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Original Article

Physiology Section

Asymptomatic Obese Hypertensives and Need of Routine Echocardiography for Left Ventricular Mass Assessment and Treatment

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ABSTRACT

Background: Echocardiographic determination of Left Ventricle Mass (LVM) – an important marker of cardiovascular disease, has been given a lot of importance in clinical diagnosis and in planning of treatment. Clinically asymptomatic compensated hypertensives show some pathological findings which are indicative of left ventricular dysfunction.

Methods: The study population of 106 males, after a detailed clinical examination, were evaluated by echocardiography and were classified as per the body mass index classification of WHO Western Pacific Region in 2000 for Asian population. Fasting blood samples were taken to estimate blood sugar and lipid profile.

Results: It was observed that subjects in normal range of body mass index <45 years (23.68%) and >45 years (16.1%), subjects of overweight <45 years (15.7%) and >45 years (10.29%) and obese I and II<45 years (60.52%) and >45 years (73.52%). The comparison between left ventricular mass which was indexed to height$^{2.7}$ in subjects who were <45 years and >45years was observed to be statistically significant ($p<0.03$). On comparing LVM/ht$^{2.7}$ of normal BMI group with that of those with higher BMIs, it was noted to be significantly different ($p<0.009$), which was suggestive of adverse effects of increasing BMI on LVM. It was also observed that persons with increased BMIs showed changes in left ventricular geometry – 30.13% had concentric hypertrophy, 17.80% had concentric remodeling, 8.21% had eccentric hypertrophy and that 38.35% had normal left ventricle geometry.

Conclusion: The present study therefore, indicated that it was better to do an echocardiographic screening of asymptomatic subjects who had even a marginal increase in blood pressure and BMI, to diagnose potential cardiac dysfunction.

Key words: Obesity, Hypertension, Echocardiography, Left ventricular mass, Left ventricular geometry

INTRODUCTION

Left Ventricle Hypertrophy (LVH) – an important marker of cardiovascular disease, either potential or in an established condition, has been given a lot of importance in clinical diagnosis and in planning of treatment [1]. Increase in Left Ventricular Mass (LVM) might be physiological or pathological. Several factors which are associated with increased LVM have been identified, which include age, gender, blood pressure, body size, physical activity and blood viscosity [2]. LVM progressively increases during aging [3], which is reported in both normotensives and hypertensives. The age associated LVM increment may be attributed to the physiological increase in body size and blood pressure [4] or to pathological hypertrophic changes which are caused by an increased overload. However, neuro-humoral and genetic factors have also been implicated [5]. Obesity and Hypertension (HT) which are associated with diabetes have been implicated as important determinants of LVM in most of the population based studies [6,7]. Impaired glucose tolerance [8], hyperinsulinaemia, insulin resistance [9] and microalbuminuria also show a stronger association with concentric remodeling and hypertrophy. The other risk factors like smoking [2], extra salt intake [10] and consumption of alcohol [2] also have roles, though factors like increased lipids [11], haematocrit and resting heart rate are all important determinants of increased LVM.

Obesity is an independent factor which has been implicated for LVH, along with minor reversible cardiovascular changes such as hyperdynamic circulation and subclinical morphological changes like a greater aortic root, left atrial enlargement, etc. [12] Alterations in left ventricular diastolic function were found to be more frequent, with increase in obesity, though systolic function was affected late in obese [13].

It has also been reported that clinically asymptomatic compensated hypertensives show stroke volume and cardiac output which are within normal range; still, some pathological findings have been observed, which have indicated LV dysfunction like ejection fraction, fractional fibre shortening, LV end diastolic dimensions, etc. M-mode echocardiography is a highly sensitive and a specific method which provides accurate assessment of LVH [14]. Hence, it has taken an important place in clinical medicine for identification of cardiac morphology and dynamics. Therefore, the present study was undertaken to estimate LVM by echocardiography, especially to identify subjects with asymptomatic hypertension, as it is a potentially modifiable cardiovascular risk factor [15] which can be corrected by measures like life style, exercise, balance between work and rest, including early medication.

