The effect of obesity on left ventricular geometric patterns in obese essential hypertensive patients.

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ABSTRACT

This study was performed in Baghdad Teaching Hospital to study whether obesity adds to the adverse effects of hypertension on the heart, namely on the left ventricular geometric pattern, in obese hypertensive patients compared to non-obese hypertensive patients. One hundred eighty hypertensive patients were selected and grouped into two groups, 144 obese hypertensive (group 1: obese HT) and 36 non-obese hypertensive (group 2: non-obese HT). The two groups showed non-significant differences in respect to the general and echocardiographic characteristics except for the body mass index in kg/m^2 (obese HT vs non-obese HT, 31.68± 0.36 vs 26.06±0.33, *p*= 0.0001) and for the LV mass/height $^{2.7}$ in $gm/m^{2.7}$ (obese HT vs non-obese HT, 93.96±3.79 vs 75.81±9.07, *p*=0.001). Of the obese HT patients, 12(8%) were having normal LV geometric pattern and 132(92%) showed abnormal LV geometric pattern; Of the non-obese HT patients, 9(25%) were having normal LV geometric pattern and 27(75%) showed abnormal LV geometric pattern (*p*= 0.0001 overall). There is percentage difference of 17% in the abnormal patterns in the two groups. This was attributed to the presence of obesity in the obese HT group. This study concluded that in the management of obese hypertensive patients it is very essential that not only treating hypertension per se but to plan for promotion of optimal body weight besides controlling hypertension through life style measures such as adequate exercise and proper nutrition and trying to maintain ideal body weight for the whole life of the patient.

الخلاصة

أجريت هذه الدراسة في وحدة صدى القلب في مستشفى بغداد التعليمي لدراسة هل إن السمنة تضيف للتأثيرات الضارة التي يسببها ارتفاع ضغط الدم على القلب وخصوصاً على الشكل الهندسي للبطين الأيسر. ظهر من الدراسة بأن 92% من المرضى المصابين بارتفاع ضغط الدم مع السمنة لديهم شكل هندسي غير طبيعي للبطين الأيسر مقارنةً بنسبة 75% من المرضى المصابين بارتفاع ضغط الدم لوحده أي بدون السمنة. هذا الفرق بمقدار 17% بين النسبتين يعزى الى وجود السمنة مع ارتفاع ضغط الدم لدى المريض نفسه. من هذه الدراسة نستنتج انه عند معالجة مريض مصاب بارتفاع ضغط الدم مع السمنة يجب التخطيط لمعالجة الحالتين ارتفاع ضغط الدم مع السمنة من خلال العمل على الوصول للوزن المثالي لجسم المريض بواسطة إجراءات تغيير نمط الحياة بلعب التمارين الرياضية المناسبة والتغذية المناسبة أيضاً ومحاولة الاحتفاظ بالوزن المثالي للمريض مدى حياته وبصورة مستدامة.

OBJECTIVES

To examine whether obesity adds to the adverse effects of hypertension on myocardial structure through assessing statistically the differences in LV geometric patterns in obese HT patients compared to non-obese HT patients.

INTRODUCTION

Hypertension and obesity are major independent risk factors for cardiac deaths[1,2] . In hypertensive heart disease there is association of left ventricular hypertrophy, hypokinesis, and sometimes, significant left ventricular dilation[3].

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Obesity has been linked to a spectrum of cardiovascular changes ranging from a hyperdynamic circultation[2], through subclinical cardiac structural changes[4], to overt heart failure[5] . The pathogenesis of these cardiovascular changes in obesity is that obesity is associated with haemodynamic overload[2,6]. The increased metabolic demand imposed by the expanded adipose tissue and augmented fat-free mass in obesity results in a hyperdynamic circulation with increased blood volume i.e., increased left ventricular preload. In addition to the increased left ventricular preload, left ventricular afterload is also elevated in obese individuals due to both increased peripheral resistance and greater conduit artery stiffness[7]. Right ventricular afterload may be increased, presumably due to associated sleep disordered breathing and left ventricular changes[6]. Cardiac output is often higher in obesity due to an augmented stroke volume and an increase in heart rate[2]. Ventricular systolic function as assessed by ejection fraction and fractional shortening is usually normal in obesity[8].

PATIENTS AND METHODS

Study sample

One hundred eighty hypertensive patients(untreated or inadequately treated essential hypertensive patients) with a duration of hypertension ranging from > 1 year to > 10 years, were selected and grouped into two groups, 144 obese hypertensive (group 1: obese HT) and 36 non-obese hypertensive (group 2: non-obese HT). Patients with other cardiac or extracardiac diseases were excluded from the study.

