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Cardiology

INFLUENCE OF WEIGHT ON LEFT ATRIAL VOLUME IN OVERWEIGHT OR OBESE WOMEN: A CROSS SECTIONAL STUDY.

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ABSTRACT

Background: Obesity leads to metabolic and structural cardiac abnormalities, including left atrial remodeling, which causes increased atrial volume, therefore increasing the risk of cardiovascular diseases such as AVC and acute coronary syndromes.

Objective: To evaluate whether overweight and obesity are associated with increased left atrial size.

Methods: In this cross-sectional study, we enrolled 44 non-hypertensive women referred by the obesity clinic of EBMSP for a transthoracic echocardiography, from which we obtained data regarding linear and longitudinal extent and volume of the left atrium (LA); diastolic and systolic dimensions, posterior and septal thickness, left ventricular mass, left ventricular ejection fraction, presence of segmental abnormalities, and morphology and competence of cardiac valves. Body weight, height, blood pressure and waist circumference (WC) measurements were performed.

Results: In our study population, 84% were obese, with the mean age of 37 ± 10 years. We found a significant correlation between the LA volume index (LA volume/h) and the body weight (r = 0.4; p = 0.01). Similarly, the body mass index (BMI) correlated with the LA volume (r = 0.4; p = 0.01) and the linear measurement of the LAD (r = 0.4; p = 0.01). In addition, we also demonstrated a positive correlation between waist circumference with both LA volume index (r=0,4; p = 0.006) and LA volume (r = 0,4; p = 0.01).

Conclusion: Obesity showed association with left atrial volume in young women without comorbidities.

KEYWORDS : Obesity. Left atrium. Left atrium volume.

INTRODUCTION

Obesity, defined as abnormal or excessive fat accumulation, has reached epidemic proportions1. Currently, over 10% of the world population is obese and, in Brazil, 51% of the Brazilian population is overweight[2]. The increase in the body mass index (BMI) is the major risk factor for mortality due to cardiovascular causes[3] and other diseases such as diabetes, hypertension, obstructive sleep apnea, dyslipidemia, cancer and degenerative diseases, being the higher risk directly associated with the increase in BMI[4,5].

By cardiac volume overload, obesity determines anatomical and structural changes in the left ventricle and left atrium, due to its contiguous relationship with the ventricle through the mitral valve[6]. Volume overload leads to increase in left ventricle filling pressure, increase in its mass by eccentric hypertrophy, with associated diastolic dysfunction, in addition to causing left atrial remodeling and increasing left atrial volume[7]. Studies have shown evidence of left atrial volume being more accurate for measuring its size than other measurements, such as linear and longitudinal diameter[8,9,10]. Other population-based studies, with a larger sample of obese subjects, have suggested that left atrial volume indexed by height is an independent predictor of all-cause mortality[11] as well as significant correlation between atrial volume and BMI[12].

The Strong Heart Study (SHS) had a follow up of seven years, registering by echocardiogram 368 events in middle-aged and elderly adults[13]. According to the results; LA diameter independently predicted incident cardiovascular events after adjustment for established clinical, echocardiographic and inflammatory risk factors. This simple measure of LA dilatation can identify individuals at risk who may warrant more aggressive risk factor modification[13].

Considering the strength of the evidence in the literature regarding the importance of the left atrium as a risk factor for cardiovascular diseases[8,14,15], left atrial size may be thought of as an indicator of the combined action of these factors over time, and therefore also a possible marker of their integrated action on the risk of cardiovascular diseases. Indeed, left atrial enlargement has been associated with increased risk of death, stroke, heart failure and development of atrial fibrillation[16].

The proposed study has important strengths, first among them the thorough, state-of-the-art echocardiographic examination performed group of individuals, without known cardiac disease or cardiovascular risk factors is also remarkable when risk factors and especially hypertension are highly prevalent, whose results provide information that is largely missing, or at a very preliminary stage, in the literature. In the present study, a group of "healthy" individuals was studied aiming to investigate a possible association between overweight and increase in left atrial volume, allowing one to place the results observed in the subgroup in the correct perspective of obese and overweight individuals whose increase of weight is associated to the increase in left atrial volume, an independent risk marker of mortality, without other factores present as hypertension.

