



# The role of the visceral adiposity index in the assessment of metabolic syndrome of polycystic ovary syndrome patients: a new anthropometric index

Ana Luiza L. Rocha<sup>1,2</sup> · Thais Baêta<sup>1</sup> · Isabel R. Nazareth<sup>1</sup> · Julia M. Costa<sup>1</sup> · Julia D. Caporalli<sup>1</sup> · Maraisa A. Oliveira<sup>1</sup> · Marina G. Couto<sup>1</sup> · Rosana C. Azevedo<sup>3</sup> · Fábio V. Comim<sup>3</sup> · Flávia R. Oliveira<sup>1</sup> · Fernando M. Reis<sup>1</sup> · Ana L. Cândido<sup>3</sup>

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## Abstract

**Purpose** Polycystic ovary syndrome (PCOS) is a common endocrine disorder often linked to metabolic syndrome (MS), raising the risk of cardiovascular disease and type II diabetes. Certain indicators, such as the lipid accumulation product (LAP) and homeostatic model assessment for insulin resistance (HOMA-IR), can predict MS in PCOS patients. This study aimed to assess the predictive power of the visceral adiposity index (VAI) in comparison to LAP and HOMA-IR as predictors of MS in PCOS patients.

**Methods** In this cross-sectional observational study, data from 317 diagnosed PCOS women were analyzed. VAI, LAP, and HOMA-IR were computed as indexes. Participants were categorized into two groups for index accuracy comparison: PCOS patients with and without MS. The data were assessed using a ROC curve.

**Results** Among PCOS women with MS, 92.3% had abnormal VAI results, 94.5% had abnormal LAP results, and only 50.5% had abnormal HOMA-IR results. Conversely, the majority of PCOS women without MS had normal HOMA-IR (64.6%). When comparing these indexes using the ROC curve, VAI displayed the highest accuracy, followed by LAP and HOMA-IR.

**Conclusion** The VAI index proved to be a superior predictor of metabolic MS in PCOS women when compared to other indexes.

**Keywords** Polycystic ovary syndrome (PCOS) · Metabolic Syndrome (MS) · Visceral adiposity index (VAI) · Cardiovascular risks · Type II diabetes

## What does this study add to the clinical work

The visceral adiposity index (VAI) is a superior predictor of metabolic syndrome (MS) in women with polycystic ovary syndrome (PCOS), when compared to the lipid accumulation index (LAP) and homeostatic model assessment of insulin resistance (HOMA-IR). Therefore, VAI can be a valuable tool in the early identification of cardiovascular risks and type II diabetes in PCOS patients.

✉ Ana Luiza L. Rocha  
ana\_lunardi@yahoo.com.br

<sup>1</sup> Department of Obstetrics and Gynecology, Federal University of Minas Gerais, Belo Horizonte, Brazil

<sup>2</sup> Division of Human Reproduction, Department of Obstetrics and Gynecology, Federal University of Minas Gerais, Av. Alfredo Balena, 110 – 9º Andar, Belo Horizonte, MG 30130-100, Brazil

<sup>3</sup> Department of Internal Medicine, Federal University of Minas Gerais, Belo Horizonte, Brazil

## Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women of reproductive age, with a prevalence ranging 5% and 20%, depending on the

diagnostic criteria employed [1]. According to the Rotterdam criteria, PCOS is characterized by the presence of at least two out of three criteria: clinical and/or laboratorial hyperandrogenism, oligo/anovulation, and polycystic ovarian morphology on ultrasound [1], after excluding other causes of anovulation and hyperandrogenism such as Cushing's syndrome, congenital adrenal hyperplasia, hyperprolactinemia, thyroid dysfunction, androgen-producing tumors, and ovarian hyperthecosis have been excluded [2, 3]. PCOS is characterized by dysfunction in the hypothalamic-pituitary-ovary axis and anovulation. However, unlike other causes of ovulatory dysfunction, which involve insufficient ovarian follicle development or suppressed gonadotropin secretion (or both), PCOS typically presents with excess androgens and subtle deviations in serum levels of gonadotropins and estrogens that are not readily detected by routine tests. PCOS carries the potential for significant consequences, including an elevated risk of developing endometrial hyperplasia and neoplasia. Additionally, extra-reproductive manifestations of PCOS encompass insulin resistance (IR), metabolic syndrome (MS), and low-grade chronic inflammation [3–9]. The diagnosis of PCOS has been a subject of debate, as it involves difficulties in clearly defining the specific aspects within the diagnostic criteria. Additionally, there is substantial clinical diversity within PCOS, which is further compounded by variations across different ethnicities and alterations in clinical characteristics over the course of one's life [3].