Echocardiographic methods

Imaging was obtained for each patient using a commercial instrument with a mechanical transducer of 2.5- 3.5 MHz, Voluson 530D type, Austrian made, supplied by Kretz Technik Company. Transthoracic 2-D guided M-mode and Doppler echocardiograms were recorded according to the American Society of Echocardiography guidelines[9]. Left ventricular internal dimension and interventricular septal and posterior wall thickness were measured at end-diastole and end-systole on up to 3 cycles.

Blood pressure measurement

Blood pressure (BP) was measured using a standard mercury sphygmomanometer, measurements were obtained while the patient is sitting for at least five minutes with the non-dominant arm at the level of the heart, relaxed and supported. A mean of at least three consecutive readings of systolic (phase I) and diastolic (Phase V) of Korotkoff sounds, at one minute intervals, to identify systolic and diastolic values respectively.

Using systolic blood pressure >140mmHg and/or diastolic blood pressure >90mmHg, the individuals were classified as hypertensive[10].

Calculation of general characteristics

1.Body surface area was calculated as square root of (product of height in cm x weight in kg) divided by square root of 3600[11].

2.Body mass index (Quetelet index, BMI) was calculated as weight in kg / height^2 in m^2 . All patients with a BMI > 30 were grouped as obese and those < 30 were grouped as non-obese[10].

3.Mean BP was calculated as diastolic BP + 1/3 (systolic BP– diastolic BP) in mmHg[12].

Calculation of derived echocardiographic variables

1. Relative wall thickness was expressed as the ratio of 2x posterior wall thickness / LV end-diastolic diameter[13].

2. LV mass was calculated using the Penn convention[14,15] according to the equation:

LV mass = 1.04[(LV end-diastolic diameter in cm + posterior wall thickness in cm + interventricular septum thickness in cm)^3 – (LV end-diastolic diameter in cm)^3] – 14, measured in gm.

3. LV mass was considered as an unadjusted variable and normalized for the appropriate power of its prognostically validated allometric relation to height (height$^{2.7}$)[16]. LV mass / height$^{2.7}$ values of 49.2 gm/m$^{2.7}$ in men and 46.7gm/m$^{2.7}$ in women were used as upper limits of gender- specific normal 95% confidence intervals as shown in the figure[13].

4. LV mass was also normalized for body surface area giving LV mass index (LVMI). Normal LVMI in women is < 110 gm/m^2 , while in men LVMI is < 134 gm/m^2.[17]

5. Left ventricular systolic function was assessed by measurement of:

a. LV fractional shortening [(end-diastolic – end-systolic diameter / end-diastolic diameter) x 100], normal value is 30-45%[18].

b. LV ejection fraction was estimated as [(LV end-diastolic volume in ml – LV end-systolic volume in ml / LV end-diastolic volume in ml) x 100], normal value is 50 - 85%.[11]

Left ventricular geometric patterns

LV geometry (The figure) was deduced depending on correlating the relative wall thickness with the LV mass/height$^{2.7}$. Increased relative wall thickness (>0.43) represents the 97.5th percentile in previously described normal subjects[19,20]. These geometric patterns are as follow:

a.Normal geometry was present when LV mass /height$^{2.7}$ and relative wall thickness were both normal.

b.Concentric remodeling was present when there was normal LV mass/height$^{2.7}$ with increased relative wall thickness.

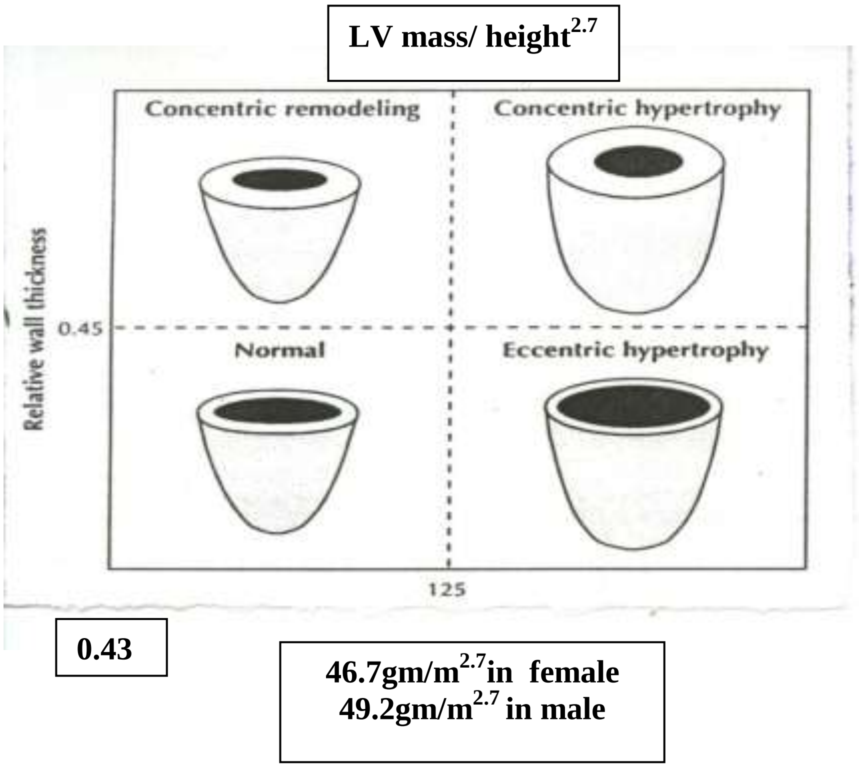
c.Eccentric LV hypertrophy was present when there was high LV mass / height$^{2.7}$ with normal relative wall thickness.

