

Relationship between pediatric visceral adiposity index and cardiometabolic risk in Brazilian adolescents

Relação do índice de adiposidade visceral pediátrico com o risco cardiometabólico em adolescentes brasileiros

Petrina Rezende de Souza¹, João Victor Mota Pereira¹, Nathália Nogueira Leite², Patrícia Feliciano Pereira³

ABSTRACT

Introduction: Visceral obesity is a global challenge and is linked to significant metabolic complications. This condition is known to be observed during childhood and adolescence but may extend into adulthood. Thus, establishing measures for early identification of individuals at risk is of great interest in public health. **Objective:** To evaluate the association of the Pediatric Visceral Adiposity Index (Pediatric VAI) with cardiometabolic markers in adolescents. **Methods:** This is a cross-sectional study conducted with 671 adolescents aged 10 to 19 years (n=353 girls) from public and private schools in the city of Viçosa, Minas Gerais. A questionnaire was administered to assess sociodemographic and behavioral variables. Anthropometric assessments (weight, height, BMI, and waist circumference) and body composition, using dual-energy X-ray absorptiometry (DEXA), were performed. The cardiometabolic parameters evaluated were glucose, insulin, insulin resistance (HOMA-IR), total cholesterol and its fractions (LDL and HDL), triglycerides, uric acid, and leukocytes. Systolic (SBP) and diastolic (DBP) blood pressure were measured. Multivariate linear regression analyses, stratified by sex, were performed to assess the predictive capacity of metabolic alterations by the Pediatric VAI. The study was approved by the Human Research Ethics Committee of the Federal University of Viçosa (Ref. No. 674.045/2014). **Results:** Pediatric VAI showed a positive linear correlation with glucose, insulin, HOMA-IR, uric acid, leukocytes, SBP, and DBP for both sexes ($p < 0.05$). Additionally, Pediatric VAI values were significantly higher in those with a greater number of alterations in the components of metabolic syndrome compared to those without alterations ($p < 0.001$). Pediatric VAI was associated with insulin resistance ($\beta = 0.2060017$; $p < 0.001$) and uric acid ($\beta = 0.0959677$; $p = 0.026$) in girls, and with uric acid ($\beta = 0.1006545$; $p < 0.001$) in boys. **Conclusion:** Pediatric VAI is associated with cardiometabolic markers in adolescents. Further studies should be conducted to clarify its utility in pediatric clinical practice.

Keywords: Visceral adiposity index; Abdominal obesity; Metabolic syndrome; Adolescence; Pediatric visceral obesity.

¹ Graduação em Medicina. Departamento de Medicina e Enfermagem, Universidade Federal de Viçosa (UFV), Viçosa, Minas Gerais, Brazil.

² Programa de Pós-Graduação em Microbiologia Agrícola, Universidade Federal de Viçosa (UFV), Viçosa, Minas Gerais, Brazil.

³ Departamento de Nutrição e Saúde, Universidade Federal de Viçosa (UFV), Viçosa, Minas Gerais, Brazil.

Associate Editor Responsible:

Cássio da Cunha Ibiapina
Faculdade de Medicina da Universidade Federal de Minas Gerais, Brasil.
Belo Horizonte/MG, Brazil.

Corresponding Author:

Petrina Rezende de Souza
E-mail: petrina.souza@ufv.br

Supporting Sources:

CAPES - Coordenação de Aperfeiçoamento de Pessoal de Nível Superior.
CNPq - Conselho Nacional de Desenvolvimento Científico e Tecnológico.

Conflicts of Interests:

None

RESUMO

Introdução: A obesidade visceral é um desafio enfrentado em todo o mundo e está relacionada com importantes complicações metabólicas. Sabe-se que essa condição é observada durante a infância e a adolescência, no entanto, pode se estender até a vida adulta. Sendo assim, estabelecer medidas para identificação precoce de indivíduos em risco é de grande interesse no âmbito da saúde pública. **Objetivo:** Avaliar a associação do Índice de Adiposidade Visceral Pediátrico (IAV Pediátrico) com marcadores cardiometabólicos em adolescentes. **Métodos:** Trata-se de um estudo transversal realizado com 671 adolescentes de 10 a 19 anos ($n=353$ meninas), das escolas públicas e privadas do município de Viçosa, Minas Gerais. Foi aplicado um questionário para verificar variáveis sociodemográficas e comportamentais. Foi feita avaliação antropométrica (peso, altura, IMC e perímetro da cintura) e da composição corporal pela absorciometria de raios-x de dupla energia (DEXA). Os parâmetros cardiometabólicos avaliados foram glicemia, insulina, resistência à insulina (HOMA-IR), colesterol total e frações (LDL e HDL), triglicerídeos, ácido úrico e leucócitos. Foi aferida a pressão arterial sistólica (PAS) e diastólica (PAD). Análises de regressão linear multivariada, específicas por sexo, foram realizadas para avaliar a capacidade de predição de alterações metabólicas pelo IAV pediátrico. O estudo foi aprovado pelo Comitê de Ética em Pesquisas com Seres Humanos da Universidade Federal de Viçosa (Ref. Nº 674.045 / 2014). **Resultados:** O IAV Pediátrico apresentou correlação linear positiva com glicemia, insulina, HOMA-IR, ácido úrico, leucócitos, PAS e PAD para ambos os sexos ($p<0,05$). Ainda, os valores do IAV Pediátrico foram significativamente maiores naqueles com maior número de alterações nos componentes da síndrome metabólica, em comparação àqueles sem alteração ($p<0,001$). O IAV foi associado à resistência à insulina ($\beta = 0,2060017$; $p<0,001$) e ácido úrico ($\beta = 0,0959677$; $p=0,026$) nas meninas e ao ácido úrico ($\beta = 0,1006545$; $p<0,001$) nos meninos. **Conclusão:** O IAV pediátrico está associado a marcadores cardiometabólicos em adolescentes. Mais estudos deveriam ser realizados a fim de elucidar sua utilidade na prática clínica pediátrica.

