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RELATIONSHIP BETWEEN ANTHROPOMETRIC MEASURES AND LEFT VENTRICULAR HYPERTROPHY IN OBESE HYPERTENSIVE PATIENTS

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ABSTRACT

Objective: To verify the relationship between anthropometric measures and left ventricular hypertrophy, as assessed by the Cornell Index, in obese hypertensive patients. **Methods:** Cross-sectional study with obese hypertensive patients of both genders, aged ≥ 20 years, treated as outpatients between 1996 and 2011 at the Hypertension Clinic of the Hospital das Clínicas of the Federal University of Pernambuco, Brazil. Data collected included age, blood pressure, heart rate, and anthropometric parameters: body mass index, waist/height ratio, waist/hip ratio, and abdominal circumference. Left ventricular hypertrophy was evaluated by the Cornell voltage criteria through an electrocardiogram. **Results:** The study included 486 individuals with a median age of 60 (IQ = 53-69) years and a predominance of women (83.5%). The median body mass index was 32.5 (30.9-34.8) kg/m², being similar between genders (p = 0.0745). Body mass index, abdominal circumference, waist/hip ratio (WHR), and waist/height ratio (WHtR) measures did not differ between patients with and without left ventricular hypertrophy (p > 0.05). The Cornell Index correlated with age (r = 0.1217; p = 0.0072), systolic blood pressure (r = 0.6838; p < 0.001), diastolic blood pressure (r = 0.6383; p< 0.001), and waist/hip ratio (r = 0.1193; p = 0.0083). **Conclusions:** Waist/hip ratio was the anthropometric measure that correlated with the Cornell Index, as did age and blood pressure levels in obese hypertensive individuals. Measures traditionally used to indicate overall (body mass index) and central (abdominal circumference, waist/height ratio) obesity did not correlate with left ventricular hypertrophy.

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INTRODUCTION

Obesity is an isolated risk factor for cardiovascular diseases, which can lead to cardiac hypertrophy followed by dilated cardiomyopathy, predisposing to fatal arrhythmias.[1,2] Obesity also causes organ-specific alterations through a direct mechanical effect of the adipose tissue, or systemically through humoral mediators and metabolic adjustments that alter hemodynamics and cardiac geometry.[3,4] The first studies that found an independent association between obesity and increased ventricular mass in the '60s[5] were later confirmed with echocardiographic studies[6,7] and reinforced with larger population studies.[8-12] Several echocardiographic studies evidence a significant association between central obesity and greater left ventricular mass in normotensive and hypertensive subjects.[13-16] Furthermore, some authors have directly related central adiposity to the diameter of the left ventricular chamber in hypertensive and normotensive individuals.[13,15,16] These observations indicate that the increase in circulating volume secondary to central adiposity can stimulate left ventricular remodeling.[15,16] On the other hand, although the relative thickness of the left ventricle also tends to increase in parallel with the accumulation of body fat regardless of blood pressure levels,[17,18] the analysis of the role of central adiposity in this process has generated conflicting results. Thus, the present research focuses on the health reality of obese hypertensive patients in order to assess, examine, and establish effective therapeutic support. In this sense, this study verifies the relationship between anthropometric measures and left ventricular hypertrophy (LVH), as assessed by the Cornell Index, in obese hypertensive patients.

METHODS

Cross-sectional study developed at the Hypertension Clinic of the Hospital das Clínicas of the Federal University of Pernambuco, Brazil, between 1996 and 2011. The study sample consisted of obese