Insulin resistance (IR) is detected in up to 70% of women with PCOS and is considered one of the main pathophysiological factors contributing to reproductive and metabolic disturbances in PCOS [10]. IR and disturbances in glucose metabolism are currently believed to contribute to the pathogenesis of the disease. IR results in compensatory hyperinsulinemia, which, in turn, amplifies ovarian androgen production through both direct ovarian effects and by triggering LH secretion. Additionally, women with PCOS often exhibit a higher prevalence of impaired glucose tolerance (IGT), obesity and metabolic syndrome (MS). This cluster of factors has been identified as a predictor of increased risk for future cardiovascular events and type 2 diabetes [11, 12]. The combination of obesity, particularly abdominal obesity, and PCOS has been shown to synergistically promote premature atherosclerosis and elevate cardiovascular mortality [13–17]. These findings underscore the importance of assessing cardiometabolic risk in women with PCOS in clinical practice [3, 11].

Despite its clinical relevance, diagnosing MS in PCOS patients poses challenges due to the heterogeneity of PCOS and variations in diagnostic criteria. Current predictive markers, such as BMI, fasting insulin levels, and lipid profiles, have limitations in accurately identifying MS in PCOS.

These markers may not fully capture the unique metabolic complexities and variations present in PCOS patients.

Some markers, including the lipid accumulation product (LAP), homeostatic model assessment for insulin resistance (HOMA-IR), and visceral adiposity index (VAI) have been proposed as predictors of cardiovascular complications and insulin resistance even prior to the diagnosis of metabolic syndrome (MS). Early identification of MS in PCOS patients has the potential to prevent further complications associated with metabolic disturbances, such as cardiovascular disease (CVD) [1, 18].

The homeostasis model assessment of insulin resistance (HOMA-IR), which utilizes fasting glucose and fasting insulin levels, serves as an alternative to the glucose clamp method for assessing insulin resistance. Despite the widespread use of HOMA-IR, there is a lack of consensus regarding the cutoff points for classifying insulin resistance. Some researchers have attempted to determine HOMA-IR cutoffs in subjects exhibiting tendencies towards insulin resistance or MS, but their findings have been inconsistent [18]. While insulin resistance may play a central role in the cluster of metabolic abnormalities characterizing MS, previous studies indicate that MS is not always synonymous with insulin resistance [18], which may limit the utility of this index as a predictor of MS.

The lipid accumulation product (LAP) was introduced by Kahn in 2005 as a superior indicator of cardiovascular diseases (CVD) risk compared to BMI [19]. Numerous studies have demonstrated a strong association between LAP and metabolic syndrome (MS) according to various diagnostic criteria [20]. LAP is a cardiovascular risk index based on the combination of waist circumference (WC) and fasting triglyceride (TG) levels. However, despite its potential, there is a scarcity of clinically relevant studies assessing the utility of LAP.

The visceral adiposity index (VAI) is an empirical-mathematical model that is gender-specific and relies on anthropometric measurements such as BMI and waist circumference, as well as biochemical parameters including triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) concentrations [21]. This index enables accurate identification of visceral adipose tissue and predicts the likelihood of obesity-related complications even before the diagnosis of metabolic syndrome [1, 20, 22, 23]. A VAI value greater than 1.675 enables differentiation between women with metabolically unhealthy polycystic ovary syndrome (MU-PCOS) and those with metabolically healthy polycystic ovary syndrome (MH-PCOS) [22].