Palavras-chave: Índice de adiposidade visceral; Obesidade abdominal; Síndrome metabólica; Adolescência; Obesidade visceral pediátrica.

Ethics Committee:

O estudo foi aprovado pelo Comitê de Ética em Pesquisas com Seres Humanos da Universidade Federal de Viçosa.

Número do parecer - Ref. Nº 674.045 / 2014.

Received on: December 13rd, 2024

Approved on: August 17th, 2025

Publication Date: January 9th, 2026.

DOI: 10.5935/2238-3182.2025e35118-en

INTRODUCTION

Obesity is defined as the abnormal or excessive accumulation of fat that poses health risks¹. Global estimates reveal that more than 340 million children and adolescents aged 5 to 19 are overweight or obese². This condition is concerning, especially among individuals in early age groups with fat accumulation, who tend to remain in this state into adulthood^{3,4}, thereby increasing cardiovascular disease (CVD) mortality at that stage⁵.

Increasing attention has been given to the regional distribution of body fat, which is as important as or more important than the quantity of fat regarding the risk of metabolic complications⁶⁻¹⁰, such as the inflammatory process clinically known as atherosclerosis¹¹. Studies indicate that atherosclerosis can begin in childhood and adolescence since obesity and other components of metabolic syndrome occur during these stages^{12,13}.

Central obesity plays an important role in increasing the production of pro-inflammatory and atherogenic

substances¹⁴. It is known that adipose tissue is an active endocrine organ that secretes various cellular factors and mediators, such as C-reactive protein (CRP), measured using high-sensitivity CRP (hs-CRP), which detects low serum levels. CRP functions not only as an inflammatory marker but also as a molecule found and produced within the atherosclerotic plaque, playing a key role in its vulnerability and, consequently, being responsible for coronary events. Other secretions include interleukin 6, which primarily stimulates CRP production, tumor necrosis factor, plasminogen activator inhibitor-1 (PAI-1)¹⁵, a soluble adrenal lipid factor secretagogue¹⁶, and angiotensinogen¹⁷. The product of the latter's activation, angiotensin II, stimulates prostacyclin production and release, acting as a potent adipogenic signal promoting the final differentiation of adipocytes¹⁷.

Given this scenario, the demand for tools to analyze excess weight, particularly in the abdominal region, and the risk of associated morbidities has grown. Recently, the Visceral Adiposity Index (VAI) was proposed to assess visceral adipose tissue distribution and dysfunction, based on anthropometric (BMI and WC) and functional markers (triglycerides and high-density lipoprotein)¹⁸. In the last 10 years, several publications have demonstrated the VAI's ability to express possible adipose tissue dysfunction and its associated cardiometabolic risk, especially for applications in specific populations, across different age groups, from children to the elderly¹⁹⁻²³. Moreover, the VAI's association with serum adipokines, insulin resistance, and glucose metabolism disorders in adults has been reported^{24,25}. Particularly in children and adolescents, studies using VAI are still scarce^{19,26-30}. Additionally, the current version of the index is unsuitable for pediatric populations, as it is based on observations of healthy Caucasian adults²⁴. In this context, the VAI was adapted for sex-specific pediatric populations. However, many gaps remain regarding the index's ability to predict cardiometabolic risk in early life stages.

Recognizing the significant influence of visceral fat in triggering various metabolic complications, studies focused on investigating simple and low-cost tools for identifying at-risk individuals are essential. Therefore, VAI is considered a promising tool for identifying at-risk individuals in early age groups. This study aims to investigate the association between Pediatric VAI and insulin resistance, subclinical inflammation, metabolic and hormonal profiles in adolescents.

METHODS

This is a cross-sectional study conducted with adolescents aged 10 to 19 years, of both sexes, selected by simple random sampling from all public and private schools in the rural and urban areas of Viçosa, a city in the state of Minas Gerais, Brazil. This study is part of the project "Comparative study between the three phases of adolescence, concerning excess body fat and cardiovascular risk factors for metabolic syndrome," whose data collection took place between 2010 and 2013³¹.

The sample size was calculated using Epi Info StatCalc software, version 6.04, with a specific formula for cross-sectional studies, considering the total population of 11,898 adolescents in Viçosa/MG³², an expected prevalence of 50% as the study considered multiple cardiovascular risk factors, and an acceptable variability of 5%, totaling a minimum sample of 372 adolescents with a 95% confidence level.

