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Article

Association Between Systemic Arterial Hypertension with Laboratory Markers, Body Composition, Obstructive Sleep Apnea and Heart Rate Variability in Obese Adults

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Abstract: Background: Elevated fasting plasma glucose and visceral fat area (VFA) is highly prevalent in obese adults. This study investigated the associations between systemic arterial hypertension (SAH) and laboratory, anthropometric, heart rate variability (HRV), and obstructive sleep apnea markers. Methods: Cross-sectional study with 95 obese patients treated at Obesity Treatment and Surgery Center, located in Salvador, BA, Brazil. SAH data were obtained from electronic medical records of patients. To evaluate the association of SAH with the predictor variables, the sample was stratified in Normotense Group (NG) and Hypertensive Group (HG), and laboratory markers, body composition, polysomnography data, and HRV were measured. Results: The average age of the NG was 36.3 ± 10.1 and HG 40.4 ± 10.6 years, 73.7% were women in the NG and 57.9% in HG; 82.4% in HG had insulin resistance. In the multivarious logistics regression model with adjustments age, sex, height, and oxyhemoglobin saturation, SAH was inversely associated with fasting plasma glucose mg/dL (odds ratio [OR] = 0.96; 95% interval confidence [CI] = 0.92 - 0.99) and VFA cm² (OR = 0.98; 95% CI = 0.97 - 0.99). The area under curve the VFA was 0.728; CI 95% (0.620 - 0.836) and fasting plasma glucose 0.693; CI 95% (0.582 - 0.804). Conclusions: Lower VFA and fasting plasma glucose concentrations were inversely associated with SAH. These results indicate opportunities to improve the outcome in obese patients through counseling and clinical interventions.

Keywords: hypertension; obesity; body composition; intra-abdominal fat; sleep apnea; obstructive

1. Introduction

Systemic Arterial Hypertension (SAH) is a multifactorial disease and can be environmental and/or genetic, such as lack of physical activity, obesity, and eating habits (1). According to the World Health Organization, it is estimated that 1.28 billion adults aged 30 to 79 years worldwide have hypertension (2). Currently, SAH is associated with a higher risk of mortality and is a significant factor for complications of kidney and cardiovascular events (3). The diagnosis of hypertension is obtained through exams such as MAP, and to be considered hypertensive the individual should be characterized with systolic blood pressure ≥ 140mmHg and diastolic blood pressure ≥ 90mmHg (4).

Hypertension can be induced by possible changes caused by obesity, such as the stimulation of mechanisms that contribute to this hypertensive state, such as hormonal changes, and inflammatory and endothelial levels. (5). Obesity is associated with a decrease in life expectancy and its prevalence has become a major worldwide health problem, as excessive weight gain predisposes an increased risk of various diseases, including cardiovascular, cerebrovascular, and metabolic disease – all of them associated with SAH (6,7).

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Prior literature data describes that some factors should be considered risk predictors for the occurrence of SAH, among them are obstructive sleep apnea syndrome, body mass index (BMI), waist circumference (WC), abdominal visceral fat, heart rate variability (8–10), some laboratory biochemical markers and associated comorbidities (11–13). Due to the number of variables and their possible associations, further research on relationships between these data and SAH is necessary, so that more reliable and independent predictors can be obtained for decision making in clinical practice, facilitating the prognostic of SAH in this population.

Sleep-related breathing disorders such as obstructive apnea can accelerate the elevation of blood pressure levels present in adults, especially acutely, and it may be due to hypoxia at night (8). The mechanisms caused by a higher value of BMI, WC, body fat, and blood glucose can cause sympathetic nervous system stimulation, changes in the renin-angiotensin-aldosterone system, an increase of inflammatory markers, and other factors responsible for balance in the circulatory system thus being able to associate with SAH (14–17). Hypertension is also related to autonomic deregulation and since HRV can also be characterized by greater sympathetic activation it can be said that this would be the mechanism associated with SAH (18).

Given the above, the objective of the present study was to investigate the SAH associations with laboratory biochemical markers, anthropometric and body composition measures, heart rate variability, and obstructive sleep apnea in obese adults.