METHODS

Study design

We conducted a cross-sectional study with women with BMI over 25 kg/m^2, with no comorbidities, followed up at the specialized outpatient clinic of the Bahiana Medical Assisting Ambulatory. The study complied with the Declaration of Helsinki, and was approved by the Research Ethics Committee of the Bahiana School of Medicine and Public Health under the number 176.990 / 2012 and CAAE: 02780612.8.0000.5544. All subjects signed written informed consent before study inclusion.

Study population

We enrolled consecutively 44 women with BMI over 25 kg/m^2, who were referred to transthoracic echocardiogram (TTE), as part of the study of subjects with overweight (PWO). Inclusion criteria were female gender, age between 18 and 61 years, BMI over 25 kg/m^2, and sinus rhythm. Exclusion criteria were known comorbidities, atrial or ventricular arrhythmia, pacemaker use, degree of valvopathy higher than discrete, congenital cardiopathy, hypertrophic cardiomyopathy, anemia and systemic arterial hypertension.

Clinical assessments

We obtained structured medical history and physical examination, including height, body weight, BMI, cardiac rate, waist circumference (WC) and blood arterial pressure.

We considered as overweight women with body mass index above 25 kg/m^2 and obese women with a body mass index above 30 g/m^2. Arterial hypertension was defined by previous history of hypertension with use of antihypertensive medications, or systolic levels ≤ 140mmhg and or diastolic levels ≤90mmhg, measured on two different occasions on both arms. Diagnosis of diabetes mellitus and dyslipidemia was based on previous history or use of specific medications for both. Regarding smoking, participants who reported never having smoked were classified as nonsmokers; those who reported having smoked in the past even though they were not currently smoking were classified as ex-smokers; and those who reported smoking at least one cigarette per day were classified as smokers. Diagnosis of coronary artery disease was based on previous clinical history, and/or presence of segmental contractile dysfunction by echocardiogram in individuals with risk factors.

Transthoracic echocardiogram

All subjects underwent transthoracic echocardiogram exam, performed by a cardiologist with ten years of experience and certificate of activity in the field of echocardiography by the Brazilian Cardiology Society and Brazilian Medical Association. We used the echocardiography equipment of the General Electrics, model VIVID 3, equipped with a transducer with a frequency range of 2.5 to 4MHz, for performing a complete study in M, two-dimensional and Doppler (pulsating, continuous, in color and tissue) modes, associated with the electrocardiogram, in accordance with the recommendations of the American Society of Echocardiography (ASE), for quantification of the chambers[17]. In 25 patients, new measurements of A4C and A2C volumes, previously recorded at the time of the first examination, were performed, considering the mean between the two measurements and indexing them by height. They were then correlated with the previous data by Spearman correlation, presenting a significant intraobserver linear association ($r = 0.89$, $p < 0.0001$).

The linear measurements of the left ventricle (LV) were obtained in M mode (thickness of the interventricular sept, posterior wall thickness and final systolic diameter of the LV). The cut-off value for the left ventricle final diastolic diameter (LVDD) > 53 mm for women was used. LV systolic function was calculated by the ejection fraction according to the method of Teicholz[17], for which the bottom limit was defined as 55%. LV mass was evaluated using the formula of Devereux. The upper limit of normal mass was considered 198 g for women. The LV mass indexes were calculated by dividing the LV mass (LVM) by the body surface area (LVM/BSA) and the LVM by height (LVM/h). The relative thickness (RWT) was calculated by the sum of the diastolic thicknesses of the interventricular septal and posterior wall, in relation to the left ventricle end-diastolic dimension: RWT = (2 X posterior wall thickness)/(LV internal diameter at end diastole), considered increased if ≤0.42. LV hypertrophy was considered when the LV mass/h was ≤99 g/m. Diastolic function was evaluated by pulsating Doppler by means of the mitral flow velocities, with the patient in apnea. The E wave, A wave, E/A ratio, DT (E wave deceleration time) and isovolumetric relaxation time (IRVT) were measured.

The Tissue Doppler measuring the velocities at the level of the septal and lateral mitral annulus obtained the following velocities: lateral e', lateral a', septal e', septal a', septal e'/a', lateral e'/a', septal E/e' and lateral E/e'.The left atrial (LA) volume was measured by means of the biplane method of discs (modified Simpson's rule), using the apical cut 4-chambers and apical cut 2-chambers, at the ventricular end-systole, preceding the mitral valve opening, where the maximum size of the LA was obtained. The volume was calculated by the sum of the discs, with the following formula: Volume = $\pi/4$ (h) Σ (D1) (D2), where h is the height and D1 and D2 correspond to the largest and smallest diameter of each disc. The final measurement of the LA volume was the mean of the two cuts. The LA volume index was obtained by dividing the final volume by the body surface area (ml/m2) and by the height in meters (ml/m)[17]. The LA volume indexed by the height was considered increased when ≤ 34 ml/m, based on the study of MONIKA/KORA[11].