MATERIAL AND METHODS

The present study was conducted at SRMSIMS Hospital, Bareilly, Uttar Pradesh state, India. The study was approved by institutional ethics committee and informed written consents were obtained from subjects.

Study Material: 106 males were enrolled for comprehensive health checkups, whose ages ranged from 27-75 years. A detailed medical history which included a history of hypertension (HT), diabetes mellitus, smoking, alcohol consumption, physical activity and family history was taken. Clinical examinations were then carried out to record their heights, weights, Blood Pressure (BP) measurements- Systolic Blood Pressure (SBP) Diastolic Blood Pressure (DBP) and resting heart rates.

Study design: Subjects were divided into four groups – normal BMI (18.5-22.9 kg/m^2), overweight (23-24.9 kg/m^2), obese I (25-29.9 kg/m^2) and obese II (≥30 kg/m^2) as per the recommendations of WHO Western Pacific Region 2000 [16]. Further, subjects were divided into two age groups- <45 years and >45 years.

Inclusion criteria: All male subjects who enrolled for the comprehensive health check ups after fulfilling exclusion criteria were recruited in the study.

Exclusion criteria: Those with any history of recent surgeries, uncontrolled diabetes mellitus, congenital heart disease, rheumatic heart disease, unstable and 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: WHO defines hypertension as a chronic medical condition in which the SBP is ≥ 140 mmHg and/or DBP is ≥90 mmHg on two readings which are taken apart or a reported diagnosis of HT and treatment with recognized anti- hypertensive within 2 weeks before the visit [17]. Dyslipidaemia was defined according to NCEP, ATP III guidelines [18]. Type-2 Diabetes mellitus was diagnosed according to the American Diabetes Association (ADA) criteria [19].

Fasting blood samples were taken for biochemical estimation of glucose and lipid profile. Two dimensional M-mode echocardiograms (Siemens Acuson P300) of all participants were obtained by trained cardiologists. Left ventricular dimensions were obtained in parasternal short axis view, with measurement of interventricular septal thickness in diastole (IVSTd), LV dimension in End diastole (LVDd), LV dimension in systole (LVDs) and LV posterior wall thickness in diastole (LVPWTd) according to guidelines of American Society of Echocardiography (ASE) [20].

Echocardiographic Measurements

Body surface area (BSA) [21]

$$BSA = 0.6 \times \text{height (m)} + 0.0128 \times \text{weight (kg)} - 0.1529$$

Left Ventricular Mass (LVM): Devereux's modified American Society of Echocardiography (ASE) cube equation [22]

$$LVM = 0.8 \times [1.04 \times (LVDd + LVPWTd + IVSTd)^3 - LVDd^3] + 0.6 \text{ g}$$

Left Ventricular Mass Index (LVMI): LVM divided by body surface area (LVM/BSA, g/m^2). Since this index could fail in identifying left ventricular hypertrophy in obese individuals, a second index was calculated by height (LVM/ht, g/m) or height$^{2.7}$ (LVM/ht$^{2.7}$, g/m$^{2.7}$) [23].

Relative wall thickness (RWT)

RWT = (IVSTd + LVPWTd)/LVDd). LVMI / RWT was used to identify the left ventricle geometry patterns, considering normal value [24] for Indian Asian males—118 g/m /0.50. The subjects were categorized as having: (i) Normal Geometry (NG) – normal RWT and LVMI; (ii) Concentric Remodeling (CR) – increased RWT and normal LVMI; (iii) Eccentric Hypertrophy (EH) – normal RWT and increased LVMI; and (iv) Concentric Hypertrophy (CH)-increased LVMI and RWT.