2. Materials and Methods

2.1. Study design and sample

This study was based on cross-sectional data of 95 patients aged ≥ 21 years with obesity diagnosis and elective to bariatric surgery in a private clinic of surgery and treatment of obesity in the city of Salvador in Brazil. Data were collected from May 2016 to August 2018. Patients with a cognitive deficit and without all clinical and laboratory data were not included in the study. The study volunteers were categorized into two groups according to the clinical diagnosis of SAH: normotensive and hypertensive. The study was submitted and approved by the Research Ethics Committee of the Bahiana School of Medicine and Public Health, under number 1.530.178.

2.2. Measuring instruments

2.2.1. Body composition

Body composition data were measured by octopolar electrical bioimpedance through the Inbody 720 equipment (Inbody Canada Corp, Ottawa, Ontario, Canada) fulfilling the procedures specified in the literature. This bioimpedance uses eight electrodes, two in contact with the palm (E1, E3) and the thumb (E2, E4) of each hand and two in contact with the anterior (E5, E7) and posterior (E6, E8) of the plant of each foot. Five segmental impedances (right arm, left arm, right leg, left leg, and trunk) are measured at 1, 5, 50, 250, 500, and 1000 kHz. The body contact points with the electrodes were previously cleaned with an electrolytic fabric recommended by the manufacturer and participants were told to comply with the following preparation standards: to be fasting for at least 4 hours, not consuming alcohol the 48 hours before testing, do not perform moderate to high-intensity exercise within 12 hours before evaluation, do not perform the exam in the presence of a feverish state of dehydration, do not use metal parts or dental implants (when possible to remove) and don't ingest coffee. As a result of bioimpedance, the following variables were determined: total body mass (kg), body fat mass (kg), skeletal muscle mass (kg), visceral fat area (cm^2), and body mass index (kg/m^2). Age (years), height (cm), waist circumference (cm), and hip circumference (cm) were collected from the base of the clinic system medical records.

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2.2.2. Laboratory biochemical variables

The collected biochemical markers were HOMA-IR, insulin, fasting blood glucose, total cholesterol, HDL cholesterol, and triglycerides. Total cholesterol, HDL, and triglycerides were quantified in the serum by the coloring system. The values of the methodology applied by the laboratory were considered as references, which are based on the values presented by the Brazilian Diabetes Society and the Brazilian Society of Cardiology (19). All data were collected from the clinic system of medical records, preoperatively.

2.2.3. Analysis of Heart Rate Variability

For cardiac beats, a heart rate monitor (V800 Polar Heart Rate Monitor®) was used, calculated through the ratio between the RR interval, and transferred to a computer program to analyze HRV through the Polar Precision Performance which was imported into the Kubios HRV software (version 2.0), used to calculate linear time and frequency domain methods. For the analysis of HRV in the time domain, the square root of the average of square differences between the normal RR intervals (RMSSD) and the standard deviation of the average of all normal RR intervals (SDNN) was used. For HRV analysis in the frequency domain, low-frequency spectral components (LF, 0.04-015 Hz) and high frequency (HF, 0.15 to 0.40 Hz) were used in normal units (LFun and HFun, respectively), which represents a value for each spectral component to the total power minus the very low-frequency components (VLF), and the relationship between these components (LF/HF ratio).

Spectral analysis was calculated using the Fast Fourier Transform algorithm. The sample participants were invited to remain at rest, in the supine position, without exposure to excessive light, and in a no-noise environment for 10 minutes to analyze a 5-minute cutting point, checking in the preoperative period.

2.2.4. Obstructive sleep apnea

Polysomnography data were obtained through computed equipment from Respironics (Healthdyne Alice System 4), and the report was reviewed, independently, by trained experts. A third expert would be consulted in case of inconsistencies in the final report. The exam was conducted all night, in spontaneous sleep, without any sedation or sleep deprivation. It was recorded: electroencephalogram (electrodes C3, C4), Oculogram (O1, O2), electromyogram (electrodes in the Mentonian, Submention, and MMII), electrocardiogram, airflow (nasal and oral thermistor), respiratory effort (thoracic and abdominal strap), snoring (microphone on the chin) and body position (sensor in the thoracic strap)(20).

Oxyhemoglobin saturation was measured through pulse oximetry. Respiratory events were thus defined: Apnea, such as airflow interruption for 10 seconds or more, and hypopneas, such as the 50% or more reduction of inspiratory airflow per period ≥ 10 seconds, associated with a decrease than 3% in oxyhemoglobin saturation and/or a micro awakening.