Statistical analysis

The sample size calculation was based on the estimated prevalence of 13% increase in atrial volume in obese, non-hypertensive women, as previously described[11]. For the study, 44 individuals were required, considering a precision of ± 10%, with a maximum acceptable difference and alpha of 0.05. The calculation was made with the statistical program WinPepi.

Categorical variables were expressed as numbers in absolute and relative frequencies (percentages) and continuous variables were presented as mean ± SD or median (interquartile range) The Shapiro-Wilk test was used to verify whether the quantitative variables were normally distributed. Comparison of the categorical variables with and without hypertrophy as well as the variable Grade III Obesity, with regard to increase in the LAVI/h was made by means of bivariate analysis, using the Chi-square, and Exact Fisher test, when necessary. For comparison of the means of LAV (left atrial volume) in the groups with and without hypertrophy, the Student's-*t* test for independent samples was used, with statistical significance if $p < 0.05$. Variables such as weight, BMI, LVMI/h, LVDD (left ventricle diastolic diameter) were correlated with the LAVI/h and LAV; presenting $p < 0.10$ in the univariate analysis obtained by Spearman or Pearson correlation coefficient depending on the normality test, the variables were inserted in the linear models regression model, remaining in the model if they continued to be significant ($p < 0.05$).

Analyses were performed using SPSS Inc, Chicago, II, USA and $p<0.05$ (two tailed) was considered statistically significant.

RESULTS

Baseline characteristics

The study comprised 44 women, with mean age 37 ± 10 years. The mean BMI was 36 ± 6 kg/m^2. Seven (15.9%) presented with overweight and 37 (84.1%) with obesity, according to the following distribution: 10 (22.7%) obesity grade I; 17 (38.6%) obesity grade II, and 10 (22.7%) grade III or morbid obesity. Twenty-seven (61.3%) presented with obesity grade I and II. Dyslipidemia was identified in 18 (40.9%) and sedentary lifestyle in 24 (54.5%). The mean weight was 94 ± 17 kg; 41 (93%) had a waist circumference larger than 80 cm. Systolic blood pressure (SBP) was 116 ± 8 mmHg and diastolic blood pressure (DBP) was 77 ± 7 mmHg (**Table 1**).

Table 1. Clinical and demographic characteristics of subjects

Subjects with overweight or obesity (n = 44)	
Age (yrs)	36 ± 10
BMI (kg/m^2)	36.5 ± 6
Weight (kg)	94 ± 17
Height (m)	1.61 ± 5
WC (cm)	108 ± 15
SBP (mmhg)	116 ± 8
DBP (mmhg)	77 ± 7
Sedentary	24 (54.5%)
Current smoker	2 (4.5%)
Diabetes mellitus	1 (2.3%)

Data are expressed as mean ± SD or number for continuous variables, or number (%) for discrete variables.
BMI = body mass index; WC = waist circunference; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Echocardiographic characteristics

The mean diameter of LA was 33 ± 3 mm, by the M mode, and 34 ± 3 mm by the bidimensional mode. Regarding to diastolic function, five subjects (11%) presented with diastolic dysfunction of the LV (left ventricular). The mean of the E/A ratio was 1.5 ± 1.2, whereas we found 1.2 ± 1.1 for the septal e'/a' ratio, 1.5 ± 1.3 for the lateral e'/a' ratio. The mean DT (deceleration time) was 230 ± 41 ms; and the mean IVRT (Isovolumetric relaxation time) was 82 ± 10 sec. All subjects presented normal filling pressure, measured by the lateral E/e 'ratio < 15; with the mean value of 5 ± 1.8. The mean LAV was 44 ± 9, the mean LAVI indexed by height was 27 ± 6, and the mean LAVI indexed by BSA was 22 ± 4. All echocardiographic data are shown in Table 2.