The study included adolescents who were not regular users of medications affecting glucose, insulin, lipid metabolism, blood pressure, inflammatory, or hormonal markers, non-smokers, not using contraceptives, not participating in weight reduction/control programs, and not pregnant or with a history of pregnancy. Adolescents with diagnosed infections, acute inflammation, or chronic diseases were also excluded. Adolescents who did not provide the signed assent and/or informed consent form, as required by ethical standards, were also excluded from the study.

STUDY VARIABLES

SOCIODEMOGRAPHIC VARIABLES

Sociodemographic data were collected through structured questionnaires. Participant information included age, sex, school type (public or private), area of residence (urban or rural), and socioeconomic status. The latter was assessed using the same methodology adopted by the Living Standards Survey²⁸ and classified as "adequate" or "precarious and intermediate".

BEHAVIORAL VARIABLES

Physical activity levels were assessed using the International Physical Activity Questionnaire (IPAQ), short version²⁹, validated for this population³⁰. Adolescents were classified as "sedentary," "irregularly active," and "active and very active." Due to the low prevalence of sedentary individuals in this population (6.3%, n=42), the sedentary and irregularly active individuals were grouped as "insufficiently active," and those considered active and very active were grouped as "physically active"³¹.

Dietary habits were assessed through the application of a food frequency questionnaire (FFQ), applied individually, to report the frequency of food consumption over the previous month³². The weekly consumption frequency of food groups (e.g., sugars and sweets) was categorized as <5 or ≥5 times per week³³, which was used as an indicator of unhealthy consumption.

ANTHROPOMETRIC ASSESSMENT

Waist circumference (WC) was measured using a flexible, inelastic tape measure (Cardiomed[®], São Luiz, MA, Brazil). Weight was measured with participants barefoot, wearing light clothing, and no accessories, following standardized protocols recommended by the World Health Organization (WHO), using a portable digital scale (LC 200PP, Marte[®], São Paulo, Brazil) with a maximum capacity of 150 kg. Calibration was verified weekly using a standard weight.

Height was assessed in duplicate, with participants barefoot and standing upright, according to WHO guidelines, using a portable stadiometer (Altuxata[®], Belo Horizonte, Brazil).

Measurements were recorded to the nearest centimeter, with the cursor aligned to the highest point of the head. A maximum difference of 0.5 cm between the two readings was allowed, and the mean was used as the final value.

Body Mass Index (BMI) was calculated as weight (kg) divided by height squared (m^2). Nutritional status was classified using BMI-for-age z-scores, based on sex and age, according to WHO growth reference charts.

Waist circumference (WC) was measured with a flexible, non-elastic tape (Cardiomed[®], São Luiz, MA, Brazil) at the midpoint between the last rib and the iliac crest, at the end of a normal expiration. All measurements were taken in duplicate by a single trained evaluator, with participants standing and facing forward. Values were recorded to the nearest centimeter, with a maximum acceptable variation of 0.5 cm between the two readings, and the average was used for analysis.

ASSESSMENT OF CARDIOMETABOLIC AND INFLAMMATORY MARKERS

Between 7:00 AM and 9:00 AM, after 12 hours of fasting, 12 ml of blood was collected from the antecubital vein by venipuncture using disposable syringes. Serum was separated by centrifugation at 2225xg for 15 minutes at room temperature (2-3 Sigma, Sigma Laborzentrifugen, Osterodeam Harz, Germany) at the Clinical Analysis Laboratory of the Division of Health/UFV.

Blood glucose was measured by the glucose oxidase method using the Cobas Mira Plus equipment (Roche Diagnostics, GmbH, Montclair, NJ, USA). Fasting insulin was measured by electrochemiluminescence. Insulin resistance was calculated using HOMA-IR (Homeostasis Model Assessment - Insulin Resistance), employing fasting insulin and glucose levels: $HOMA-IR = [(fasting\ insulin\ (\mu U/mL) \times fasting\ glucose\ [mmol/L]) / 22.5]$. Insulin values $\geq 15 \mu U/mL$ and HOMA-IR ≥ 3.16 were considered hyperinsulinemia and insulin resistance, respectively³³.

HDL and triglycerides were measured by the enzymatic colorimetric method using the automatic Cobas Mira Plus equipment (Roche Corp.), and LDL was calculated using the Friedewald formula for triglyceride levels below 400 mg/dL. The lipid profile was classified according to the Expert Panel and Integrated Cardiovascular Health and Risk Reduction Guidelines for Children and Adolescents³³.

Elevated values were defined as: LDL ≥ 130 mg/dL, HDL cholesterol < 40 mg/dL, triglycerides ≥ 130 mg/dL.

Uric acid was measured by the enzymatic colorimetric method, with automation using Cobas Mira Plus (Roche Corp.). Leukocytes were measured in serum by the impedance method on a Coulter[®] T890 device with a Coulter[®] Beckman kit (USA, Brea, CA). For uric acid, values were considered elevated when greater than 5.5 mg/dL for girls and 6.0 mg/dL for boys.