Mixed apneas were included in the AHI and defined as those that had the absence of respiratory effort at the beginning of the period, followed by a gradual increase. The AHI was obtained through polysomnographic examination, dividing the total respiratory events by sleep hours. Patients were classified according to AHI: without apnea - less than 5.0 events/sleep hour; with light apnea - between 5.0 and 14.9 events/sleep time; with moderate apnea - between 15.0 and 30.0 events per/hour of sleep and severe apnea - over 30.0 events/sleep time.

2.3. Statistical Plan

Descriptive and analytical analyzes were performed through the Statistical Package for Social Sciences Program software, Version 14.0 for Windows (SPSS Inc, Chicago, IL). Comparisons between Normotensive patients with SAH were conducted based on clinical diagnosis. The normality of the variables was verified through descriptive statistics and

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the Kolmogorov-Smirnov test. The categorical variables were expressed in absolute values and percentages and the chi-square test was used to test the differences between the qualitative variables. Continuous variables with normal distribution were expressed as median and standard deviation and non-normal distribution as median and interquartile range. Test-T for independent samples or the Mann-Whitney U test was used to test the differences between the quantitative variables. Multivariate models were used to estimate the association between SAH with body composition and laboratory markers. For the elaboration of the adjustment models, the variables that presented p <0.2 were considered. The odds ratio has been adjusted to age, sex, height, and oxyhemoglobin saturation. Receiver Operating Characteristic Curve (ROC Curve) were used to estimate the sensitivity and specificity between systemic arterial hypertension and abdominal visceral fat area and fasting plasma glucose, as well as their respective cutting points.

3. Results

A total of 95 participants of both sexes were selected for the study. NG consisted of 57 participants (60%), and HG of 38 participants (40%), with an average age of 36.3 ± 10.1 and 40.4 ± 10.6 years, respectively (p = 0.062). Women represented 73.7% in NG and 57.9% in HG. Table 1 presents the characteristics of patients according to the clinical diagnosis of SAH, categorized as normotensive and hypertensive. In comparison to normotensive patients, hypertensive patients had higher body mass, BMI, WC, BFM, and VFA. The percentage of patients diagnosed with insulin resistance was higher in the hypertensive group. The groups were homogeneous regarding laboratory data, polysomnography, and the severity of OSAS and HRV parameters.

Table 1. Characteristics of patients according to the diagnosis of systemic arterial hypertension.

	Normotensive (n = 57)	Hypertensive (n = 38)	p-value
Age (years)	36.3 (10.1)	40.4 (10.6)	0.062
Gender n (%)			
Male	15 (26.3)	16 (42.1)	0.123
Women	42 (73.7)	22 (57.9)	
Body composition			
Body mass (kg)	113.3 (18.5)	124.6 (25)	**0.013**
Height (cm)	166.8 (8.3)	169.6 (8.4)	0.114
BMI (kg/m²)	40.5 (4.6)	42.9 (6)	**0.027**
WC (cm)	117.3 (12.1)	124.6 (18.3)	**0.024**
SMM (kg)	32.7 (7.4)	35.8 (7.6)	0.058
BFM (kg)	55.1 (9.2)	60.7 (14.9)	**0.027**
VFA (cm²)	202.8 (54.1)	262.2 (78.6)	**0.0001**
Laboratory Data			
Total cholesterol (mg/dl)	197 (57)	201 (36.7)	0.657
HDL (mg/dl)	49.5 (11.8)	48.3 (11.5)	0.636
Triglycerides (mg/dl)	146 (74)	175.7 (113.4)	0.162
Fasting blood glucose (mg/dl)	96.9 (35.4)	105.5 (26.2)	0.180
Homa-IR	5 (3.9)	6.4 (3.9)	0.135
Comorbidities n (%)			
Diabetes Mellitus	5 (8.8)	6 (15.8)	0.338
Insulin Resistance	30 (57.7)	28 (82.4)	**0.020**
OSAS	34 (65.4)	24 (72.7)	0.633
Polysomnography measures			
AHI (eventos/h)	8.4 [3.8 – 15.7]	9.9 [4 – 17.5]	0.389
OSAS frequency	19 [13.7 – 27.5]	18 [12 – 29.2]	0.754
OS (%)	95 [93 – 96]	94 [92 – 95.7]	0.174

