

Diagnostic performance of the systemic inflammation response index and aggregate index of systemic inflammation in polycystic ovary syndrome: A ROC curve analysis

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder in reproductive-age women, characterized by hormonal, metabolic, and inflammatory changes. Chronic low-grade inflammation plays a key role in its pathogenesis. Traditional markers such as CRP and leukocyte count are often non-specific, while novel biomarkers SIRI and AISI provide a more comprehensive assessment of immune status.

Methods: In this comparative study, 35 women with PCOS and 45 healthy controls aged 18–40 years were evaluated. Age and BMI were comparable between groups. Serum fasting glucose, fasting insulin, lipid profile, CRP, and total testosterone were measured (Cobas pure, Roche Diagnostics), and insulin resistance was estimated via HOMA-IR. SIRI and AISI indices were calculated from complete blood count data using a Sysmex XN-1000 analyzer.

Results: Among women with PCOS, phenotype A was predominant (85.7%). Compared to controls, women with PCOS had higher fasting insulin, HOMA-IR, atherogenic lipid profile, CRP, total leukocyte count, total testosterone, and inflammatory indices (SIRI and AISI), while fasting glucose was similar. Both AISI and SIRI were significantly higher in PCOS (AISI median 269.4 vs. 150.1; SIRI median 0.9500 vs. 0.5600; $p = 0.001$). ROC analysis showed acceptable discriminatory ability (AUC 0.712 for AISI; 0.713 for SIRI), with moderate sensitivity (AISI 57.8%; SIRI 64.4%) and specificity (AISI 80%; SIRI 74.3%) for distinguishing PCOS from controls.

Conclusions: AISI and SIRI values are significantly higher in women with PCOS and serve as accessible, reliable biomarkers of chronic low-grade inflammation, supporting their use in routine clinical assessment for early detection of systemic inflammation.

Keywords: AISI, biomarkers, low-grade inflammation, polycystic ovary syndrome (PCOS), SIRI

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders, affecting approximately 8–13% of women of reproductive age, and is a leading cause of infertility [1,2].

The Rotterdam criteria remain the gold standard for diagnosis, requiring the presence of at least two of the following three features: biochemical or clinical signs of hyperandrogenism, oligo-ovulation or anovulation, and ultrasound evidence of polycystic ovarian morphology [3].

PCOS is characterized by reproductive, metabolic, and psychological consequences: approximately 50% of women suffer from anovulatory infertility [4,5], 75–95% exhibit insulin resistance, which is associated with hyper-

androgenism and anovulation [4–6], and about 50% of cases progress to prediabetes or type 2 diabetes [5]. Furthermore, patients often experience anxiety, depression (~37%), and psychosexual dysfunction [5, 6].

Numerous studies have focused on investigating the etiology and pathogenesis of PCOS. Low-grade chronic inflammation is a recognized component of the pathophysiology of PCOS and is closely associated with insulin resistance, obesity, and hormonal imbalance. In this context, circulating blood cells—including neutrophils, monocytes, and lymphocytes—contribute to a sustained subclinical inflammatory response. Alterations in their number and activity promote increased production of pro-inflammatory mediators such as C-reactive protein (CRP), tumor necrosis factor- α (TNF- α), and interleukin-6 (IL-6) [7].

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Hyperinsulinemia and androgen excess further enhance immune cell activation, reinforcing cytokine release and maintaining a chronic inflammatory state. This persistent inflammation contributes to ovarian dysfunction, impaired folliculogenesis, and worsening metabolic disturbances, thereby linking hematologic inflammatory changes with both reproductive and cardiometabolic complications in PCOS [8].

Traditional inflammatory markers, such as CRP and total leukocyte count, exhibit limited specificity, necessitating the integration of more precise novel indicators of systemic inflammation. These include the Systemic Inflammation Response Index (SIRI) and the Aggregate Index of Systemic Inflammation (AIS), both calculated from complete blood count parameters [9-11].

SIRI is a three-cell biomarker that incorporates three types of leukocytes (neutrophils, monocytes, and lymphocytes), reflecting diverse immune response pathways [11]. A more complex marker is the Aggregate Index of Systemic Inflammation (AIS), which integrates neutrophil, monocyte, platelet, and lymphocyte counts. The inclusion of the platelet count allows for a more detailed assessment not only of the immune response but also of the prothrombotic state and endothelial dysfunction, both of which are characteristic of PCOS pathogenesis [12].