The data were analyzed as per BMI groups by using Microsoft Excel 2010 software. Mean ± SD were calculated. Unpaired student's T-test was applied. Pearson's correlation coefficient (r) was obtained to study correlation of BMI with other variables. A 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

Among 106 subjects who were under study, we found normal BMI in 20/106 with an age wise distribution of < 45 years (23.68%) and > 45 years (16.1%); overweight (OW) in 13/106 with an age wise distribution of <45 years (15.7%) and >45 years (10.29%); obese I (OB-I) 53/106 with an age wise distribution of < 45 years (39.47%) and >45 years (55.88%) and obese II (OB-II) 20/106 with an age wise distribution of <45 yes (21.05%) and >45 years (17.64%). 47.94% obese, 23% overweight and 40% normal BMI subjects were hypertensives [Table/Fig-1].

[Table/Fig-2] shows echocardiographic parameters in various groups as per BMI. A correlation of BMI with other variables was analyzed [Table/Fig-3]. BMI showed a positive correlation for SBP (r 0.28) and DBP (r 0.25). Also, positive correlations were found for IVSD (r 0.28), LVPWTd (r 0.27), Left atrial diameter (LAD) (r 0.24), aortic root diameter (r 0.17) and LVMI (r 0.36).

[Table/Fig-4] shows that obese subjects had significantly higher SBPs, DBPs and BMIs as compared to those in normal BMI subjects. Also noted were significantly higher LVPWTds, aortic root diameters, LADs, LVMs and LVMs indexed to heights$^{2.7}$ in obese subjects in comparison with those in normal BMI subjects.

	Normal BMI		Overweight		Obese I		Obese II	
	< 45 years (n 9)	> 45 years (n 11)	<45 years (n 6)	>45 years (n 7)	<45 years (n 15)	>45 years (n 38)	<45 years (n 8)	>45 years (n 12)
Age (years)	36.67±5.66	59.27±11.26	39.00±4.29	58.43±5.09	36.80±5.75	54.92±7.07	38.50±2.67	56.00±6.66
Height (m)	1.73±0.06	1.67±0.05	1.70±0.05	1.70±0.08	1.70±0.05	1.68±0.06	1.68±0.08	1.66±0.08
Weight (kg)	63.11±8.22	59.27±5.97	70.17±5.15	70.86±6.04	78.67±8.09	77.13±6.24	91.5±8.85	91.92±14.22
BMI (kg/m^2)	20.99±1.48	21.17±1.49	24.23±0.42	24.1±0.71	27.28±1.46	27.40±1.37	32.28±1.58	33.12±4.04
SBP(mmHg)	124.89±14.84	126.91±13.90	125±13.78	128.29±8.98	127.33±8.30	135.84±14.74	132.5±16.69	143.83±14.33
DBP(mmHg)	81.11±7.42	79.45±7.95	83.33±8.16	85.14±6.72	86.13±7.27	88.42±9.88	83.75±9.16	89.50±9.11
HR (beats/min)	76.44±4.33	80.73±8.55	79.00±5.90	83.86±2.34	80.4±4.29	80.16±4.99	80.75±4.40	79.5±5.98
Hypertension (n)	4 (44.4%)	4 (36.3%)	1 (16.6%)	2 (28.57%)	4 (26.6%)	18 (47.36%)	2 (25%)	11 (91.6%)
Diabetes (n)	1	2	-	1	4	13	2	1
Exercise (n)	5	6	4	5	5	20	5	11
Family history (n)	7	6	4	4	12	28	7	9
Smoker (n)	6	3	3	3	3	10	3	4
Alcoholic (n)	5	2	3	3	5	17	4	5
Serum cholesterol (mg/dl)	199.22±34.34	174.45±49.44	176.5±29.55	166.43±29	198.93±40.47	191.42±42.93	200.63±67.71	191.42±44.39
Serum triglyceride (mg/dl)	108.56±56.66	108.09±63.57	197.5±93.57	124.71±79.54	167.53±106.90	173.42±129.89	127.88±52.08	142.33±41.11
HDL(mg/dl)	48.44±11.31	44.64±8.52	44±5.51	45.71±9.95	38.87±8.27	44.32±8.72	41.25±6.68	42.17±10.23
LDL(mg/dl)	124.6±26.95	108.2±48.05	92.87±25.05	95.97±26.87	127.09±40.83	113.69±33.2	133.73±65.99	120.9±36.4
VLDL(mg/dl)	21.71±11.33	21.62±12.71	39.5±18.71	24.94±15.91	32.97±20.95	29.58±12.27	26.33±10.32	28.35±8.21
FBS(mg/dl)	94.22±5.83	103.27±21.9	101.83±11.81	124±65.39	130±60.14	127.87±53.83	107.13±20.52	117.08±45.96