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OSAS severity n (%)			
< 5 events/h	18 (34.6)	9 (27.3)	
5 – 30 events/h	31 (59.6)	20 60.6)	0.513
> 30 events/h	3 (5.8)	4 (12.1)	
HRV Parameters			
Time-domain			
Average RR	760.4 [638–849]	726 [647–814]	0.660
SDNN (ms)	70.7 [37.7–274]	84 [43.7–422]	0.522
RMSSD (ms)	64 [21.6–340]	117 [33.8–550]	0.373
pNN50 (ms)	18.4 [1.8–50]	13.6 [3.4–40.4]	0.861
Frequency domain			
LF (ms^2)	56 [41–75]	51 [27–84.8]	0.601
HF (ms^2)	43.6 [23.6–56]	46 [15.2–64.8]	0.443
LF/HF	1.3 [0.74–3.8]	1.4 [0.54–8.5]	0.898

*BMI, Body mass index; WC, Waist Circumference; SMM, Skeletal Muscle Mass; BFM, Body Fat Mass; VFA, Visceral Fat Area; HDL, High-Density Lipoprotein; AHI, Apnea-Hypopnea Index; OS, Oxyhemoglobin Saturation; Chi-square test was used to analyze diabetes mellitus, insulin resistance, and OSAS; Values presented as mean (standard deviation) or median [interquartile range].

Table 2 presents significant associations ($p<0.05$) of SAH with measures of body composition, laboratory data, and comorbidities, through unadjusted and adjusted multivariate analyzes. The variables body mass, SMM, BMI, WC, triglycerides, HOMA-IR, and insulin resistance showed no statistical differences in multivariate logistic regression analysis. In the final analysis model, after covariable adjustments, including age, gender, stature and oxyhemoglobin saturation, the association between SAH and body composition was (OR = 0.98, 95% confidence interval (CI) = 0.97-0.99) for area of visceral fat and laboratory markers (or = 0.96, 95% ci = 0.92-0.99) for fasting plasma glucose. Both variables proved to be the only ones independently associated with has.

Table 2. Multivariate logistic regression model of the variables of body fat, laboratories, and comorbidity among obese with and without SAH.

Variáveis	**Initial Model**				**Final Model***	
	β	**OR (95% IC)**	***P***	**β**	**OR (95% IC)**	***P***
Body mass	-0.089	0.91 (0.75 – 1.10)	0.362	-	-	-
Visceral Fat Area	-0.015	0.98 (0.97 – 0.99)	**0.011**	-0.014	0.98 (0.97 – 0.99)	0.026
SMM	0.154	1.16 (0.84 – 1.61)	0.356	-	-	-
BMI	-0.026	0.97 (0.76 – 1.25)	0.842	-	-	-
WC	0.005	1.00 (0.94 – 1.06)	0.882	-	-	-
Triglycerides	-0.001	0.99 (0.99 – 1.00)	0.684	-	-	-
Fasting blood glucose	-0.035	0.96 (0.93 – 0.99)	**0.043**	-0.040	0.96 (0.92 – 0.99)	0.047
Homa - IR	0.017	1.07 (0.86 – 1.19)	0.836	-	-	-
Insulin Resistance	-1.167	0.31 (0.08 – 1.21)	0.093	-	-	-

*The final model includes age, sex, height, and oxyhemoglobin saturation.

Figures 1 and 2 present the data related to the sensitivity and specificity of SAH with the visceral fat and fasting plasma glucose, respectively, in addition to its cutting points for screening of SAH. The visceral fat area had an area under the curve = 0.728 (95% ci = 0.620-0.836) and Cutoff Point for SAH: > 220.3 cm^2, while fasting plasma glucose presented an area under the curve = 0.693 (95% CI = 0.582-0.804) and cutoff point for SAH: > 95 mg/dl. All sensitivity and specificity values are presented in tables 1 and 2 for the visceral fat area and fasting plasma glucose respectively.