METHODS

The study was conducted in full accordance with the principles of the Declaration of Helsinki. The protocol was formally approved by the Ethics Committee of the Medical University of Sofia (Contract №150/2025). All participants provided written informed consent prior to inclusion in the study.

Study design and participants

A clinical and anamnesic method was employed. The study included 80 women divided into two groups:

1. PCOS group: 35 women aged 18-40 years, newly diagnosed and treatment-naïve, with PCOS diagnosed according to the Rotterdam criteria [13]. Participants were selected by a specialist in obstetrics and gynecology. According to the Rotterdam classification, participants were categorized into 4 phenotypes:

1.1. Phenotype A (classic PCOS): hyperandrogenism (HA) + oligo / anovulation (OA) + polycystic ovarian morphology (PCOM)

1.2. Phenotype B: HA + OA.

1.3. Phenotype C (ovulatory PCOS): HA + PCOM.

1.4. Phenotype D (non-hyperandrogenic PCOS): OA + PCOM

2. Control group: 45 age-matched healthy women with regular menstrual cycles and no comorbidities.

Sampling type

Participants were recruited consecutively from the outpatient clinic. Efforts were made to minimize selection bias by applying strict inclusion and exclusion criteria.

Exclusion criteria: To ensure sample homogeneity, women with thyroid disorders, hyperprolactinemia, premature ovarian failure, hypothalamic amenorrhea, congenital adrenal hyperplasia, androgen-producing tumors, Cushing's syndrome, autoimmune diseases, pregnancy, or lactation were excluded. Women who had used corticosteroids, oral contraceptives, or anti-androgens within the previous 3-6 months were also excluded.

Anthropometric data (simplified)

Age and body mass index (BMI) were recorded at enrollment. The BMI was calculated using the formula $\text{weight (kg)} / \text{height square meters (m}^2\text{)}$.

Laboratory methods

Blood collection: Venous blood samples were obtained following overnight fasting and collected into EDTA and serum tubes.

Complete blood count (CBC) and WBC differential: Freshly collected whole blood samples with K₂EDTA were analyzed within 2 hours of venipuncture. Until processing, specimens were maintained at room temperature. Samples showing visible clots or inadequate volume were excluded from analysis. CBC and WBC differential were performed using the Sysmex XN-1000 automated hematology analyzer (Sysmex Corporation, Kobe, Japan), based on fluorescent flow cytometry.

Systemic inflammatory indices calculation: SIRI = neutrophil $\times$ monocyte / lymphocyte count; AISI = neutrophil $\times$ monocyte $\times$ platelet / lymphocyte count.

Biochemical analyses: Venous blood samples were obtained following overnight fasting. The following parameters were measured, using the Cobas Pure Integrated System (Roche Diagnostics): fasting blood glucose (mmol/L), fasting insulin (μ U/mL), total cholesterol (mmol/L), low-density lipoprotein cholesterol (LDL-C, mmol/L), high-density lipoprotein cholesterol (HDL-C, mmol/L), triglycerides (TG, mmol/L), C-reactive protein (CRP, mg/L), and total testosterone (nmol/L).

Insulin resistance (HOMA-IR) was estimated using the formula: $\text{glucose (mmol/L)} \times \text{insulin (}\mu\text{U/mL)} / 22.5$.

Statistical Analysis

Normality was assessed using the Shapiro–Wilk test. As most variables were non-normally distributed, data are presented as medians and interquartile ranges (IQR). Comparisons between groups were performed using the non-parametric Mann–Whitney U test.

Sensitivity analyses were conducted for SIRI and AISI to assess the robustness of the findings. Outliers were defined using Tukey's method (1.5 $\times$ interquartile range), and all comparisons were repeated after exclusion of extreme values. ROC curve analyses were repeated in the sensitivity datasets.

RESULTS

Participants with polycystic ovary syndrome (PCOS) were classified into four phenotypic groups according to the Rotterdam criteria. Phenotype A was predominant (30/35, 85.7%), whereas phenotypes B (2/35, 5.7%), C (2/35, 5.7%), and D (1/35, 2.9%) were less frequent (Table 1).