[Table/Fig-1]: Showing baseline parameters (mean±SD) in males (n 106) according to BMI classification

IVSD- interventricular septal wall thickness at end diastole, LVDD- left ventricular diameter at end diastole, LVDS- left ventricular diameter at end systole, LVPWD- left ventricular posterior wall thickness at end diastole, LVEF- left ventricular ejection fraction, LVM- left ventricular mass

	Normal BMI		Overweight		Obese I		Obese II	
	< 45 years (n 9)	> 45 years (n 11)	<45 years (n 6)	>45 years (n 7)	<45 years (n 15)	>45 years (n 38)	<45 years (n 8)	>45 years (n 12)
LVDd (mm)	42.33±3.84	43.45±4.06	43±3.85	40.57±4.72	44.53±3.94	43.71±3.35	42.5±5.07	44.33±3.39
LVDs (mm)	26.67±4.95	27.18±4.26	27.17±2.32	25.29±2.43	27.67±2.82	25.92±3.31	27±2.67	26.67±2.39
LVPWTd (mm)	10.67±1.73	10.73±1.35	10.67±1.63	10.71±1.98	10.97±0.93	11.16±1.58	11.5±2.07	12.79±2.37
LVEF (%)	63.89±0.03	61.36±0.04	62.5±0.03	62.14±0.02	63.73±0.05	62.36±0.02	63.38±0.02	62.5±0.03
Aortic root diameter (mm)	23.89±4.11	25.09±5.22	23±6.03	24.71±4.11	26±4.97	27.22±4.02	25.63±3.16	27.33±4.14
Left Atrial diameter (mm)	27.11±3.30	30.18±2.64	30.33±3.83	32.57±4.31	30.8±4.18	31.57±3.61	32.88±2.85	31.25±2.49
LVM (g)	155.89±34.50	162.48±31.39	166.18±31.20	148.26±35.46	172.59±35.39	177.34±42.76	178.92±50.21	215.91±63.68
LVM/BSA (g/m^2)	92.22±19.68	101.46±22.11	93.85±15.76	83.21±19.31	92.11±17.26	96.26±21.88	89.51±30.83	107.65±32.73
LVM/HT2.7 (g/m$^{2.7}$)	35.45±7.52	40.63±8.09	39.47±6.36	34.69±8.23	41.32±7.57	43.88±10.0	44.62±16.01	55.52±18.41
RWT	0.51±0.09	0.50±0.07	0.51±0.05	0.53±0.07	0.49±0.05	0.52±0.06	0.55±0.09	0.57±0.12

[Table/Fig-2]: Echocardiographic parameters (mean± SD) according to BMI classification in males (n=106)

IVSD- interventricular septal wall thickness at end diastole, LVDd- left ventricular diameter at end diastole, LVDs- left ventricular diameter at end systole, LVPWTd- left ventricular posterior wall thickness at end diastole, LVEF- left ventricular ejection fraction, LVM- left ventricular mass, LVM/BSA- left ventricular mass indexed to body surface area, LVM/Ht$^{2.7}$- left ventricular mass indexed to height$^{2.7}$, RWT- relative wall thickness

	r value
SBP	0.28
DBP	0.25
Total cholesterol	0.09
Triglycerides	0.05
HDL	-0.118
LDL	0.12
VLDL	0.09
IVSD	0.28
LVDD	0.14
LVDS	0.09
LVPWTd	0.27
Aortic root diameter	0.17
LAD	0.24
LVM/BSA	0.03
LVM/HT$^{2.7}$	0.36
RWT	0.15