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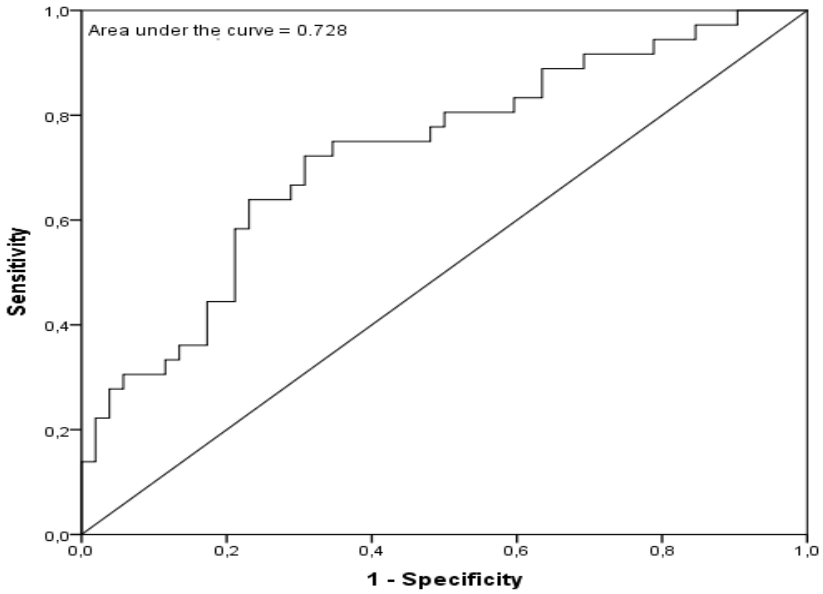

Figure 1. ROC curve for the visceral fat area as screening for SAH.

Area under the curve = 0.728; IC 95% (0.620 – 0.836).

Cutoff point for SAH: > 220.3 cm^2.

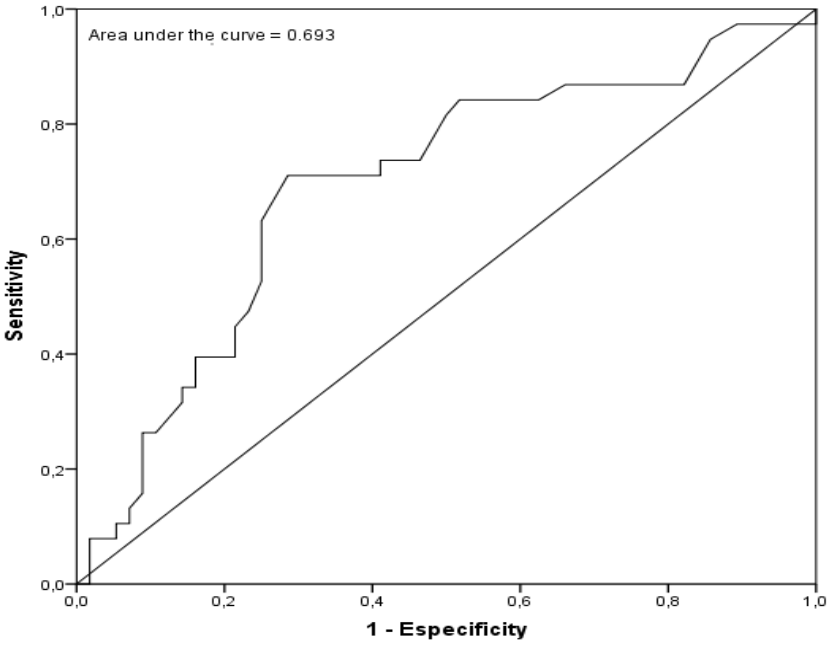

Figure 2. ROC curve for fasting blood glucose screening for SAH.

Area under the curve = 0.693; CI 95% (0.582 – 0.804);

Cutoff point for SAH: > 95 mg/dl

4. Discussion

In the present study, comparative analyzes between the groups showed that body composition measures, laboratory data, and comorbidity were higher in the HG. However, the only variables independently associated with SAH were visceral fat area and fasting plasma glucose. The forces of these associations described in the unadjusted analyzes were slightly changed after adjustments to potential confusion variables. These results provide additional support on the importance of maintaining low abdominal

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visceral fat storage levels, as well as the control of fasting plasma glucose as potential SAH protection factors.