No significant differences were observed between the PCOS and control groups in terms of age or body mass index (Mann–Whitney U test, $p=0.3198$ and $p=0.3425$, respectively; Table 1), indicating comparable baseline anthropometric characteristics. Women with PCOS exhibited significantly higher fasting insulin levels and HOMA-IR values compared to controls, whereas fasting glucose did not differ significantly between groups (Table 2).

The PCOS group demonstrated a more atherogenic lipid profile, characterized by elevated LDL-C and triglyceride levels and reduced HDL-C concentrations. Total cholesterol showed a non-significant trend toward higher values in women with PCOS.

Markers of systemic inflammation, including total leukocyte count, C-reactive protein (CRP), SIRI, and AISI, were significantly elevated in the PCOS group. Total testosterone levels were also markedly higher in women with PCOS.

Effect size analysis revealed small-to-large effects, with the strongest magnitudes observed for total testosterone, insulin resistance indices (HOMA-IR and fasting insulin), and the composite inflammatory indices (SIRI and AISI) (Table 2).

Given the observed elevation of composite inflammatory indices in women with PCOS, the diagnostic performance of SIRI and AISI as potential biomarkers of chronic low-grade inflammation associated with PCOS was further evaluated. The AISI index was significantly higher in women with PCOS compared to controls (median: 269.4 vs. 150.1; $p=0.001$, Mann–Whitney U test). Receiver operating characteristic (ROC) curve analysis demonstrated an area under the curve (AUC) of 0.712 (95% CI: 0.5989–0.8246), indicating acceptable discriminatory ability. The optimal cut-off value for identifying PCOS was 157.1, yielding a sensitivity of 57.78% and a specificity of 80% (Figure 1A).

Similarly, SIRI values were significantly higher in the PCOS group (median: 0.9500 vs. 0.5600; $p=0.001$, Whitney U test). ROC analysis showed an AUC of 0.713 (95% CI: 0.5997–0.8264). The optimal cut-off value for SIRI was 0.6250, corresponding to a sensitivity of 64.44% and a specificity of 74.29% (Figure 1B).

Sensitivity analyses confirmed the robustness of the findings for both indices. For SIRI, one extreme value was excluded in the PCOS group and one in the control group. For AISI, two extreme values were excluded in the PCOS group, while no outliers were detected among controls. The differences between PCOS and controls for both SIRI and AISI remained statistically significant after exclusion of outliers. ROC curve analyses demonstrated comparable AUC values in the sensitivity datasets, indicating stable discriminatory performance of both indices.

Table 1. Clinical and demographic characteristics of the two study groups

Clinical characteristic	PCOS (n = 35)		Controls (n = 45)	p-value
	Age (years), median (IQR)	29.0 (24.0–33.0)	27 (22.5–32.00)	0.319
	BMI (kg/m²), median (IQR)	23.1 (20.9–24.7)	22.7 (20.8–24.5)	0.342
Rotterdam PCOS phenotypes, n (%)	A (HA + OA + PCOM)	30 (85.7%)	-	-
	B (HA + OA)	2 (5.7%)	-	-
	C (HA + PCOM)	2 (5.7%)	-	-
	D (OA + PCOM)	1 (2.9%)	-	-
	Total PCOS	35 (100%)	-	-

Abbreviations: BMI – body mass index, HA – hyperandrogenism, IQR – interquartile range, OA – oligo/anovulation, PCOM – polycystic ovarian morphology, PCOS – polycystic ovary syndrome.

Table 2. Comparative analysis of laboratory parameters between the two study groups