[Table/Fig-3]: Showing coefficient of correlation (r) between BMI and other variables

Further, when subjects were divided on the basis of age but not according to BMI, it was observed that <45 years gr. had significantly lower LVM/ht$^{2.7}$ (p<0.03) in comparison to >45years gr. (not shown in table). When the subjects were divided into two groups based on SBP of <140 mmHg and above and DBP of <90 mmHg and above, it was observed that SBP of >140 mmHg and DBP of >90 mmHg groups had significantly (SBP p<0.001; DBP p<0.01) higher LVM/ht$^{2.7}$ values. When subjects were divided into smokers (n 35) and non-smokers (n 71) and not considering other risk factors like age, BMI and BP, no significant difference (p<0.11) was noted in LVM/Ht$^{2.7}$. When subjects were divided into alcoholic (n 44) and non-alcoholic (n 62) groups, no significant difference (p<0.33) was noted in LVM/Ht$^{2.7}$.

Parameter	Normal Bmi (n 20)	Overweight (n13)	Obese (n 73)	p Value
AGE	49.1±17.8	49.4±11.05	49.57±10.4	NS
BMI	21.09±4.81	24.16±0.57	28.85±3.17	
SBP	126±30.68	126.76±11.06	135.04±14.48**	**p<0.007
DBP	80.2±18.99	84.30±7.15	87.61±9.18**	**p<0.0006
HR	78.8±18.55	81.61±4.85	80.16±4.88	NS
IVSTd	10.87±2.72	11.23±1.09	11.63±1.78	NS
LVDd	42.95±10.11	41.69±4.34	43.84±3.66	NS
LVDs	26.95±7.31	26.15±2.47	26.52±3.03	NS
LVPWTd	10.7±2.74	10.69±1.75	11.42±1.76**	**p<0.04
LVEF	62.5±0.14	62.3±0.02	62.7±0.03	NS
Aorta root diameter	24.55±7.03	23.92±4.94	26.80±4.14**	**p<0.01
Left atrial diameter	28.8±7.04	31.53±4.09*	31.5±3.48**	*p<0.02 ** p<0.001
LVM	159.51±46.80	156.53±33.47	182.87±47.64**	**p<0.02
LVM/BSA	97.30±29.52	88.12±17.90	96.53±24.29	
LVM/ht2.7	38.30±11.48	36.89±7.54	45.34±12.69**	**p<0.01
RWT	0.50±0.14	0.51±0.10	0.52±0.08	NS

[Table/Fig-4]: Analysis of various parameters among sub-groups of BMI

*On comparing normal BMI with overweight, ** on comparing normal BMI with obese, p< 0.05 significant, p<0.01 very significant, p<0.001 highly significant, NS not significant

[Table/Fig-5] shows that among obese subjects; 30.13% had concentric hypertrophy, 17.80% had concentric remodeling, 8.21% had eccentric hypertrophy and that 38.35% had normal left ventricle geometry.

[Table/Fig-6] shows analysis of various parameters in obese subjects when hypertension was taken into consideration. OB uncontrolled HT had significantly higher ages, SBPs and DBPs as compared to obese normotensives. This group also showed significantly higher

Numbers	Normal BMI		Overweight		Obese I		Obese II	
	<45 years (n 9)	> 45 years (n 11)	<45 years (n 6)	>45 years (n 7)	<45 years (n-15)	>45 years (n-38)	<45 years (n-8)	>45 years (n-12)
Normal Geometry	66.6 (n 6)	45.4 (n 5)	33.3 (n 2)	38.9(n 3)	53.3 (n 8)	39.4 (n 15)	25 (n 2)	25 (n 3)
Concentric Remodelling	22.2 (n 2)	45.4 (n 5)	66.6(n 4)	57.1 (n 4)	20 (n 3)	10.5 (n 4)	50 (n 4)	16.6 (n 2)
Eccentric Hypertrophy	_	9.09 (n 1)	_	_	13.3 (n 2)	5.2(n2)	12.5 (n 1)	8.3 (n 1)
Concentric Hypertrpohy	11.1 (n 1)	_	_	_	13.3 (n 2)	34.2 (n 13)	12.5 (n 1)	50 (n 6)