BLOOD PRESSURE ASSESSMENT

Blood pressure was measured after a minimum rest period of 15 minutes using an automatic inflation blood pressure monitor (Omron[®] model HEM-741 CINT, Kyoto,

KYT, Japan) with an appropriately sized cuff for the arm circumference. The value was measured three times on the arm with the higher pressure, with a 1- minute interval between measurements, and the average of the last two measurements was used.

Elevated blood pressure was defined according to the American Academy of Pediatrics: < 13 years (≥ 90 th to < 95 th percentile or 120/80 mmHg to < 95 th percentile, whichever is lower), and ≥ 13 years 120/ < 80 to 129/ < 80 mmHg.

CALCULATION OF PEDIATRIC VISCERAL ADIPOSITY INDEX

The Pediatric Metabolic Index (PMI) was calculated according to sex and age, evaluated continuously as follows: Female (≥ 10 years): $\sqrt{((WC / (-0.02 * BMI^2 + 3.67 * BMI + 3.24)) * (TG / 1.04) * (1.34 / HDL))}$; Male (≥ 10 years): $\sqrt{((WC / (-0.02 * BMI^2 + 3.62 * BMI + 3.72)) * (TG / 1.06) * (1.30 / HDL))}$. The cutoff points and the pediatric VAI formula used in this study were based on the adaptations proposed by Ejtahed et al. (2019)²⁹.

STATISTICAL ANALYSIS

Results were presented as mean (standard deviation) for continuous variables with symmetric distribution and median (interquartile range) for those with asymmetric distribution. The normality of variables was assessed using the Shapiro- Wilk test.

The Student's t-test and Mann-Whitney test were used for comparisons between genders for anthropometric, cardiometabolic, inflammatory, and hormonal markers for symmetric and non-symmetric distribution variables, respectively.

The correlation of the Pediatric Visceral Adiposity Index (PVAI) with cardiometabolic, inflammatory, and hormonal markers was analyzed using Pearson or Spearman correlation coefficients, depending on the normality and homogeneity of quantitative variables.

The Kruskal-Wallis test and ANOVA were used to compare the PVAI according to the number of metabolic abnormalities for symmetric and non- symmetric distribution variables, respectively.

A multiple linear regression model was used, by sex, to verify the association of the PVAI with insulin resistance (HOMA), uric acid, and leukocytes (adjusted for age, unhealthy diet, physical activity level, and socioeconomic condition). Results were presented as adjusted coefficient of determination, β coefficient $\pm$ standard error, and p-value. Linearity and multicollinearity were evaluated using graphical methods (scatter plots) and tests (variance inflation factor). Residuals were tested for normality and homoscedasticity using graphical methods (scatter plots, box plots, and histograms) and statistical tests. The significance level was set at 5%. These analyses were performed using SPSS version 20.0 and STATA version 13.0.

RESULTS

In this study, among the 671 evaluated adolescents (356 girls), with a mean age of 14.3 (3) years, 14.3% (n=96)

were overweight (14.4% of girls and 14.2% of boys) and 5.8% (n=39) (5.1% of girls and 6.6% of boys) were obese. Considering the total sample, the mean Pediatric Visceral Adiposity Index was 0.88 (± 0.58). Additionally, 16.1% (n=109) had low HDL, 11% (n=74) had abdominal obesity, 7.6% (n=51) had elevated LDL, 6.4% (n=43) had hypertriglyceridemia, 4.6% (n=31) had hypertension, and 0.9% (n=6) had hyperglycemia.

Higher values of waist circumference, BMI, insulin, HOMA-IR, HDL, leukocytes, diastolic blood pressure (DBP), and lower values of systolic blood pressure (SBP) and

uric acid were observed in females (Table 1). The Pediatric Visceral Adiposity Index showed correlations with glucose, insulin, HOMA-IR, uric acid, leukocytes, SBP, and DBP for both sexes ($p < 0.05$) (Table 2).

Still, the IAV values were significantly higher in those with a greater number of alterations in the components of metabolic syndrome in both sexes (Table 3).

Table 4 presents the multiple linear regression analysis for the association between the pediatric visceral adiposity index (VAI) and insulin resistance (HOMA-IR), leukocytes, and uric acid, by sex. After controlling for age,

Table 1. Anthropometric and Body Composition, Biochemical, and Clinical Characteristics of Adolescents, According to Sex.

<i>Variables</i>	<i>Girls</i> (N=353)	<i>Boys</i> (N=318)	<i>p-value</i>
Age (Years)	14.45 (5.69)	13.34 (6.07)	0.152
Weight (Kg)	48.30 (14.33)	47.30 (24.95)	0.536
Height (m)	1.57 (0.14)	1.60 (0.267)	0.001
BMI (kg/m ²)	19.26 (4.57)	18.87 (4.45)	0.038
WC (cm)	71.00 (12.00)	70.00 (13.00)	0.007
VAI Pediatric	0.74 (0.54)	0.74 (0.50)	0.297
HOMA-IR	1.80 (1.21)	1.40 (1.02)	<0.001
LDL (mg/dL)	92.00 (34.00)	91.00 (29.00)	0.361
HDL (mg/dL)	52.00 (16.00)	48.00 (17.00)	<0.001
Triglycerides (mg/dL)	64.00 (37.00)	63.00 (35.00)	0.118
Uric Acid	2.90 (1.00)	3.40 (1.60)	<0.001
SBP (mmHg)	97.00 (11.00)	100.00 (16.50)	<0.001
DBP (mmHg)	61.00 (7.50)	58.50 (9.00)	<0.001
Leukocytes (/mm ³)	6000.00 (2300.00)	5500.00 (1600.00)	<0.001
Insulin (mcU/mL)	8.85 (5.60)	6.40 (5.20)	<0.001
Glucose (mg/dL)	84.35 (7.37)	85.72 (6.94)	0.094

