakili B, Okin P, Devereux RB. Prognostic implications of left ventricular hypertrophy. Am Heart J. 2001:141;334-341.
25. Silangei LK, Maro VP, Diefenthal H, et al. Assessment of left ventricular geometrical patterns and function among hypertensive patients at a tertiary hospital, Northern Tanzania. *BMC Cardiovascular Disorders.* 2012; 12:109
26. Sahan D. J, De Maria A, Kisslo J, Weyman A. Recommendations regarding quantitatation in M-mode echocardiography:results of a survey of echocardiographic measurements. Circulation 1978;58:1072-1083.
27. Zoccali C, Benedetto FA, Mallamaci F, et al. Prognostic impact of the indexation of left ventricular mass in patients undergoing dialysis. *J Am Soc Nephrol.* 2001; 12:2768-74.
28. Misra A, Misra R, Wijesuriya M, Banerjee D. The metabolic syndrome in South Asians: Continuing escalation and possible solutions. *Indian J Med Res.* 2007;125: 345-54
29. Chiang BN, Perlman LV, Epstein FH. Overweight and hypertension: A review. *Circulation*. 1969; 39(3):403-21
30. Staessen J, Fagard R, Amery A. The relationship between body weight and blood pressure. *Journal of Human Hypertension.* 1988; 2: 207-17
31. Kathrotia R. et al. Impact of different grades of body mass index on left ventricular structure and function. Indian J Physiol Pharmacol 1010;54(2):149-156.
32. Grandi AM, Zanzi P, Piantanida E, et al. Obesity and left ventricular diastolic function: noninvasive study in normotensives and newly diagnosed never-treated hypertensives. *International Journal of Obesity.* 2000; 24:954-58
33. Pandey AK, Das A, Kumar A, Babu M, Bhawan G, Himabindu. Myocardial performance in asymptomatic essential hypertension. Indian J Physiol and Pharmacol. 2011; 55 (2); 110- 18.

34. Ghali JK, Liao Y, Cooper RS. Influence of left ventricular geometric patterns on prognosis in patients with or without coronary artery disease. *J Am Coll Cardiol*. 1998; 31(7): 1635-40.
35. Navtej S Chahel, Tiong K. Lim, Piyush Jin et al. New insights into the relationship of left ventricular geometry and left ventricular mass with cardiac function: a population study of hypertensive subjects. Europian Heart Journal. 2010;31:588-594.

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