[Table/Fig-4]: Left ventricle geometrical patterns

Values shown are as % age of subjects

Parameter	Obese Non HT (n 17)	Obese HT First Time Noted (n 21)	Obese HT Controlled on Treatment (n 10)	Obese HT Uncontrolled on Treatment (n 25)	p value
AGE	47.29±10.48	46.42±12.37	49.20±8.31	53.92±8.28***	***≤0.02
BMI	28.34±2.45	28.38±2.37	29.20±2.89	29.44±4.19	NS
SBP	121.17±5.74	137.42±15.25*	127.00±4.83**	145.68±10.88***	*≤0.0001,**≤0.01, ***HS
DBP	78.47±4.44	91.80±7.97*	79.00±3.16	93.76±6.17***	*HS, ***HS
HR	78.58±5.51	81.61±3.49*	79.40±3.89	80.32±5.61	*≤0.04
IVSTd	11.20±1.38	11.50±1.61	11.10±1.71	12.24±2.09	NS
LVDd	44.58±4.25	44.00±2.82	43.80±3.61	43.24±3.97	NS
LVDs	27.41±4.38	26.61±3.07	26.10±2.23	26.00±2.04	NS
LVPWTd	10.35±1.05	11.54±1.34*	11.25±1.58	12.12±2.19***	*≤0.004,***≤0.007
LVEF	64.00±0.03	63.00±0.04	64.00±0.02	62.00±0.03***	***≤0.02
Aorta root iameter	27.35±4.30	26.35±3.84	27.10±4.77	26.68±4.21	NS
Left atrial diameter	32.47±3.65	31.75±3.66	31.10±3.17	30.80±3.35	NS
LVM	169.90±36.64	182.62±39.15	176.04±58.97	194.64±55.22	NS
LVM/BSA	89.95±18.03	97.09±22.03	91.39±26.10	102.60±28.52	NS
LVM/$ht^{2.7}$	41.34±7.72	45.11±11.60	43.22±14.21	49.11±15.08***	***≤0.05
RWT	0.48±0.04	0.52±0.06	0.50±0.05	0.56±0.10***	***≤0.007
Left ventricle geometry	(NG-8, CR-4, CH-2, EH-3)	(NG-8, CR-6, CH-6, EH-1)	(NG-5, CR-2, CH-2, EH-1)	(NG-6, CR-6, CH-12, EH-1)	

[Table/Fig-6]: Analysis of various parameters among sub-groups of obese and hypertensive

*On comparing obese non HT with obese first time noted HT, ** on comparing obese non HT with obese HT controlled with treatment, ***on comparing obese non HT with obese uncontrolled HTp≤0.05 significant, p≤0.01 very significant, p≤0.001 highly significant

LVPWTds, RWTs and LVMs indexed to $heights^{2.7}$ as compared to obese normotensives. Abnormal left ventricle geometrical patterns were observed in 76% obese uncontrolled HT (19/25), 61% obese first time noted HT (13/21), 50% obese controlled HT (5/10) and 52.94% obese normotensives (9/17).

DISCUSSION

A marked shift in lifestyle has been noted in south Asian countries, which has resulted from urbanization, affluence and an increased intake of high calorie foods, including physical inactivity. This has resulted in an increased trend of obesity which predisposes to ailments like HT and cardiovascular diseases [25]. The present study also indicated increasing adiposity with increasing age in the population which was studied [Table/Fig-1]. However, Kalra S et al., have reported a decreasing trend in obesity with increasing ages of above 33-45 years [26]. A trend of increasing BMI has also been observed in children and adolescents in neighbouring Asian countries [12]. Obesity is a predisposing factor for cardiovascular diseases, which results in subclinical or clinical changes in cardiac morphology and function. These changes can result in life threatening complications like stroke and myocardial infarction.