Caption: WC = Waist Circumference; VAI = Visceral Adiposity Index; HOMA-IR = Homeostasis Model Assessment of Insulin Resistance; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure. Variables are presented as mean (standard deviation) or median (interquartile range). Student's t-test or Mann-Whitney test. Significance set at $p \leq 0.05$.

Table 2. Spearman correlation between pediatric visceral adiposity index and cardiometabolic markers in adolescents.

<i>Variables</i>	<i>Girls</i> (N=353)	<i>Boys</i> (N=318)
HOMA-IR	0.354	0.257
LDL (mg/dL)	0.243	0.084
Uric Acid (mg/dL)	0.178	0.201
SBP (mmHg)	-0.026	0.154
DBP (mmHg)	-0.005	0.012
Leukocytes (/mm ³)	0.100	0.154
Insulin (mcU/mL)	0.358	0.274
Glucose (mg/dL)	0.032	-0.013

Caption: WC = Waist circumference; VAI = Visceral adiposity index; HOMA = Homeostasis Model Assessment of Insulin Resistance; SBP = Systolic blood pressure; DBP = Diastolic blood pressure. Significance level set at $p \leq 0.05$.

income, physical activity level, and unhealthy diet, the VAI predicted insulin resistance ($\beta=0.2060017$; $p<0.001$) and uric acid ($\beta=0.0959677$; $p=0.026$) for girls, and uric acid ($\beta=0.1006545$; $p<0.001$) for boys.

DISCUSSION

The accumulation of body fat, particularly in the abdominal region, is one of the main risk factors for cardiometabolic diseases, which aligns with the primary finding of this study, where higher values of the pediatric VAI were positively associated with cardiometabolic risk factors (abdominal obesity, elevated LDL, hypertension, low HDL, insulin resistance, and elevated uric acid).

The distinction between the metabolic functions of subcutaneous and visceral obesity was proposed in the early 1980s³⁴. Visceral adipose tissue is stored around internal organs in the abdominal cavity and communicates with other organs through the synthesis and secretion of molecules known as adipokines³⁵. The accumulation of visceral adipose tissue and adipokine levels induce chronic inflammation and metabolic disturbances³³. Evidence indicates that visceral adipose tissue is metabolically more active and capable of predicting obesity-related morbidity

and mortality^{36,37}. As adipose tissue expands, lipolysis occurs, and adipocytes become hypoperfused, causing the release of mediators involved in inflammation and macrophage infiltration³⁸. These cells trigger and increase the state of chronic, systemic, low-grade inflammation. Therefore, signals generated by adipose tissue indicate endothelial dysfunction, which influences not only insulin action in skeletal muscle but also the activation of the acute phase in the liver. This increases the risk of metabolic disorders by reducing nitric oxide production and impairing endothelial-mediated vasodilation^{39,40}.

The VAI has been adopted as an important tool for identifying at-risk individuals by reflecting possible adipose tissue dysfunction and related metabolic disorders¹⁸. Available evidence has shown interesting results with predictive potential for various outcomes, such as polycystic ovary syndrome, metabolic syndrome, and its components²³. Thus, we emphasize that this study contributes to a growing body of evidence, considering the recent topic and the association of elevated VAI values with metabolic, inflammatory, and hormonal changes.

A study conducted by Koloverou et al. (2019)²² found an association between VAI and hepcidin, showing that higher levels of this hormone in adolescents with obesity

Table 3. Pediatric visceral adiposity index in adolescents and accumulation of metabolic alteration components, by sex.

<i>Number of Metabolic Alteration</i>	<i>Girls (N=353)</i> <i>VAI</i>	<i>Boys (N=318)</i> <i>VAI</i>	<i>p Value</i>
0	0.68 ± 0.40	0.68 ± 0.40	0.68 ± 0.40
	1.00 ± 0.89	1.00 ± 0.89	1.00 ± 0.89
	1.88 ± 0.80	1.88 ± 0.80	1.88 ± 0.80
1	0.68 ± 0.40	0.68 ± 0.40	0.68 ± 0.40
	1.00 ± 0.89	1.00 ± 0.89	1.00 ± 0.89
	1.88 ± 0.80	1.88 ± 0.80	1.88 ± 0.80
2 or more	0.68 ± 0.40	0.68 ± 0.40	0.68 ± 0.40
	1.00 ± 0.89	1.00 ± 0.89	1.00 ± 0.89
	1.88 ± 0.80	1.88 ± 0.80	1.88 ± 0.80

Caption: Significance level $p \leq 0.05$.

Results are presented as median ± interquartile range. Kruskal-Wallis test.