hypertensive patients. The data used in this research were retrieved from a previously collected database, from the research "*Doença renal em hipertensos portadores de síndrome metabólica*" ("Kidney disease in hypertensive patients with metabolic syndrome"), comprising 2122 individuals. The present study included individuals of both genders, aged ≥ 20 years, with a body mass index (BMI) ≥ 30 kg/m^2. Patients with secondary arterial hypertension, diabetes *mellitus*, cardiac arrhythmias, electrical conduction disturbances on the electrocardiogram (ECG), heart failure, previous myocardial infarction, and dyslipidemia with the use of lipid-lowering medication were excluded. Considering an α error of 5%, a β error of 20%, with an estimated correlation between BMI and the Cornell Index of 0.5 (p), and a variability of 0.07 (d²), using the formula n = [(Zα/2 + Zβ/2)2 x (px (1—p)] / d^2, we reached a minimum sample size of 400 individuals. To cover any errors, the sample was increased by 20%, making a total of 500 individuals to be evaluated. Data were collected in the first consultation of patients (year 2000), considering: age, blood pressure, heart rate, and anthropometric parameters. The following anthropometric parameters were collected: weight, height, abdominal circumference (AC), and hip circumference (HC). From these measures, body mass index (BMI), waist/height ratio (WHtR), and waist/hip ratio (WHR) were analyzed. All measures were collected in duplicate by a single observer and repeated when the measurement error between them was greater than 0.1 cm (height, AC, and HC) or 0.1 kg (weight). The final measure considered was the mean between the two closest values. Body weight and height were measured using the techniques recommended by Lohman, Roche, and Martorell.[19] For this, we used an electronic scale (brand FILIZOLA®) with 150kg capacity, 100g division, equipped with a millimeter-graduated anthropometer. Measurements were taken with the patient wearing light clothing, barefoot, in a supine position, with heels together. Weight and height values were used to calculate BMI.[20] Abdominal circumference was obtained using an inelastic measuring tape, accurate to 0.1 cm, directly on the skin, at the midpoint between the last rib and the iliac crest.[(20)] The bone marks of the last rib and the iliac crest were located and palpated by the examiner at the level of the midaxillary line. The measuring tape was placed in a horizontal plane around the abdomen at the location described above, and special attention was paid to ensuring that the tape was parallel to the floor. The measurement was performed at the end of normal expiration with the inelastic tape adjacent to the skin but without compressing it, with the participant standing upright.[(20)] Waist-hip ratio was determined by the quotient of the waist (cm) and hip (cm) perimeters, obtained by measuring the hip region in the area of greatest protuberance and the waist region in the narrowest area between the chest and hip.[(20)]

The waist-height ratio was obtained from the quotient between waist circumference (cm) and height (cm).[21] Left ventricular hypertrophy (LVH) was diagnosed by electrocardiogram (ECG) using repolarization, voltage, and duration criteria.[22-25] The Cornell voltage criteria was considered (sum of the amplitude of the R wave in the VL lead with the S wave in the V3 lead greater than 28 mm in men and greater than 20 mm in women).[26,27] The conventional resting electrocardiogram, with 12 leads (peripheral and precordial), was performed using a "CARDIOLINE Digital ECG" electrocardiograph, with a standard of 25 mm/sec, 10 mV/mm, HDM1, Auto1 or Auto2 HR77. The auscultatory method was used to measure blood pressure (BP) through a duly calibrated mercury column sphygmomanometer (brand TAKAOKA, model 203) and a Littmann cardiological stethoscope, following the technique recommended by the VI Brazilian Guidelines on Hypertension.[28] In the first evaluation, measurements were taken in the sitting position, in both arms, and in case of difference between pressure levels, the arm with the highest value was used for subsequent measurements. Three measures were taken with an interval of two to three minutes between them, and the arithmetic mean was used as the final value in the consultation. This study was submitted for evaluation and approved by the Ethics Committee for Research Involving Human Beings of the Health Sciences Center of the Federal University of Pernambuco (CEP/CCS/UFPE), under Registration No. 343/09. Ethical principles were considered according to Resolution 466/12 of the National Health Council. Data were analyzed using the Statistical Package for Social Sciences – SPSS version 13.0 (SPSS Inc., Chicago, IL, USA) and Stata14 software (StataCorp., College Station, TX, USA). Initially, exploratory data analysis was performed (outliers exclusion) and the normality of continuous variables was tested using the Kolmogorov Smirnov test. The data showed nonGaussian distribution, thus being presented as medians and respective interquartile ranges (IQ). In the description of proportions, the binomial distribution was approximated to the normal distribution using the 95% confidence interval. The Mann Whitney U test was used to compare two medians. The Spearman linear correlation was used to assess the correlation between anthropometric measures, age, BP, HR, and the CornellIndex. Statistical significance was established when $p < 0.05$.