In the present study, significant increases in SBP and DBP were noted in obese group as compared to those in normal BMI group [Table/Fig-4]. Similar observations were made by other researchers also [27-29]. In addition, obesity is known to produce haemodynamic changes and a neuro-hormonal activation, which causes an increase in blood pressure [12]. Structural changes in heart have been observed, with significantly higher LVPWTds, LADs, aortic root diameters, LVMs and LVMs/$Hts^{2.7}$ in obese subjects as compared to normal BMI subjects [Table/Fig-4]. Similar observations were made by Kathrotia et al., [29]. The mechanisms which have been postulated for LAD increase are volume overload and diastolic dysfunction [12]. The available literature also proves that obesity and hypertension significantly influence LV geometry, with obesity affecting both LV diastolic diameter and wall thickness, whereas hypertension only influences wall thickness [30].

Based on the structural changes in the heart, left ventricular geometric patterns have been defined, which are known to have important prognostic implications. It was also observed in our study, that 31.50% had NG, 17.80% had CR, 8.21% had EH and that 30.13% had CH among obese subjects and that 55% had NG, 35% had CR, 5% had EH and that 5% had CH in normal BMI group. A study from Tanzania has reported that 16.7% had NG, 12.6% had CR, 46.0% had EH and that 24.7% had CH in obese group and that 71.3% had NG, 22.5% had CR, 3.7% had EH and that 2.5% had CH in the control group [31]. Interestingly, such structural alterations in left ventricle geometry have been reported in Asian subjects with high BMI, who reside in UK [32]. Among the mentioned geometric alterations, concentric hypertrophy has been suggested to be associated with a higher risk of adverse cardiovascular events [33]. This pattern of remodeling ultimately progresses to left ventricular dilatation and failure in hypertensives [32]. 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 BMIs [29]. Diastolic dysfunction with normal ejection fraction was identified in 53% overweight subjects and in 35% obese subjects by echocardiography. Recent studies have reported that diastolic dysfunction can be present in absence of overt features of congestive heart failure, further increasing cardiovascular mortality [34]. The early diagnosis of diastolic dysfunction has been facilitated by the availability of non-invasive Doppler echocardiography [35]. Hence, early screening of obese hypertensive population can help in reducing the burden of adverse cardiovascular events.

Further, it was also observed when hypertension co-existed with obesity, there was a significant increase in LVPWTd, RWT and LVM/$Ht^{2.7}$ in obese uncontrolled hypertension group [Table/Fig-6]. It was also noted that a higher no. of obese hypertensive subjects had concentric hypertrophy. On the contrary, Adebiyi et al reported eccentric hypertrophy as commonest LV geometry in their study which was done on hypertensive Nigerians [36]. Observations in obese subjects with hypertension which was controlled with treatment showed values which were similar to those of obese non hypertensive group. The present observations probably indicated that an important role was played by an early initiation of curative treatment and prevention. It has been reported by other researchers that various pharmacologic and non-pharmacologic therapeutic interventions can cause regression of LVH [37-39].

CONCLUSION

LVH is an important predictor of cardiovascular morbidity and mortality in obese and hypertensive patients. Considering the adverse outcomes which are associated with LVH, it becomes essential to diagnose it at an early stage. Among the various diagnostic tools, echocardiography is a relatively simple and a non-invasive test with a good predictive value. Regression of LVH can significantly reduce adverse cardiovascular events. The positive lifestyle modifications such as regular physical exercise, dietary changes and proper control of hypertension with medication can help in regression of LVH. The present study therefore, recommends an echocardiographic screening of obese and hypertensive subjects, to diagnose potential cardiac dysfunction and early institution of treatment.

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