Table 4. Multiple linear regression analysis to assess the ability of the pediatric visceral adiposity index to predict metabolic alterations, by sex.

HOMA	0.1725	0.2060017	0.0316373	<0.001
Leukocytes	0.0797	0.0000313	0.0000175	0.074
Uric Acid	0.0844	0.0959677	0.0429030	0.026
<i>Boys</i>				
HOMA	0.0885	0.0641824	0.0327782	0.051
Leukocytes	0.0814	0.0000250	0.0000229	0.274
Uric Acid	0.0384	0.1006545	0.0272225	<0.001

Caption: Multiple linear regression with significance at $p<0.05$. R^2 = Coefficient of determination; β : β value for multiple linear regression; Variables adjusted for: age, income, physical activity level, and unhealthy diet.

are associated with visceral fat, dyslipidemia, and Metabolic Syndrome (MS). Additionally, studies have concluded that VAI has greater specificity and sensitivity compared to classical parameters such as waist circumference, BMI, and biochemical parameters. Moreover, VAI showed good predictive capacity regarding the risk of hypertension and MS, with some studies confirming its validity in other populations²⁴. Therefore, investigating at-risk individuals enables early interventions, which directly impacts health and, consequently, their quality of life.

Considering that the VAI originally proposed by Amato was developed using a mathematical model based on a healthy adult Caucasian population, the author emphasizes that it should not be extrapolated to the pediatric population¹⁸. Moreover, another study identified that, in contrast to what is observed in adult populations, BMI showed a better association with adipokines, metabolic parameters, insulin resistance, and subclinical inflammation in the pediatric population compared to VAI³, indicating its limitation. However, other studies have shown favorable results for the use of VAI in this population. An Italian study conducted with children with a mean age of 11 years identified VAI as a promising tool for predicting metabolic syndrome in children with obesity, despite the sample containing only Caucasian individuals. Another study conducted in Brazil with a target population of 12 to 17 years old demonstrated good performance of VAI in identifying changes in blood pressure and insulin resistance³¹, indicating that VAI can perform adequately regardless of ethnicity, sex, or age.

These discrepancies have opened the possibility of extrapolating the VAI cut-off points and formulas to better detect metabolic changes in pediatric populations. In this regard, determining cutoff values for this indicator in children and adolescents is of vital importance, as there is still no universally accepted reference value for this population. Recent studies have aimed to address these gaps. A cross-sectional study with 3,843 Iranian students aged 7 to 18 years identified the following VAI cut-off points for predicting Metabolic Syndrome: 1.58 for the total population, 1.30 for males, and 1.78 for females. These values were associated with an increased cardiometabolic risk. However, this study did not compare VAI values with other direct and indirect measures of visceral fat, nor did it account for the pubertal stage of the individuals. Additionally, as it was a cross-sectional study, no causality could be inferred between VAI and metabolic disturbances. Moreover, due to the focus on a specific ethnic group, these values need to be evaluated in studies with different populations to determine more suitable cutoff values for this age group.

Another possibility is to introduce modifications to the proposed mathematical model, as adopted in the present article. Thus, a broad range of BMI categories was considered to calculate the first component of the adjusted formula, and unlike the linear correlation used for VAI calculation in adult populations, the author used a nonlinear quadratic regression model that accurately explained the relationship between waist circumference and BMI in both male and

female³⁰ pediatric populations. Another study adapted VAI following the steps of the originally proposed formula. Hypothesis tests were applied according to the distribution of variables, and crude and adjusted regression analyses were performed. It was observed that the linear regression model showed a good explanatory capacity among the anthropometric markers that compose the equation. Additionally, medians of triglycerides and HDL cholesterol values for boys and girls were included to compose the new adjusted mathematical model for adolescents.

Furthermore, an important factor to highlight is the correlation of leukocytes with pediatric VAI, as excess fat is associated with immune dysfunction, accompanied by changes in leukocyte count. In this sense, obese adolescents have a higher leukocyte count compared to eutrophic individuals, suggesting a chronic pro-inflammatory state that may be associated with excessive adiposity and decreased cardiorespiratory fitness. Thus, pediatric VAI proved to be a good predictor of the inflammatory state in overweight adolescents.

Therefore, despite the early onset of metabolic disorders, it is known that clinical manifestations of different health outcomes are usually observed and accentuated in adulthood. Given that health habits are formed in childhood and adolescence, it is evident that early-life changes can be strong predictors of cardiometabolic diseases in adulthood. Our analyses revealed that pediatric VAI was correlated with variables related to cardiometabolic risk. Additionally, another study conducted with individuals aged 10-18 years of both sexes demonstrated significant differences between sexes for age²⁰, HDL-c, and VAI, which contrasted with our study, as there were no differences in pediatric VAI between sexes. It was also observed that the variables used for the VAI calculation were worse for adolescents with altered nutritional status (non-eutrophic), indicating a higher cardiometabolic risk associated with excess weight, also seen in the correlation between the number of metabolic changes and pediatric VAI demonstrated in this study.

This study has limitations. Due to the cross-sectional design, we cannot ensure the temporality of the associations between pediatric VAI and cardiometabolic risk factors. However, an important dose-response relationship was observed, with increased chances of metabolic changes for higher values of pediatric VAI. Therefore, it is highlighted that this is a population-based study with rigorous methodological data collection and the use of validated methods.