Body composition measures, biochemical markers, and comorbidities evaluated in this study impact differently in the mechanisms related to SAH. Pre-studies corroborate our findings. In this sense, Chandra et al. demonstrated that higher measures of BMI were significantly associated with SAH in participants (21). Still in this sense, Lee et al. found that at each 1kg/m² increase in BMI there was also a 19% increase in the risk of arterial hypertension. (22) and Holmes et al. showed that for each increase in 1kg/m² in IMC the systolic blood pressure increased by 0.70mmHg (23). A likely explanation for the association of IMC measure with SAH, knowing that it is an index with cutting points for the obesity classification with good accuracy in prediction (24), is the fact that the obese phenotype, even when metabolically healthy, is directly linked to an increased risk to hypertension (25,26), since there are pathological mechanisms such as hyperinsulinemia, stimulation of sympathetic nervous system and abnormal levels of adipocytokines that affect vascular endothelium, responsible for maintaining vascular homeostasis (14).

Still dealing with measures of body composition, in our findings, the waist circumference also demonstrated association with SAH, as well as in the study by Guilherme et al., which demonstrated that in Brazilian adolescents the WC obtained a positive association as an independent anthropometric indicator for SAH, and those classified with central obesity were 130% more likely to have high blood pressure compared to adolescents without the diagnosis of abdominal obesity (27). Carba et al. found that at each 1cm increase in WC the chances of hypertension increased by 5% for women without overweight and 3% for overweight women (28). Since WC is an indicator of abdominal obesity (29), it can be said that a possible explanation for the association of WC with has is related to excess fat deposits in this region, since visceral adipose tissue plays an important role in activating the renin-angiotensin-aldosterone system, which can influence central and systemic hemodynamics (15).

As we can see, the change of a normotensive phenotype to hypertensive involves different factors. In addition to the variables already mentioned, BFM also interferes with hemodynamics so that fat distribution can dictate the risk of cardiovascular disease (30). In this way, Han et al. found that compared to normotensive individuals, the percentage of body fat was significantly higher in the hypertensive group (31). Park et al. also demonstrated that individuals with a high percentage of body fat were associated with an increased risk of hypertension even with low BMI, WC, or waist-hip ratio, and the increased risk was proportional to the increased percentage (32). In this case, by increasing BFM, levels in the plasma of inflammatory biomarkers such as C-reactive protein and interleukins may also increase, which consequently may predispose to the development of cardiovascular disease, including hypertension (16).

As mentioned earlier, fat distribution can dictate the risk of cardiovascular disease and, in this sense, individuals with higher visceral adipose tissue and ectopic fat deposits have an even greater prevalence of metabolic disorders such as hypertension (33,34). In figures 1 and 2 can be visualized the area under the ROC curve for sensitivity and specificity for the visceral fat area and fasting plasma glucose found in our study, demonstrating that both variables obtained independent associations with SAH, highlighting mainly the area of visceral fat. Excessive visceral adipose tissue performs the secretion of hormones and molecules that accentuate cardiovascular disease and also becomes resistant to insulin and leptin and may contribute to vascular resistance and sympathetic system dysfunction (33). Intra-abdominal adipose tissue at high levels can be considered as part of a phenotype whose result is associated with a dysfunctional alteration of subcutaneous adipose tissue and ectopic storage of triglyceride, leading to this morphological change to be part of a set of cardiometabolic risk factors (35).

Once insulin resistance can contribute to vascular resistance and sympathetic system dysfunction (33), it is important to highlight its relationship with fasting plasma glucose levels, since as fasting blood glucose increases, insulin sensitivity rate decreases. (36). In a study conducted in Japan, it was observed that high fasting glucose levels were

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independently and significantly associated with hypertension, and the risk rate in participants with glucose above or equal to 7.0 mmol/L was 1.79 compared to participants with glycemia rate above 5.6 mmol/l (17). These results corroborate our findings, as fasting plasma glucose has been independently associated with SAH.

Fasting plasma glycemia associations and abdominal visceral fat with SAH described in the present study have potential implications for treatment interventions to improve results in obese patients and are probably generalizable for populations worldwide. However, there are limitations to determine whether statistical associations are causal, and the direction of associations should be taken into consideration before definitive conclusions are reached. Since the study is observational, it is not possible to rule out the effects of residual or unsuccessful confusion as an explanation for the results. In addition, the transverse design does not allow to determine whether the clinical picture of SAH preceded or was influenced by the metabolic and morphological profile. The observed SAH associations with biochemical and body markers may be bidirectional.