d.Concentric LVH was present when there was an increase in both LV mass/ height$^{2.7}$ and relative wall thickness[21].

Data handling and statistical analyses

Data were presented as mean ± standard error of the mean (SEM) for continuous variables or as numbers with percentages for categorical variables. The unpaired Student's t test was used to compare means of two independent variables. The chi-square test was used to assess differences among proportions of categorical data. A 2-tailed *p* value < 0.05 was considered statistically significant[22].

All calculations were performed by software package statistical system (SPSS version 12 for windows, SPSS Inc., Chicago, Illinois).



The figure: Normal and abnormal LV geometric patterns[13].

RESULTS

Table 1 shows the general and echocardiographic characteristics of the two groups included in the study. This table shows the sex of patients in each group, age(year), body mass index(gm/m^2), resting heart rate(beat/minute) , systolic, diastolic, mean blood pressure (mmHg), interventricular wall thickness in diastole and in systole, LV posterior wall thickness in diastole and in systole, LV internal dimension in diastole and in systole(all in mm), LV end-diastolic volume, LV end-systolic volume, LV stroke volume
(all in ml), LV fractional shortening during systole %, LV ejection fraction %, LV mass index (gm/m^2), LV mass/height$^{2.7}$(gm/m$^{2.7}$) , and relative wall thickness.

All the above-mentioned parameters show non-significant differences between the means of the two groups except for the body mass index in kg/m^2 (p=0.0001), and for the LV mass/height$^{2.7}$ in gm/m$^{2.7}$ (p=0.001).

Table 2 shows the numbers and percentages of the normal and abnormal geometric patterns in each of the two groups.. The obese HT (group 1) shows 8% normal LV geometric pattern and 92% abnormal LV geometric patterns. The non-obese HT (group 2) shows 25% normal LV geometric pattern and 75% abnormal LV geometric patterns There is additional 17% abnormal LV geometric patterns in the obese HT group different from the non-obese HT group (p=0.0001).

Table 3 shows the numbers and percentages of each category of the abnormal LV geometric patterns in both groups. The obese HT group showed 132 (92%) with abnormal LV geometric patterns, distributed as follows, 9(7%) showed concentric remodeling, 18(54%) showed eccentric hypertrophy, and 69(52%) showed concentric hypertrophy. The non-obese HT group showed 27 (75%) with abnormal LV geometric patterns, distributed as follows, 3(11%) showed concentric remodeling, 9(33%) showed eccentric hypertrophy, and 15(56%) showed concentric hypertrophy (p=0.0001). In case of concentric remodeling, obeseHT vs non-obese HT is 7% vs 11%; in case of eccentric hypertrophy, obese HT vs non-obeseHT is 54% vs 33%; in case of concentric hypertrophy obese HT vs non-obese HT is 52% vs 56%.

Table 1: General and echocardiographic characteristics of the two groups included in the study.

Dependent variable	Obese HT (n=144) (mean±SEM)	Non-obese HT (n=36) (mean±SEM)	*P* value*
Gender (male:female)	96:48	21:15	-
Age (year)	60.81±1.31	64.83±4.00	NS
Body mass index (kg/m^2)	31.68±0.36	26.06±0.33	0.0001
Resting heart rate (beat/minute)	77.75±0.63	78.33±1.12	NS
Systolic BP (mmHg)	188.12±2.96	186.25±7.57	NS
Diastolic BP (mmHg)	114.17±1.53	111.25±3.59	NS
Mean BP (mmHg)	139.24±1.85	137.91±4.35	NS
Interventricular septal thickness in diastole (mm)	12.28±0.26	11.47±0.66	NS
Intrventricular septal thickness in systole(mm)	17.44±0.41	16.22±0.99	NS
LV posterior wall thickness in diastole(mm)	12.49±0.27	11.60±0.67	NS
LV posterior wall thickness in systole (mm)	17.70±0.41	16.62±1.02	NS
LV internal dimension in diastole(mm)	57.96±0.76	55.73±2.09	NS
LV internal dimension in systole(mm)	34.02±0.47	32.65±1.09	NS
LV end-diastolic volume (ml)	135.64±1.89	132.50±3.49	NS
LV end-systolic volume (ml)	57.41±1.20	56.72±2.69	NS
Stroke volume(ml)	78.23±1.04	75.77±1.27	NS
LV fractional shortening%	41.04±0.34	41.10±0.51	NS
LV ejection fraction%	57.68±0.47	57.36±1.02	NS
LV mass index (gm/m^2)	193.78±7.87	179.04±22.19	NS
LV mass/height $m^{2.7}$	93.96±3.79	75.81±9.09	0.001
Relative wall thickness	0.43±0.01	0.41±0.01	NS