This study aimed to assess the predictive role of VAI compared to LAP and HOMA-IR indexes in identifying metabolic syndrome (MS) among patients with polycystic ovary syndrome (PCOS). This study aims to address the critical need for more accurate predictive markers for MS

in PCOS, intending to overcome the limitations of current markers and provide a more precise assessment of MS risk in PCOS patients.

## Material and methods

This cross-sectional observational study was conducted at the Hyperandrogenism Outpatient Clinic of the Clinical Hospital of the Federal University of Minas Gerais (UFMG), Belo Horizonte, MG, Brazil. The study population consisted of 317 women aged 18–40 years, diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam criteria, between November 2009 and June 2019. A secondary database was utilized, and data were included for secondary analysis if all the prerequisite variables required for calculating the indexes were available.

The collected data from the database included BMI, weight, height, waist circumference (WC), total cholesterol, HDL, LDL, total testosterone, triglyceride (TG), homeostatic model assessment for insulin resistance (HOMA-IR), lipid accumulation product (LAP), and visceral adiposity index (VAI). The indexes were calculated using the following formulas:  $HOMA-IR = \text{fasting blood glucose} \times \text{fasting insulin} / 405$ ,  $LAP = (WC [cm] - 58) \times TG (mmol/L)$  and  $VAI = (WC/36.58 + [1.89 \times BMI]) \times (TG/0.81) \times (1.52/HDL)$ . Reference values for the indexes were defined as  $VAI \leq 1.67$ ,  $LAP < 34.5$ , and  $HOMA-IR < 2.7$ .

Metabolic syndrome (MS) was diagnosed based on the National Cholesterol Education Program Adult Treatment Panel (NCEP-ATP) III criteria, which required the presence of at least three of the following criteria: waist circumference (WC)  $> 88$  cm, triglyceride level  $\geq 150$  mg/dl, high-density lipoprotein (HDL)  $< 50$  mg/dl, blood pressure  $\geq 130/85$  mmHg, and fasting glucose level  $\geq 110$  mg/dl [24].

The participants were categorized into two groups: PCOS patients with metabolic syndrome (MS) and PCOS patients without metabolic syndrome.

## Ethical aspects

The present research was approved by research ethics committees from UFMG (Federal University of Minas Gerais). All the participants signed a term of informed consent.

## Statistical analyses

Sample calculation considered a prevalence of 10% for PCOS and, among these patients, 60% for MS [2]. A 95% confidence interval and a maximum error of 0.03 were

defined to estimate the true value. The data from 238 patients were required in this study.

Descriptive (frequencies, percentages, means, standard deviation) and comparative analyses were used for the statistical analyses. Comparative analyses included the Student's t-tests to compare the means of two independent groups and the chi-squared test to compare categorical variables. Statistically significant associations were considered when the P-value was less than or equal to 0.05.

## Results

Figure 1 presents the characteristics of the 317 included PCOS patients, with 28.7% having metabolic syndrome (MS) and 71.3% without MS.

Table 1 describes the clinical and laboratory data of the study population, presenting basic statistical information.

The comparison of variables between patients with and without MS (Table 2) revealed no significant differences in height, insulin level, Ferriman score, and total testosterone level. However, patients with MS had higher mean values of weight, waist circumference (WC), body mass index (BMI), fasting blood glucose, and triglycerides. Conversely, patients without MS had a higher mean value of high-density lipoprotein (HDL) compared to patients with MS.

The association between VAI and MS was assessed using the chi-squared test (Table 3), which demonstrated a statistically significant difference between the groups with and without MS ( $P = 0.000$ ). The majority of patients exhibited elevated VAI values, albeit in varying proportions. Specifically, VAI changed in 92.3% of patients with MS and 53.1% of patients without MS. The odds ratio indicated that an MS patient was less likely to have a normal VAI compared to a changed VAI (odds ratio = 0.094).