Table 2. Echocardiographic findings

Subjects with overweight or obesity (n = 44)	
Diastolic dimension (cm)	4.8 ± 3
Systolic dimension (cm)	2.9 ± 2
Septal thickness (cm)	0.7 ± 1
Posterior wall thickness (cm)	0.7 ± 1
Relative Wall thickness (cm)	0.4 ± 1
LV mass/BSA (g/m^2)	150 ± 30
LV mass/height (g/m)	76 ± 13
LV EF Teichholz (%)	93 ± 19
E/A ratio	67 ± 4
E/e' ratio	1.5 ± 5
Lateral e/a' ratio	6 ± 1.8
Septal e'/a' ratio	1.5 ± 0.5
LAVI (ml)	1.2 ± 0.5
LAVI/h (ml/m)	44 ± 9
LAVI/BSA (ml/m2)	27 ± 7
DT, ms	230 ± 4
IVRT, seg	82 ± 10
Diastolic dysfunction	5 (11%)
Hypertrophy	18 (41%)

Data are expressedas mean ± SD or median for continuous variables, or number (%) for discrete variables.
BMI = body mass index; WC = waist circunference; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Eight (18.2%) subjects presented increase in LA volume, as follows: 7 (15.9%) increase of a weightless, and 1 (2.3%) of a moderate degree. Seven (15.9%) showed increase by means of the LA volume indexed for height, and three (6.8%) when the LA volume was indexed by BSA. Two subjects (4.5%) showed increase in the LV size, by means of its linear diameter. All subjects with increase in LAVI/BSA and in LAD also presented with increase in the volume indexed by height. There was no increase in LAVI in subjects with overweight. Out of seven who presented increase in the LAVI/h, five (71.4%) had obesity grade III, whereas two (28.6%) had obesity grade I and grade II. The prevalence of obesity grade III in subjects with increase in the LAVI/h was significantly higher than in those who did not have this condition (p = 0.009).

Seven subjects in the sample (15.9%) presented with increase in LAVI/h and eighteen (40.9%) with increase in atrial volume. Eighteen subjects (33.3%) with Left ventricle hypertrophy had increased LAVI (Left atrial volume index). Regarding to LV geometry, 18 (41%) subjects presented LV hypertrophy, with 14 (31.8%) being of a light degree; 1 (2.3%) moderate, and 3 (6.8%) an important degree, all with hypertrophy of the eccentric type, based on LVMI/h (left ventricular mass indexed by height), with predominance of the light type. Five (11.4%) presented with LV hypertrophy, when the LVMI/BSA (left ventricular mass indexed by body surface area) was used. Considering the subjects with LV hypertrophy, 6 (33.3%) had increase in LAVI/h, with 5 (71.4%) of them having hypertrophy of a light degree and 1(14.3%) hypertrophy of the important degree. Therefore, 33.3% of those who had increase in LAVI/h > 34 ml/m had left ventricular hypertrophy, and when the groups with and without LVH were compared with regard to the increase in LAVI/h, statistical significance was shown (p=0.009). Figure 1.

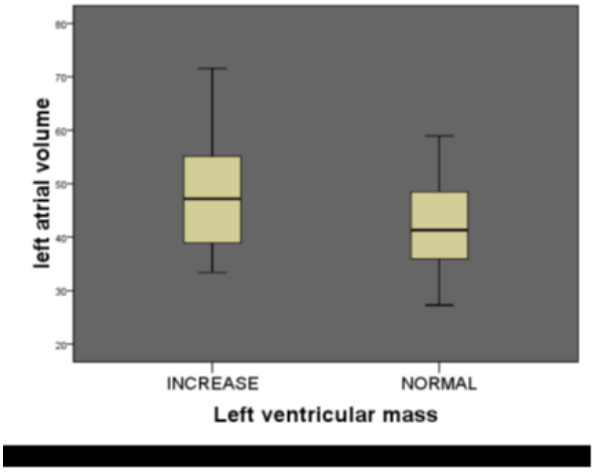

Figure 1. Left atrial volume in subjects with or without left ventricular hypertrofy (T test, p<0.001)

We found linear association between the variables weight and atrial volume indexed by height, through the dispersion graph (Figure 2), evidencing the influence of weight in increasing atrial volume.