RESULTS

The study initially included 500 patients. After eliminating losses due to inconsistency of information, the final sample comprised 486 obese hypertensive outpatients with a median age of 60 (IQ = 53-69) years and a predominance of women (83.5%). The median Cornell index was 20 (IQ = 16-26) mm, being higher in males [28 (IQ = 17-32.5) vs 19.5 (IQ: 16-25) mm; $p < 0.001$]. The median BMI was 32.5 (30.9-34.8) kg/m², being similar between genders ($p = 0.0745$). The median AC and WHR were higher among men ($p < 0.001$), and the median WHtR was higher among women ($p < 0.001$). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) did not differ between genders ($p > 0.05$) (Table 1). When comparing the variables related to LVH, patients with this condition had a higher median age ($p = 0.0416$). However, when the sample was stratified by gender, this difference was not maintained, with age being similar between the groups of men and women with and without LVH (Table 2). Regarding anthropometric measures, BMI, AC, WHR, and WHtR did not differ between patients with and without LVH ($p > 0.05$). Patients of both genders with LVH had higher medians of SBP and DBP ($p < 0.001$) (Table 2). The Cornell Index correlated with age ($r = 0.1217$; $p = 0.0072$), SBP ($r = 0.6838$; $p < 0.001$), and DBP ($r = 0.6383$; $p < 0.001$). Among the anthropometric measures, only WHR correlated with the Cornell Index ($r = 0.1193$; $p = 0.0083$) (Table 3). When stratifying the sample by gender, only age, SBP, and DBP correlated with the index ($p < 0.05$) in males, and only SBP and DBP correlated with the index in females ($p < 0.05$) (Table 3). Multiple linear regression analysis was not possible due to the non-normal distribution pattern of the Cornell Index.

DISCUSSION

Early identification of electrocardiographic findings that assess the Cornell Index, which helps to diagnose LVH, together with anthropometric analyses that detect global and central adiposity will contribute to prophylactic and therapeutic actions on cardiovascular risk. Some studies have shown that BMI and central obesity indicators were determinants of LVH development.[29-33] Okin et al. reported higher Cornell Index (CI) for overweight and obese patients than for patients with normal weight.[(29)] In the present study, CI increased with the progression of the nutritional status (as shown by the BMI), corroborating previous reports.[34] Despite this evidence, our results did not show this relationship with BMI, only with WHR in the total population. When stratifying the sample by gender, this correlation was not maintained. Body mass index (BMI), waist-height ratio (WHtR), and abdominal circumference (AC) did not correlate with the Cornell Index (CI) in the sample under study and did not have different measures according to the presence or absence of left ventricular hypertrophy (LVH). The waist-hip ratio reflects the type of fat distribution (gynecoid or android).[35] Some studies have associated an android distribution model - with excess fat in the upper abdomen - with increased incidence of cardiovascular involvement, especially in relation to the gynecoid model. This latter model is characterized by increased fat in the lower body region, particularly hips and thighs.[36]

Table 1. Anthropometric and clinical variables stratified by sex of obese hypertensive patients treated as outpatients at the Hypertension Clinic of a University Hospital, Recife-PE, (n=486)

Variables	Total sample	Male	Female	p-value*
	n=486	n=80	n=406	
Age (year)	60 (53-69)	58.5 (53 -71)	60.5 (54-68)	0.5756
Cornell (mm)	20 (16-26)	28 (17-32.5)	19.5 (16-25)	<0.001
BMI (kg/m²)	32.5 (30.9-34.8)	32.0 (30.8-33.7)	32.8 (31-35)	0.0745
AC (cm)	102 (98-108)	106 (100-110)	100 (96.7-107.9	<0.001
WHtR	0,65(0,63-0,70)	0,64 (0,61-0,67)	0,66 (0,63-0,70)	<0.001
WHR	0 .91 (0.88-0.93)	0 .98 (0.96-1.02)	0.90 (0.88-0.92)	<0.001
SBP (mmHg)	162 (150-180)	162 (153-179)	162 (150-180)	0.6747
DBP (mmHg)	100 (92-108)	101 (92-109)	100 (92- 108)	0.8014
HR (bpm)	82 (76-88)	84 (74-90)	82 (76-88)	0.3285

* U de Mann Whitney Test; BMI: Body Mass Index; AC: Abdominal Circumference; WHtR: Waist/height ratio; WHR: Waist/hip ratio; SBP: systolic blood pressure; DBP: Diastolic blood pressure; HR Heart Ratepressão arterial sistólica; PAD: pressão arterial diastólica; FC: frequência cardíaca.