The chi-squared test also revealed a significant difference between patients with and without MS regarding LAP ( $P = 0.000$ ) (Table 4). The majority of patients had elevated LAP values, with 94.5% of women with MS and

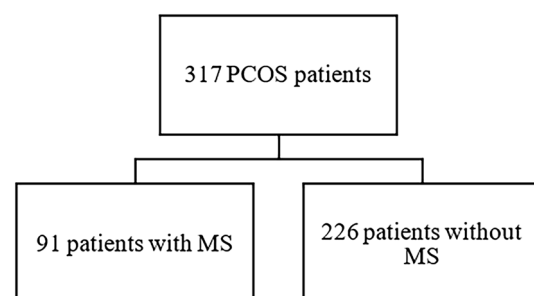


Fig. 1 Patient selection flowchart

**Table 1** General descriptive analyses

Variables	N	Minimum	Maximum	Mean	Standard-deviation	Median
Weight (kg)	317	40.9	151	80.3	17.9	80.5
Height (cm)	317	1.45	1.8	1.61	0.07	1.61
Waist (cm)	317	64	147	97.76	14.61	97
BMI (kg/m <sup>2</sup> )	317	17	54.1	30.74	6.35	30.3
HDL (mg/dl)	317	21	86	46.26	10.97	45
Fasting blood glucose	317	55	141	86.63	11.84	85
TGL (mg/dl)	317	28	390	124.75	62.44	111
Insulin	317	1	183	15.08	16.04	11
Ferriman score	194	0	32	10.37	6.31	10
Total testosterone	185	0	860	56.4	68.47	53
Age (years)	317	24.5	30.9	30.6	6.4	29

**Table 2** Student's *t*-test for comparison between clinical and laboratory data of polycystic ovary syndrome patients with and without metabolic syndrome

Variables	Metabolic syndrome		P-value
	Yes	No	
Weight (kg)	87.0 (16.4)	77.6 (± 17.8)	0.000*
Height (cm)	1.62 (0.06)	1.61 (± 0.07)	0.389
WC (cm)	103.63 (12.9)	95.40 (± 14.62)	0.000*
BMI (kg/m <sup>2</sup> )	33.12 (5.53)	29.78 (± 6.41)	0.000*
HDL (mg/dl)	39.68 (6.43)	48.91 (± 11.31)	0.000*
Fasting blood glucose	90.26 (15.21)	85.17 (± 9.83)	0.000*
TGL (mg/dl)	176.95 (65.95)	103.70 (± 46.78)	0.000*
Insulin	17.34 (14.39)	14.17 (± 16.60)	0.111
Ferriman score	11.24 (7.47)	10.06 (± 5.85)	0.257
Total testosterone	54.50 (39.13)	57.10 (± 76.65)	0.819
Age (years)	30.9 (± 6.4)	30.3 (± 4.5)	0.641

**Table 3** Chi-squared test comparing the metabolic syndrome and visceral adiposity index variables

VAI	Metabolic syndrome		P-value
	Yes%	No%	
Normal	7.70	46.90	0.000*
Changed	92.30	53.10	

**Table 4** Chi-squared test comparing the metabolic syndrome and lipid accumulation product variables

LAP	Metabolic syndrome		P-value
	Yes%	No%	
Normal	5.50	43.80	0.000*
Changed	94.50	56.20	

**Table 5** Chi-squared test comparing the metabolic syndrome and homeostatic model assessment for insulin resistance variables

HOMA-IR	Metabolic syndrome		P-value
	Yes%	No%	
Normal	49.50	64.60	0.013*
Changed	50.50	35.40	

56.2% of women without MS showing a change. The odds ratio indicated that an MS patient had a lower likelihood of having a normal LAP compared to a changed LAP (odds ratio = 0.075).