CONCLUSION

Pediatric VAI was associated with cardiometabolic and inflammatory markers in adolescents of both sexes. This index may be an effective strategy for identifying adipose tissue dysfunction, indirectly reflecting early risk of metabolic diseases.

Further studies should be conducted to elucidate its utility in pediatric clinical practice.

AUTHORS' CONTRIBUTIONS

We describe contributions to the papers using the taxonomy (CRediT) provided above:

Conceptualization, formal analysis, and funding acquisition of data: PR de Souza; NN Leite. *Formal analysis of the manuscript:* JVM Pereira. *Conceptualization, data curation, formal analysis, and supervision of study:* PF Pereira. All authors writing, review and editing the final manuscript.

COPYRIGHT

Copyright© 2020 Souza et al. This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original article is properly cited.

REFERENCES

1. Organização das Nações Unidas (ONU). *OMS revela principais causas de morte e incapacidade em todo o mundo entre 2000 e 2019*. Brasília, DF: ONU; 2020; [access in 2021 May 25]. Available from: <https://brasil.un.org/pt-br/104646-oms-revela-principais-causas-de-morte-e-incapacidade-em-todo-o-mundo-entre-2000-e-2019>.
2. Organização das Nações Unidas (ONU); Organização Pan-Americana da Saúde (OPAS). *Representante da OPAS/OMS no Brasil faz chamado à ação para acabar com estigma, prevenir e controlar a obesidade*. Brasília, DF: ONU; OPAS; 2021; [access in 2021 May 25]. Available from: <https://www.paho.org/pt/noticias/5-3-2021-representante-da-opasoms-no-brasil-faz-chamado-acao-para-acabar-com-estigma>.
3. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. *Pediatrics*. 1999;103(6 Pt 1):1175-82.
4. Srinivasan SR, Myers L, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: The Bogalusa Heart Study. *Metabolism*. 1996;45(2):235-40.
5. Carmienke S, Freitag MH, Pischon T, Schlattmann P, Fankhaenel T, Goebel H, et al. General and abdominal obesity parameters and their combination in relation to mortality: A systematic review and meta-regression analysis. *Eur J Clin Nutr*. 2013;67(6):573-85.
6. Oliveira CL, Mello MT, Cintra IP, Fisberg M. Obesity and metabolic syndrome in infancy and adolescence. *Rev Nutr*. 2004;17(2):237-45.
7. Mathieu P, Poirier P, Pibarot P, Lemieux I, Després JP. Visceral obesity: The link among inflammation, hypertension, and cardiovascular disease. *Hypertension*. 2009;53(4):577-84.
8. Ribeiro Filho FF, Mariosa LS, Ferreira SRG, Zanella MT. Visceral fat and metabolic syndrome: More than a simple association. *Arq Bras Endocrinol Metabol*. 2006;50(2):230-8.
9. Tchernof A, Després JP. Pathophysiology of human visceral obesity: An update. *Physiol Rev*. 2013;93(1):359-404.
10. Trandafir LM, Russu G, Moscalu M, Miron I, Lupu VV, Constantin MML, et al. Waist circumference: A clinical criterion for prediction of cardiovascular complications in children and adolescents with overweight and obesity. *Medicine*. 2020;99(30):e20923.
11. Barbalho SM, Bechara MD, Quesada K, Gabaldi MR, Goulart RA, Tofano RJ, et al. Metabolic syndrome, atherosclerosis, and inflammation: An inseparable triad? *J Vasc Bras*. 2015;14(4):319-27.
12. Berenson GS, Srinivasan SR, Bao W, et al. Cardiovascular risk factors and atherosclerosis in children and young adults. *Eur J Pediatr*. 1998;157(11):947-48.
13. McGill HC Jr, McMahan CA, Zieske AW, et al. Association of coronary heart disease risk factors with the progression of coronary atherosclerosis in children and young adults. *J Clin Endocrinol Metab*. 2000;72(April):[Page numbers not provided].
14. World Health Organization (WHO). Obesity and overweight. Geneva: WHO; 2025. [access in 2025 Abr 27]. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>.
15. Calabro P, Yeh ETH. Intra-abdominal adiposity, inflammation, and cardiovascular risk: New insight into global cardiometabolic risk. *Curr Hypertens Rep*. 2008;10(1):32-8.
16. Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006;444(7121):860-7.
17. Funder JW, Reincke M. Aldosterone: A cardiovascular risk factor? *Biochim Biophys Acta*. 2010;1802(12):1188-92.
18. Aubert J, Darimont C, Safonova I, Ailhaud G, Negrel R. Regulation by glucocorticoids of angiotensinogen gene expression and secretion in adipose cells. *Biochem J*. 1997;328(Pt 2):701-6.
19. Amato MC, Giordano C, Galia M, Criscimma A, Vitabile S, Midiri M, et al. Visceral adiposity index: A reliable indicator of visceral fat function associated with cardiometabolic risk. *Diabetes Care*. 2010;33(4):920-2.
20. Al-Daghri NM, Al-Attas OS, Wani K, Alnaami AM, Sabico S, Al-Ajlan A, et al. Sensitivity of various adiposity indices in identifying cardiometabolic diseases in Arab adults. *Cardiovasc Diabetol*. 2015;14(1):101.
21. Oliveira CM de, Ulbrich AZ, Neves FS, Dias FAL, Horimoto ARVR, Krieger JE, et al. Association between anthropometric indicators of adiposity and hypertension in a Brazilian population: Baependi Heart Study. *PLoS One*. 2017;12(10):1-10.
22. Koloverou E, Panagiotakos DB, Kyrou I, Stefanadis C, Chrysohou C, Georgousopoulou EN, et al. Visceral adiposity index outperforms common anthropometric indices in predicting 10-year diabetes risk: Results from the ATTICA study. *Diabetes Metab Res Rev*. 2019;35(6):1-9.
23. Yang J, Li H, Han L, Zhang L, Zhou Y. Association between Visceral Adiposity Index and hypertension among Chinese Adults: A nationwide cross-sectional study in the China Health and Nutrition Survey. *Blood Press Monit*. 2020;25(5):271-7.