Table 2. Comparison of anthropometric and clinical variables according to left ventricular hypertrophy (LVH) stratified by sex in obese hypertensive patients treated as outpatients at the Hypertension Clinic of a University Hospital, Recife-PE, (n=486)

Variables	Total sample		p-value*	Male		p-value*	Female		p-value*
	Without LVH	With LVH		Without LVH	With LVH		Without LVH	With LVH	
	n= 254	n= 232		n= 42	n= 38		n=212	n=194	
Age (year)	59 (52-67)	61 (55-70)	0.0416	56.5 (51-66)	60 (55-74)	0.0713	60 (52-67.5)	61 (55-69)	0.1753
BMI (kg/m²)	32.5 (31-34.6)	32.55 (30.85-35.3)	0.9204	31.8 (30.8-33.7)	32.1 (30.4-35.3)	0.8888	32.8 (31-34.8)	32.7 (30.9-35.2)	0.8393
AC (cm)	101.2 (98-108)	102 (97-108)	0.9850	107 (100.3-110)	105.4 (98-110)	0.8203	100 (97.5-106.2)	101 (96.5-108)	0.8855
WHtR	0,65 (0,63 -0,68)	0,65(0.63-0,70)	0.7067	0,64(0,61-0,66)	0,63 (0,62-0,63)	0.9846	0,66 (0,63-0,69)	0,66 (0,63-0,70)	0.6950
WHR	0.9(0.88-0.93)	0.91 (0.89-0.93)	0.2580	0.98 (0.96-1.02)	0.98 (0.96-1.02)	0.7599	0.90 (0.88-0.91)	0.90 (0.88-0.92)	0.1585
SBP (mmHg)	150 (146-160)	180 (168-188)	<0.001	154.5 (142-160)	179 (165-189)	<0.001	150 (146-160)	180 (168-188)	<0.001
DBP (mmHg)	94 (88-100)	108 (104-112.5)	<0.001	96 (90-100)	108.5 (104-113)	<0.001	92.5 (88-99.5)	108 (104-112)	<0.001
HR (bpm)	80 (76-88)	84 (77-88)	0.3187	81 (70-90)	84 (80-88)	0.4279	80 (76-88)	84 (76-88)	0.4632

* U de Mann Whitney Test; BMI: Body Mass Index; AC: Abdominal Circumference; WHtR: Waist/height ratio; WHR: Waist/hip ratio; SBP: systolic blood pressure; DBP: Diastolic blood pressure; HR Heart Rate pressão arterial sistólica; PAD: pressão arterial diastólica; FC: frequência cardíaca.

Table 3. Correlation of anthropometric and clinical variables stratified by sex in obese hypertensive patients treated as outpatients at the Hypertension Clinic of a University Hospital, Recife-PE (n=486)

Variables	Total sample		Male		Female	
	r	p-value*	r	p-value*	r	p-value*
Age (year)	0.1217	0.0072	0.285	0.011	0.075	0.133
BMI (kg/m²)	-0.0354	0.4364	-0.081	0.477	-0.008	0.866
AC (cm)	0.0183	0.6867	-0.039	0.732	-0.017	0.726
WHtR	0.0282	0.5357	-0.009	0.934	-0.003	0.957
WHR	0.1196	0.0083	0.099	0.383	-0.001	0.992
SBP (mmHg)	0.6838	<0.001	0.650	<0.001	0.701	<0.001
DBP (mmHg)	0.6383	<0.001	0.529	<0.001	0.678	<0.001
HR (bpm)	0.0616	0.1749	-0.013	0.097	0.073	0.141

*Spearman Correlation; BMI: Body Mass Index; AC: Abdominal Circumference; WHtR: Waist/height ratio; WHR: Waist/hip ratio; SBP: systolic blood pressure; DBP: Diastolic blood pressure; HR Heart Rate.