5. Conclusion

In conclusion, the present study expands previous observations about SAH associations with biochemical markers and body composition, showing independent SAH associations and having as lower protective factors fasting plasma glucose concentrations and abdominal visceral fat in obese patients. The results draw attention to the importance of interventions to improve the control of biochemical and body composition variables, prevent changes in plasma blood glucose and attenuate increased abdominal visceral fat in obese patients.

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Quadro 1. Sensitivity/specificity statistics for the visceral fat area.

Criterion	Sensitivity	95% CI	Specificity	95% CI	+LR	-LR
≥64.8	100.00	90.7 - 100.0	0.00	0.0 - 6.3	1.00	-
>145.3	100.00	90.7 - 100.0	8.77	2.9 - 19.3	1.10	0.00
>145.7	97.37	86.2 - 99.9	8.77	2.9 - 19.3	1.07	0.30
>155.4	97.37	86.2 - 99.9	14.04	6.3 - 25.8	1.13	0.19
>156	94.74	82.3 - 99.4	14.04	6.3 - 25.8	1.10	0.38
>161.4	94.74	82.3 - 99.4	19.30	10.0 - 31.9	1.17	0.27
>163.2	92.11	78.6 - 98.3	19.30	10.0 - 31.9	1.14	0.41
>171.8	92.11	78.6 - 98.3	28.07	17.0 - 41.5	1.28	0.28
>175.6	89.47	75.2 - 97.1	28.07	17.0 - 41.5	1.24	0.37
>179.7	89.47	75.2 - 97.1	33.33	21.4 - 47.1	1.34	0.32
>181.2	84.21	68.7 - 94.0	33.33	21.4 - 47.1	1.26	0.47
>186.6	84.21	68.7 - 94.0	36.84	24.4 - 50.7	1.33	0.43
>187	81.58	65.7 - 92.3	36.84	24.4 - 50.7	1.29	0.50
>190	81.58	65.7 - 92.3	45.61	32.4 - 59.3	1.50	0.40
>191.3	78.95	62.7 - 90.4	45.61	32.4 - 59.3	1.45	0.46
>194.4	78.95	62.7 - 90.4	47.37	34.0 - 61.0	1.50	0.44
>195.7	76.32	59.8 - 88.6	47.37	34.0 - 61.0	1.45	0.50
>212.9	76.32	59.8 - 88.6	59.65	45.8 - 72.4	1.89	0.40
>216.1	73.68	56.9 - 86.6	59.65	45.8 - 72.4	1.83	0.44
>220.3	73.68	56.9 - 86.6	63.16	49.3 - 75.6	2.00	0.42
>225.3	68.42	51.3 - 82.5	63.16	49.3 - 75.6	1.86	0.50
>229.5	68.42	51.3 - 82.5	64.91	51.1 - 77.1	1.95	0.49
>231.2	65.79	48.6 - 80.4	64.91	51.1 - 77.1	1.88	0.53
>234.2	65.79	48.6 - 80.4	70.18	56.6 - 81.6	2.21	0.49
>244.7	60.53	43.4 - 76.0	70.18	56.6 - 81.6	2.03	0.56
>247.5	60.53	43.4 - 76.0	71.93	58.5 - 83.0	2.16	0.55
>254.9	47.37	31.0 - 64.2	71.93	58.5 - 83.0	1.69	0.73
>259.3	47.37	31.0 - 64.2	75.44	62.2 - 85.9	1.93	0.70
>264.3	39.47	24.0 - 56.6	75.44	62.2 - 85.9	1.61	0.80
>272.3	39.47	24.0 - 56.6	78.95	66.1 - 88.6	1.88	0.77
>274.6	36.84	21.8 - 54.0	78.95	66.1 - 88.6	1.75	0.80
>278.9	36.84	21.8 - 54.0	80.70	68.1 - 90.0	1.91	0.78
>281.4	34.21	19.6 - 51.4	80.70	68.1 - 90.0	1.77	0.82
>283.9	34.21	19.6 - 51.4	85.96	74.2 - 93.7	2.44	0.77
>286.6	31.58	17.5 - 48.7	85.96	74.2 - 93.7	2.25	0.80
>289.3	31.58	17.5 - 48.7	87.72	76.3 - 94.9	2.57	0.78
>298.8	26.32	13.4 - 43.1	87.72	76.3 - 94.9	2.14	0.84
>310.9	26.32	13.4 - 43.1	89.47	78.5 - 96.0	2.50	0.82
>327.1	18.42	7.7 - 34.3	89.47	78.5 - 96.0	1.75	0.91
>328.6	18.42	7.7 - 34.3	91.23	80.7 - 97.1	2.10	0.89
>487.5	5.26	0.6 - 17.7	91.23	80.7 - 97.1	0.60	1.04
>9999	0.00	0.0 - 9.3	100.00	93.7 - 100.0	-	1.00