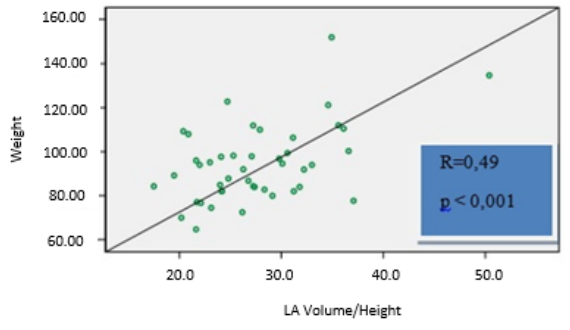

Figure 2. Correlation between height indexed left atrial volume and weight (Spearman Correlation, r:0.49, p<0.001)

We showed a moderate correlation between BMI and the following parameters: LAVI/h (r = 0.4; p =0.01), LAD (r = 0.4; p = 0.007), longitudinal LAD (r = 0.35; p = 0.01), and LAV (r = 0.4; p = 0.01). In addition, we found that weight was significantly correlated with LAVI/h (r = 0.4; p = 0.015), LAD (r = 0.4; p = 0.01), longitudinal LAD (r = 0.3; p = 0.01) and LAV (r = 0.4; p = 0.01).

The other clinical and echocardiographic variables related to left ventricular geometry and diastolic function were tested in a univariate analysis, only after showing linear association in the dispersion graph, by means of Spearman or Pearson correlation with LAV and LAVI/h (Table 3).

Table 3. Relation of echocardiographic and clinic characteristics to Indexed Left Atrial Volume/h

Variables	Standardized Coefficients *β*	Probability *p*
Medical history		
Age (years)	0.20	0.14
BMI	0.40	0.01
LV mass/BSA (kg/m²)	0.40	0.01
SBP (mmHg)	0.01	0.91
DBP (mmHg)	- 0.06	0.68
Waist circunference (m)	0.40	0.006
Echocardiographic variables		
Diastolic dimension (cm)	0.20	0.03
Systolic dimension (cm)	0.20	0.10
Septal thickness (cm)	0.01	0.92
Posterior wall thickness (cm)	0.10	0.51
Relative wall thickness (cm)	0.02	0.90
LV mass/h (kg/m)	0.30	0.04
E/A ratio	- 0.03	0.81
Lateral E/e' ratio	- 0.07	0.60
Septal e'/a' ratio	0.12	0.41
Lateral e'/a' ratio	0.006	0.96

p Values for mean values were tested by Spearman correlation coeficient
BSA, body surface área; SBP, systolic blood pressure; DBP, systolic blood pressure; EF, ejection

Atrial volume

The following variables were selected for evaluation by multivariate linear regression: BMI, WC (waist circumference), LVDD and LVMI/h, because they presented moderate correlation with LAVI/h and age, mean SBP, mean DBP, which are considered risk for increase in LAV. The variables WC and BMI were analyzed independently, with the other variables, by the collinearity between them. After analysis, only BMI (P = 0.001) and LVMI/h (p = 0.03) remained as variables associated with LAVI/h (Table 4). When WC was included, it remained as the only variable influencing the size of the LAVI/h (p = 0.0001). In the multivariate analysis, including BMI, LVDD, WC, LV mass/h, Mean SBP, mean DBP, and age, only BMI (p = 0.001), LVMI/h (p = 0.02) and LVDD (p = 0.02) were shown to be independently associated with the increase in LAV.

Table 4. Multivariable linear regression for cross-sectional association of modifiable risk factors with left atrial volume

	Standardized regression coefficients *β*	Probability *p*
BMI	0.48	0.001
LV mass/h	0.31	0.003

Cross-sectional regression models adjusted for age, systolic blood pressure, diastolic blood pressure and LV internal dimension, diastolic dimension.

DISCUSSION

Our results clearly demonstrated that there was predominance of left atrial volume increase when this variable was indexed by height in relation to body surface area. Previous studies have shown greater accuracy of cardiac measurements when height in relation to bsa was used[9], with the LAVI/h recently being most frequently used as a measure of the left atrial size[11,18] indicated as the best variable for indexing in the calculation of LAV.

This study included only women with overweight and obesity, without other comorbidities such as arterial hypertension or diabetes, that can be confounding factors and interfere directly in remodeling of the left atrium, and ultimately, in increase in atrial volume. The literature mentions two large longitudinal, population-based studies[11,12] which included individuals with obesity. One of these is the study of MONIKA, that enrolled subjects between 25 to 71 years of age, with age being a confounding factor for increase in atrial volume. The present study included women who had a body mass index > 25 g/m^2; up to 61 years of age, with overweight or obesity and normal pressure levels.