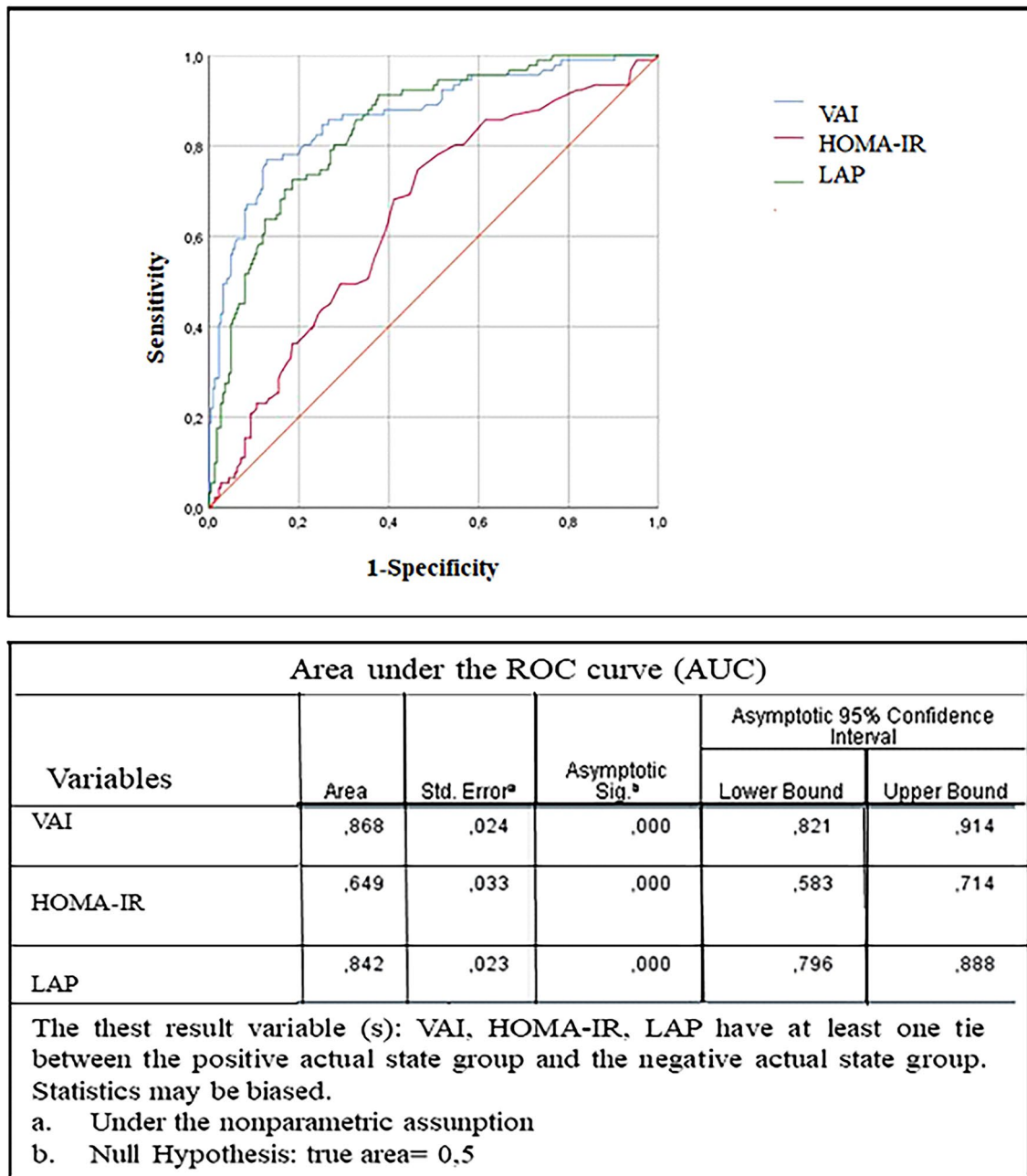
Regarding HOMA-IR, the chi-squared test showed a significant difference between patients with and without MS ( $P = 0.013$ ) (Table 5). Among patients with MS, 50.5% had a changed HOMA-IR, while 49.5% had a normal HOMA-IR. The majority of patients without MS exhibited a normal HOMA-IR. The odds ratio indicated that an MS patient had a lower likelihood of having a normal HOMA-IR compared to a changed HOMA-IR (odds ratio = 0.536).