Variable	PCOS median (IQR) (n=35)	Controls median (IQR) (n=45)	p-value	Effect size (r)
Fasting glucose (mmol/L)	5.05 (4.73 – 5.28)	4.92 (4.71 – 5.07)	0.0988	0.18
Fasting insulin (µIU/mL)	8.83 (5.95 – 10.50)	7.43 (5.86 – 8.29)	0.0016	0.35
HOMA-IR	2.02 (1.27 – 2.43)	1.53 (1.32 – 1.89)	0.0009	0.37
Total cholesterol (mmol/L)	4.63 (4.07 – 5.30)	4.32 (3.96 – 4.67)	0.0620	0.21
LDL-C (mmol/L)	2.70 (2.20 – 3.13)	2.31 (2.02 – 2.66)	0.0192	0.26
HDL-C (mmol/L)	1.48 (1.12 – 1.72)	1.65 (1.52 – 1.88)	0.0093	0.29
Triglycerides (mmol/L)	0.820 (0.57 – 1.28)	0.61 (0.50 – 0.78)	0.0033	0.33
WBC (×10 ⁹ /L)	6.80 (5.40 – 8.40)	5.70 (4.85 – 6.70)	0.0118	0.28
CRP (mg/L)	2.20 (0.60 – 5.40)	0.80 (0.50 – 1.40)	0.0114	0.28
Total testosterone (nmol/L)	1.58 (1.18 – 2.12)	1.06 (0.66 – 1.32)	< 0.0001	≥ 0.44
AISI	269.4 (174.3 – 334.8)	150.1 (85.02 – 262.7)	0.001	0.37
SIRI	0.950 (0.620 – 1.160)	0.560 (0.380 – 0.890)	0.001	0.37

Abbreviations: AISI – aggregate index of systemic inflammation, CRP – C-reactive protein, HDL-C – high-density lipoprotein cholesterol, HOMA-IR – homeostatic model assessment of insulin resistance, IQR – interquartile range, LDL-C – low-density lipoprotein cholesterol, PCOS – polycystic ovary syndrome, SIRI – systemic inflammation response index, WBC – white blood cells.

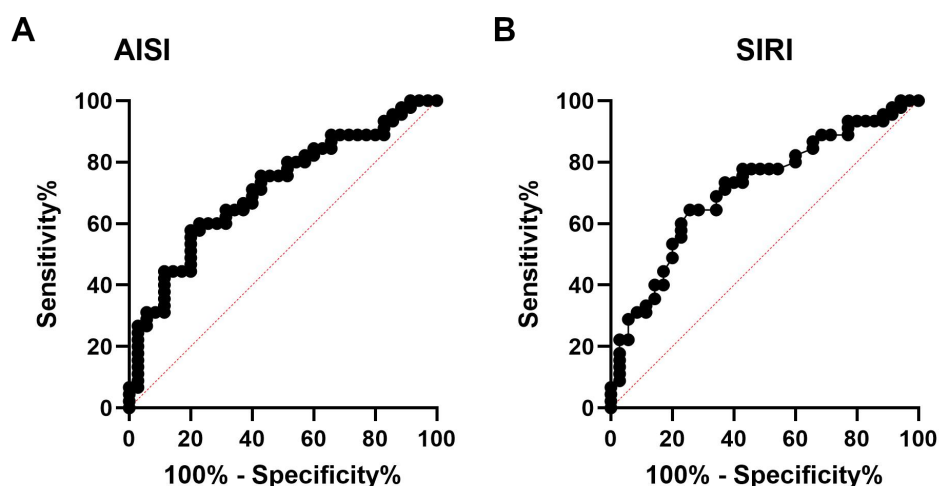


Figure 1. ROC curve analysis for PCOS detection

The composite indices were elevated in women with PCOS, suggesting aggregate index of systemic inflammation (AISI) and systemic inflammation response index (SIRI) are potential biomarkers of chronic low-grade inflammation associated with PCOS. **(A)** AISI performance: AUC 0.712 (95% CI: 0.598-0.824), optimal cut-off 157.1 (57.8% sensitivity, 80.0% specificity). **(B)** SIRI performance: 0.713 (95% CI: 0.599-0.826), optimal cut-off 0.625 (64.4% sensitivity, 74.3% specificity).

DISCUSSION

Our study confirmed the growing body of evidence supporting the presence of chronic low-grade inflammation in polycystic ovary syndrome (PCOS) [14-20].

The meta-analysis by Gnanadass et al. demonstrates that an imbalance in CRP levels and inflammatory cytokines such as TNF- α , IL-6, IL-8, IL-10, IL-18, and IL-33 plays a central role in the pathogenesis of the syndrome [14]. In support of this theory, Rudnicka et al. also report a significant increase in a wide range of proinflammatory markers and CRP in women with PCOS [16]. Qu et al. further contribute to the understanding of the immune genesis of the syndrome by identifying specific immune response-related genes with high diagnostic value and reporting enhanced infiltration of neutrophils and dendritic cells [17]. These data on cellular infiltration directly correspond with our results, which demonstrate alterations in differential leukocyte counts in women with PCOS.