LR+ and LR-: positive and negative likelihood ratios, respectively.

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Quadro 2. Sensitivity/specificity statistics for the fasting blood glucose.

Criterion	Sensitivity	95% CI	Specificity	95% CI	+LR	-LR
≥66	100.00	90.7 - 100.0	0.00	0.0 - 6.4	1.00	-
>66	97.37	86.2 - 99.9	0.00	0.0 - 6.4	0.97	-
>79	97.37	86.2 - 99.9	10.71	4.0 - 21.9	1.09	0.25
>81	94.74	82.3 - 99.4	14.29	6.4 - 26.2	1.11	0.37
>82	86.84	71.9 - 95.6	17.86	8.9 - 30.4	1.06	0.74
>85	86.84	71.9 - 95.6	33.93	21.8 - 47.8	1.31	0.39
>86	84.21	68.7 - 94.0	37.50	24.9 - 51.5	1.35	0.42
>89	84.21	68.7 - 94.0	48.21	34.7 - 62.0	1.63	0.33
>90	81.58	65.7 - 92.3	50.00	36.3 - 63.7	1.63	0.37
>91	73.68	56.9 - 86.6	53.57	39.7 - 67.0	1.59	0.49
>93	73.68	56.9 - 86.6	58.93	45.0 - 71.9	1.79	0.45
>93.5	71.05	54.1 - 84.6	58.93	45.0 - 71.9	1.73	0.49
>95	71.05	54.1 - 84.6	71.43	57.8 - 82.7	2.49	0.41
>96	63.16	46.0 - 78.2	75.00	61.6 - 85.6	2.53	0.49
>97.9	52.63	35.8 - 69.0	75.00	61.6 - 85.6	2.11	0.63
>98	47.37	31.0 - 64.2	76.79	63.6 - 87.0	2.04	0.69
>99	44.74	28.6 - 61.7	78.57	65.6 - 88.4	2.09	0.70
>101	39.47	24.0 - 56.6	78.57	65.6 - 88.4	1.84	0.77
>102	39.47	24.0 - 56.6	83.93	71.7 - 92.4	2.46	0.72
>103	34.21	19.6 - 51.4	83.93	71.7 - 92.4	2.13	0.78
>105	34.21	19.6 - 51.4	85.71	73.8 - 93.6	2.39	0.77
>106	31.58	17.5 - 48.7	85.71	73.8 - 93.6	2.21	0.80
>107	28.95	15.4 - 45.9	87.50	75.9 - 94.8	2.32	0.81
>110	26.32	13.4 - 43.1	89.29	78.1 - 96.0	2.46	0.83
>113	26.32	13.4 - 43.1	91.07	80.4 - 97.0	2.95	0.81
>121	15.79	6.0 - 31.3	91.07	80.4 - 97.0	1.77	0.92
>124	13.16	4.4 - 28.1	92.86	82.7 - 98.0	1.84	0.94
>125	10.53	2.9 - 24.8	92.86	82.7 - 98.0	1.47	0.96
>126	10.53	2.9 - 24.8	94.64	85.1 - 98.9	1.96	0.95
>130	7.89	1.7 - 21.4	94.64	85.1 - 98.9	1.47	0.97
>139	7.89	1.7 - 21.4	98.21	90.4 - 100.0	4.42	0.94
>220	0.00	0.0 - 9.3	98.21	90.4 - 100.0	0.00	1.02
>337	0.00	0.0 - 9.3	100.00	93.6 - 100.0	-	1.00

LR+ and LR-: positive and negative likelihood ratios, respectively.