The receiver operating characteristic (ROC) curve analysis comparing the accuracy of VAI, LAP, and HOMA-IR indexes demonstrated that VAI had the highest accuracy, followed by LAP, and subsequently HOMA-IR (Fig. 2).

## Discussion

The present study provides evidence that VAI is a robust predictor of metabolic syndrome (MS) in polycystic ovary syndrome (PCOS) patients, surpassing the accuracy of LAP and HOMA-IR. Moreover, a positive association was observed among the three indexes.

Previous research by Schuster et al. demonstrated a direct correlation between the diagnostic criteria for MS and VAI in young adults, suggesting VAI as a reliable predictor of



**Fig. 2** Accuracy of the three models on the receiver operating characteristic curve

the condition [25]. Joshi et al. reported significantly higher VAI values in the PCOS patient group compared to the control group, highlighting its effectiveness as a predictive biomarker in the studied population [26]. Similarly, Techatraisak et al., in a study comprising 399 PCOS patients and 42 controls, found an association between the recommended reference value for VAI in the literature and MS. However, their ROC curve analysis suggested the need for a new cutoff value. Furthermore, the prevalence of MS in PCOS (24.6%) reported by Techatraisak et al. aligns with the findings of the

present study, where 28.7% of PCOS patients presented MS, while 71.3% did not.

Although this study did not observe a statistical difference in HOMA-IR values between patients with and without MS, significant differences were detected in patients without MS. Pontes et al. conducted a study involving 189 PCOS patients, demonstrating a strong association between HOMA-IR and LAP as biomarkers for insulin resistance [28]. The prevalence of insulin resistance varied according to the assessment method and was directly related to BMI. Existing

literature indicates that increased HOMA-IR and LAP values are commonly associated with insulin resistance.

Soares's study indicated that LAP is a reliable predictor of MS in adults, with an area under the ROC curve greater than 0.85 [29]. Similarly, Ribeiro et al. conducted a cross-sectional study revealing that both LAP and VAI can serve as sensitive predictors of MS and insulin resistance in PCOS patients [30]. However, the present study found LAP to be a more sensitive index than VAI, emphasizing its simplicity and practicality, which makes it an encouraging choice for clinical application.

Strengths of this study include a sample size surpassing the minimum requirement and comparable populations in various measures, such as height, insulin, and indicators of clinical and laboratory hyperandrogenism (Ferriman score and total testosterone, respectively). Additionally, the study introduced a novel index that is cost-effective, user-friendly, and highly accurate in predicting MS in PCOS patients, adding value to the assessment of PCOS.

Limitations of the study include its cross-sectional design, reliance on a secondary database with data collected by different professionals, and the possibility of information bias. Furthermore, the study highlighted the non-comparability of variables such as BMI, HDL, TG, WC, weight, and fasting blood glucose between groups, which was expected as these variables were used in the calculation of the studied indexes.

In conclusion, this study establishes VAI as a superior biomarker compared to other indexes, proving its efficacy in predicting MS in PCOS patients. Its simplicity and accuracy make it a valuable tool in the evaluation of PCOS. However, further research is necessary to explore the applicability of VAI due to its relatively recent introduction in the field.

**Author contributions** BT: conceptualization, data curation, writing—original draft preparation; NIR; CJM; CJD; OMA; CMG: data curation, writing—original draft preparation; ARC: conceptualization, data curation, writing—review & editing; OFR: data curation—original draft preparation; CFV: data curation, writing—original draft preparation; CAL: project administration, supervision, writing—review & editing; RFM and R ALL: conceptualization, data curation, methodology, supervision, writing—review & editing.

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**Data availability** Data supporting this paper are available on request.

## Declarations

**Conflict of interest** The authors certify that they have NO affiliations with or involvement in any organization or entity with any financial, or non-financial interest in the subject matter or materials discussed in this manuscript.

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