The present study confirmed and expanded upon the findings of Ozgan et al. regarding the diagnostic value of AISI and SIRI in PCOS [20]. Although specific inflammatory markers such as cytokines, interleukins, and TNF provide a precise pathophysiological profile, their assessment in routine clinical practice remains impractical due to high costs and methodological complexity. It is precisely here that the clinical advantage of the AISI and SIRI indices becomes evident. They offer a reliable alternative for assessing immune dysregulation and leukocytosis using accessible and standardized parameters derived from the complete blood count.

In addition to SIRI and AISI, our study evaluated traditional inflammatory and metabolic parameters, including total leukocyte count, C-reactive protein (CRP), fasting in-

sulin, HOMA-IR, lipid profile, and total testosterone. Women with PCOS demonstrated significant elevations in these markers compared to controls, consistent with previous evidence of low-grade chronic inflammation and metabolic dysregulation in this population [14-17]. The observed correlations between these conventional markers and the composite indices further support the validity of SIRI and AISI as reliable, easily obtainable surrogates for systemic inflammatory status, highlighting their potential role in comprehensive clinical assessment of PCOS patients.

Our data demonstrate a highly significant correlation ($p=0.001$), which minimizes the risk of statistical error and establishes these indices as more stable biomarkers of systemic inflammation in this patient population.

The results of the ROC analysis of the AISI index highlight its role as an effective biomarker. Its high specificity indicates that it can successfully identify patients with pronounced inflammatory status while minimizing the risk of false-positive results in healthy women. An AUC value of 0.712 is comparable to established metabolic markers, confirming the benefit of including AISI in the diagnostic panel for PCOS. In parallel, the SIRI index demonstrates similar potential as a reliable and accessible biomarker for identifying low-grade systemic inflammation in women with PCOS. With significant diagnostic accuracy (AUC = 0.713), good sensitivity, and specificity at a cut-off value of 0.6250, SIRI represents a valuable parameter for clinical screening.

The robustness of the observed associations was supported by sensitivity analyses, as the exclusion of extreme values did not materially alter the statistical significance or direction of the differences in SIRI and AISI between women with PCOS and controls. These findings suggest that the discriminatory performance of both indices is stable and not driven by a small number of influential observations.

The primary advantage of both indices lies in their ease of use in routine clinical practice through complete blood count analysis and differential leukocyte counts, enabling effective monitoring without the need for costly specialized investigations.

The present study has several limitations that should be acknowledged. First, the sample size is relatively small, which may limit the generalizability of the findings and the statistical power to detect subtle correlations between inflammatory indices and specific PCOS phenotypes. Second, the cross-sectional design of the study allows for the identification of associations but precludes the establishment of a causal relationship between elevated AISI/SIRI levels and the development of PCOS-related complications. Nevertheless, the high precision of automated hematological analysis and the use of a well-defined control group strengthen the reliability of our preliminary results. Future large-scale prospective studies are required to validate these findings in more diverse populations.

CONCLUSIONS

With their good diagnostic accuracy and high specificity, the AISI and SIRI indices may offer an opportunity for the early identification of systemic inflammation in patients with PCOS. Their main advantage lies in their cost-effectiveness and accessibility, as they are calculated directly from routine complete blood count and white blood cell differential parameters. While other investigated indices also showed associations with systemic inflammation, AISI and SIRI appeared to demonstrate more consistent performance in the present cohort. The incorporation of AISI and SIRI into diagnostic algorithms for PCOS could potentially support more precise clinical screening and long-term patient monitoring; however, further validation in larger, prospective cohorts is warranted before routine clinical implementation.

ABBREVIATIONS

AISI- Aggregate Index of Systemic Inflammation

AUC – Area Under the Curve

CRP – C-reactive protein

HA – Hyperandrogenism

OA – Oligo/anovulation

PCOS – Polycystic ovary syndrome

PCOM – Polycystic ovarian morphology

ROC – Receiver Operating Character

SIRI – Systemic Inflammation Response Index

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AUTHORS' CONTRIBUTION

NND: conceptualization, methodology, investigation, formal analysis, data curation, writing – original draft, writing – review and editing.

PA: resources, patient selection.

ID: formal analysis, supervision.

MV: conceptualization, formal analysis, supervision.

All authors have read and approved the final version of the manuscript.

CONFLICT OF INTEREST

None to declare.

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