* Unpaired Student's t test to compare means of continuous data of two independent variables[22].

N: number, mm: millimeter, ml: milliliter, NS: non-significant, gr: gram, m: meter, LV: left ventricle, *p*: probability value.

Table 2: Numbers and percentages of the normal and abnormal LV geometric patterns in the two groups included in the study.

Independent variable	Dependent variable Left ventricular geometric pattern		*P* value*
	Normal (n=21)	Abnormal (n=159)	
Obese hypertensive (n=144)	12(8%)	132(92%)	0.0001
Non-obese hypertensive (n=36)	9(25%)	27(75%)	
Abnormal % difference in presence of obesity		+17%	

* Chi square as non-parametric test to compare proportions of categorical data[22].

Table 3: Numbers and percentages of each category of the abnormal LV geometric patterns in the two groups included in the study.

Independent variable	Dependent variable Abnormal LV geometric pattern (n=159)			*P* value*
	Concentric remodeling (n=12)	Eccentric hypertrophy (n=63)	Concentric hypertrophy (n=84)	
Obese hypertensive (n=132)	9(7%)	54(41%)	69(52%)	0.0001
Non-obese hypertensive (n=27)	3(11%)	9(33%)	15(56%)	

* Chi square as non-parametric test to compare proportions of categorical data[22].

DISCUSSION

As far as the left ventricular geometric pattern is deduced depending on two cardiac structural parameters ,namely, the first is the relative wall thickness which showed non-significant difference between the two groups of patients, while, the second parameter is the LV mass/height$^{2.7}$ (gm/m$^{2.7}$) which showed significant difference between the two groups of patients. As shown in the results, the obese HT group showed 92% of patients with abnormal LV geometric pattern, while the non-obese group showed 75% of patients with abnormal LV geometric patterns, i.e., there is additional 17% in the obese HT group is attributed to the presence of obesity besides hypertension. All categories of abnormal LV geometric patterns were noticed in both groups ranging from concentric remodeling to eccentric hypertrophy, to concentric hypertrophy, with the eccentric hypertrophy being more common in the obese HT group, while, the concentric hypertrophy is more common in the non-obese HT group. This finding is attributed to that, obese individuals have expanded intravascular volume and altered LV filling properties[4] and obese individuals develop increase in LV end-diastolic volume[23], i.e., LV volume overload will result in eccentric hypertrophy geometric pattern. This LV volume overload in obesity results from neuro-endocrine activation, renal sodium retention, and heightened systemic oxidative stress on the myocardium that results in hypertrophy and dilation of the left ventricle giving the picture of eccentric hypertrophy[24]. Both the concentric and eccentric hypertrophies are marked by increased myocardial mass. Concentric hypertrophy is characterized by an increase in wall thickness and only a small increase in the external diameter of the heart. In contrast, eccentric hypertrophy is characterized by an increased ventricular diameter but a wall thickness of normal dimensions, see the figure[20]. Thus, in patients with concentric hypertrophy, the left ventricular chamber is small and stiff, while in eccentric hypertrophy, the left ventricular chamber is enlarged and non-stiff. In concentric hypertrophy, the cardiac output is decreased because of the reduced stroke volume, and intraventricular pressures are increased. These changes result in the typical signs and symptoms of congestive heart failure[25].

CONCLUSION

This study has concluded that obesity adds further 17% to the original percentage of abnormal LV geometric pattern induced by hypertension as an adverse effect on the heart. The eccentric geometric pattern of LV hypertrophy is more common in the obese hypertensive patients, and the concentric geometric pattern of LV hypertrophy is more common in the non-obese hypertensive patients. Thus, in the management of hypertensive patients it is very essential to plan for promotion of optimal body weight besides controlling elevated blood pressure through life-style measures such as adequate exercises and proper nutrition and trying to maintain ideal body weight throughout the patient's life.

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