Research has shown that a high WHR is a risk factor for cardiovascular diseases such as arterial hypertension.[37,38] Despite the weak correlation of this factor, which was not maintained in the analysis stratified by gender in our results, it should be considered that this is a simple, low-cost, and easily applicable measure.[35,39] Cardiac hypertrophy is a compensatory response to cardiac insult of any cause. It is caused by interactions between genetic and nongenetic factors that involve multiple etiologies and complex mechanisms.[40] The literature relates obesity to the structure and mass of the left ventricle (LV).[(31)] A study in the Brazilian population showed that BMI and AC were the main determinants of LV mass increase in normotensive individuals.[41] One of the possible mechanisms postulated to explain this relationship refers to compensatory cardiac physiology. In this sense, obese individuals have increased intravascular volume and cardiac output in order to meet the increased metabolic demands from the increase in adipose tissue. Moreover, these individuals seem to present greater sympathetic activity,[42] which is one of the mechanisms participating in the genesis of LVH.[43] Another possible factor is the presence of arterial hypertension (AHT), a common finding in obese individuals with accumulation of abdominal fat, which in itself corresponds to the main risk factor for LV remodeling.[44] The increase in pressure increases parietal systolic stress, with parallel addition of sarcomeres and an increase in wall thickness.[45] In this case, patients with AHT associated with obesity would have a double stimulus for the development of hypertrophy, increasing the risk of developing heart failure.[(44)] Thus, in our findings, it is not surprising that systolic and diastolic pressures correlated with CI in both genders. Noteworthy, females have lower blood pressure levels, possibly due to greater adherence to the treatment of hypertension. As already established in the literature, the female gender is the one that most seeks health units.[46]

The occurrence of LVH or LV remodeling is considered an important risk factor for future cardiovascular morbidity and mortality.[(45)] The risk of myocardial infarction, heart failure, sudden death, and other cardiovascular events is six to eight times higher in patients with LVH.[47] In LVH, there seems to be a decrease in the coronary reserve flow and an increase in the risk of ectopic ventricular activity.[45] Another finding of the present research was the relationship between age and CI. This factor correlates with AHT and LVH due to the decrease in vascular compliance with the advance of time. There may be changes in the microvasculature, arterial dysfunction, and atherosclerosis, contributing to the increase in arterial stiffness. The sustained increase in blood pressure interferes with elastin, collagen, and muscle cells that modify functionality and accelerate vessel aging.[28] Some limitations must be considered when interpreting the results. First, it is a cross-sectional study, which does not allow distinguishing the relationship between cause and effect. Furthermore, the population was recruited from a hospital service, limiting the generalizability of the results. It should also be considered that the study only assessed individuals with established AHT. The present study did not evaluate other important variables such as disease duration, inflammation mediators, and insulin resistance. However, other authors have already described LV remodeling in obese individuals, even in the absence of AHT and other risk factors.[48]

CONCLUSION

Waist-hip ratio (WHR) was the anthropometric measure that correlated with CI, as did age and blood pressure levels in obese hypertensive individuals. In this sample, measures traditionally used to indicate global obesity (BMI) and central obesity (AC, WHtR) did not correlate with LVH, but are still useful and accessible in clinical practice and in the context of public health. Further studies are needed to investigate the relationship between obesity and LVH in specific subgroups and to consider other predictors of this condition, such as disease duration, total fat percentage, and intra-abdominal fat composition. These aspects can help in the adoption of preventive and therapeutic measures to minimize the effects of obesity on cardiac physiology.

Authors' Contributions

CCL: Conceptualization, Data Curation, Writing – Original Draft. CPSP: Formal Analysis, Writing – Review & Editing. DAM: Formal Analysis, Validation. CAF: Data Curation, Validation. IKGA: Data Curation, Validation. HCCJ: Data Curation. MCCL: Conceptualization, Writing – Review & Editing.

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