The majority of subjects who presented increase in the LAVI/h had hypertrophy of the eccentric type (exclusively), as the probable

adaptive mechanism of the heart with regard to volume overload associated with obesity, according to present-day studies[6]. Left ventricular hypertrophy was diagnosed more when the LVMI indexed by height than by BSA was used, in accordance with the guidance of the American Society of Echocardiography[18], in which the cut-off point for the diagnosis of hypertrophy is higher. There was predominance of hypertrophy in those with increase in the LAVI/h.

The increase in cardiac mass appears to be an early cardiac structural change in subjects without comorbidities, determining atrial remodeling and increase in LA volume. The presence of LV hypertrophy would determine an increase in left ventricular diastolic pressure, and thereby, change in relaxation, consequently leading to diastolic dysfunction and left atrial remodeling. According to other studies about obesity[20], diastolic dysfunction is expected to be found in those subjects with hypertrophy and possibly also in those with increase in LAVI/h.

In our sample, women with LV hypertrophy and increase in LAVI/h, presented a higher BMI, weight, waist circumference and obesity grade III, when compared to those, without increase in LAVI/h. These data are in agreement with those of Crisóstomo et al., who demonstrated and increase in LV volume and mass when indexed by height, in a group of obese subjects without comorbidities[21].

Obesity, by means of increase in preload as a result of the presence of volume overload, triggers adaptive mechanisms in the heat, such as: eccentric hypertrophy and diastolic dysfunction. This in turn increases the left ventricular filling pressure and causes remodeling and increase in the LA size resulting from its contiguity to the LV through the mitral valve[4].

Diastolic dysfunction was found in their study population presenting a E/A<1 ratio and e'/a'<1 ratio, classified as a light degree according to the American Society of Echocardiography[18]. The mean E/A ratio was comparable with that found in the study of Crisóstomo et.al, in which it was demonstrated that alteration in the E/A ratio can indicate initial diastolic dysfunction in obese subjects without comorbidities.

The increase E/e 'ratio, associated with more severe diastolic dysfunction, whose magnitude expresses the concomitant increase in LAVI, presented values considered normal (lower than 15) demonstrating normal left ventricular filling pressure, and can be explained by small frequency of moderate diastolic dysfunction and important in this sample, with a smaller increase in preload. El Aouar et al[19] demonstrated correlation between the LA volume and diastolic dysfunction in 500 individuals in a Brazilian population, showing there was progressive increase in the LAV as the diastolic dysfunction worsened from moderate to important[20]. In our study, the small number of obese grade III subjects, may justify the predominance of light diastolic dysfunction with even normal filling pressure. On the other hand, Tavares, et al[20], in a transversal study, showed high frequency of diastolic dysfunction and increase in LA, in a sample of predominantly obesity grade III subjects, whose enlargement of LA is a marker of both the severity and chronicity of diastolic dysfunction and the magnitude of pressure rise in the left atrium[19].

In this study, a moderate correlation was observed between the measure of the left atrial size (LAD, longitudinal LAD, LAV, LAVI/h and LAVI/BSA) with weight, and with BMI, suggesting the influence of weight and body mass index on the increase in left atrial volume. Armstrong et al carried out the study CARDIA[12], evaluating modifiable risk factors over the course of 20 years, with regard to LAD indexed by height and by body surface area, identifying two single predictors with strong association with left atrial increase: increase in AP and BMI[12]. These data corroborate those of the present study, that demonstrated a moderate association between atrial size and BMI. After univariate analysis, variable such as BMI remained in the multivariate analysis associated with LAVI/h.

Considering the most recent recommendation for the quantification of cardiac chambers by the echocardiogram, that was published in 2015 by the American Society of Echocardiography (ASE) and the European Association of Echocardiography (renamed European Association of Cardiovascular Imaging (EACVI)), left atrial volume, after data analysis, remains as an independent variable associated with weight gain in patients with obesity[13].

This study evidenced independent association of obesity with left atrial volume increase, possible risk marker, without being able to establish a direct causal relationship. Although the sample was small, it was representative, those women with overweight and obesity, healthy, with no pressure, referenced to obesity clinic.

CONCLUSIONS

Obesity presents independent association with increase in left atrial volume in young women without comorbidities. Furthermore, left ventricular hypertrophy is associated with left atrial volume with higher frequency when indexed by height.

Competing interests

No competing interests to disclose.

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