24. Zheng SH, Li XL. Visceral adiposity index as a predictor of clinical severity and therapeutic outcome of PCOS. *Gynecol Endocrinol*. 2016;32(3):177-83.
25. Amato MC, Giordano C. Visceral adiposity index: An indicator of adipose tissue dysfunction. *Int J Endocrinol*. 2014;2014:730423.
26. Schuster J, Vogel P, Eckhardt C, Morelo SDB. Applicability of the visceral adiposity index (VAI) in predicting components of metabolic syndrome in young adults. *Nutr Hosp*. 2014;30(4):806-12.
27. Thivel D, O'Malley G, Pereira B, Duché P, Aucouturier J. Comparison of total body and abdominal adiposity indexes to dual x-ray absorptiometry scan in obese adolescents. *Am J Hum Biol*. 2015;27(3):334-8.
28. Klisic A, Kavacic N, Bjelakovic B, Zvrko E, Soldatovic I. Visceral adiposity index is not superior over anthropometric parameters with regards to inflammation in healthy adolescent girls. *P R Health Sci J*. 2018;37(4):195-9.
29. Ejtahed H-S, Kelishadi R, Hasani-Ranjbar S, Angoorani P, Motlagh ME, Shafiee G, et al. Discriminatory ability of visceral adiposity index as an indicator for modeling cardio-metabolic risk factors in pediatric population: the CASPIAN-V study. *J Cardiovasc Thorac Res*. 2019;11(4):280-6.
30. Ismail NA, Ragab SH, El Baky AMNEDA, Ibrahim MH. Potential role of new anthropometric parameters in childhood obesity with or without metabolic syndrome. *Open Access Maced J Med Sci*. 2019;7(23):3930-6.
31. Villanueva-Ortega E, Garcés-Hernández MJ, Herrera-Rosas A, López-Alvarenga JC, Laresgoiti-Servitje E, Escobedo G, et al. Gender-specific differences in clinical and metabolic variables associated with NAFLD in a Mexican pediatric population. *Ann Hepatol*. 2019;18(5):693-700.
32. Cota BC, Ribeiro SAV, Priore SE, Juvanhil LL, Faria ER, Faria FR, et al. Anthropometric and body composition parameters in adolescents with the metabolically obese normal-weight phenotype. *Br J Nutr*. 2022;127(10):1458-66.
33. Instituto Brasileiro de Geografia e Estatística (IBGE). Censo Demográfico 2010. Minas Gerais: IBGE; 2010; [access in 2025 Apr 27]. Available from: <http://www.cidades.ibge.gov.br/xtras/temas.php?lang=&codmun=317130&idtema=67&search=minas-gerais%7Cviciosa%7CCenso-demografico-2010:-resultados-do-universo-caracteristicas-da-populacao-e-dos-domicilios>.
34. Saliba S, Batista MC, Martino H, et al. Importance of abdominal fat parameters for the prediction of arterial stiffness in adolescents. *J Clin Endocrinol Metab*. 2020;105(9):1-7.
35. Fischer A, Souza R, Camargo M, et al. Visceral fat and its anthropometric measurements: Analysis of new criteria in obesity. *Rev Bras Obesidade Nutr Emagrecimento*. 2018;12(4):226-31.
36. Fonseca-Alaniz MH, Takada J, Andreoli MF, et al. Adipose tissue as a central regulator of metabolism. *Arq Bras Endocrinol Metabol*. 2006;50(2):216-29.
37. Girard J, Lafontan M. Impact of visceral adipose tissue on liver metabolism and insulin resistance. Part II: Visceral adipose tissue production and liver metabolism. *Diabetes Metab*. 2008;34(5):439-45.
38. Capurso C, Capurso A. From excess adiposity to insulin resistance: The role of free fatty acids. *Vasc Pharmacol*. 2012;57(2-4):91-7.
39. Yudkin JS. Adipose tissue, insulin action, and vascular disease: inflammatory signals. *Int J Obes Relat Metab Disord*. 2003;27(Suppl 3):S25-8.
40. Seravalle G, Grassi G. Obesity and hypertension. *Pharmacol Res*. 2017;122:1-7.

