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Original Article

Inflammatory Markers in Adolescents with Polycystic Ovary Syndrome: Association with Androgen Levels

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ABSTRACT

Aim: Polycystic ovary syndrome (PCOS) is increasingly recognized as a systemic disorder associated with metabolic abnormalities and chronic inflammation. In this study, we aimed to investigate the relationship between androgen levels and inflammatory markers in adolescents with PCOS.

Materials and Methods: Eighty-nine patients with PCOS were analyzed retrospectively. Inflammatory markers, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII), were assessed. Androgen levels and metabolic parameters were also evaluated.

Results: The inflammatory markers were not significantly associated with androgen levels or hyperandrogenism. No significant differences in NLR, PLR, or SII were observed between adolescents with and those without hyperandrogenism or between patients with obesity and those without obesity (all $p>0.05$). Body mass index (BMI) and BMI-standard deviation scores were not correlated with inflammatory markers. In contrast, homeostasis model assessment of insulin resistance showed a weak but statistically significant positive correlation with SII.

Conclusion: Our findings demonstrate that inflammatory markers, including NLR, PLR, and SII, were not significantly associated with androgen levels. Furthermore, these markers did not differ according to the presence of obesity or hyperandrogenism.

Keywords: Polycystic ovary syndrome, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte, systemic immune-inflammation index

Introduction

Polycystic ovary syndrome (PCOS) represents a significant global health concern which affects approximately 10-13% of females worldwide (1). It is characterized by a heterogeneous clinical presentation, including hyperandrogenism, ovulatory dysfunction, and polycystic ovaries in ultrasound (1). Since PCOS often begins during adolescence, early recognition of its clinical and biochemical features is essential in preventing

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long-term metabolic and reproductive complications. In adolescents, the diagnosis of PCOS is challenging due to the physiological overlap between normal pubertal development and PCOS-related features, such as menstrual irregularity and acne (1,2).

Regardless of age and body mass index (BMI), women with PCOS have an increased risk of developing cardiovascular disease, impaired glucose metabolism, metabolic syndrome, Type-2 diabetes, and obstructive sleep apnea (1-3).

In addition to its hormonal irregularities, PCOS is increasingly recognized as a systemic disorder associated with metabolic abnormalities and chronic inflammation (4,5). The relationship between PCOS and chronic inflammation is complex and remains unclear (5).

Routinely measured biochemical and hematological markers, as well as their derived ratios, can be used to assess systemic inflammation in various diseases (6-10).

Previous studies have demonstrated elevated levels of inflammatory markers, such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio (LMR), mean platelet volume (MPV), high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-a) and systemic immune-inflammation index (SII) in women with PCOS (4,5,11-15).

However, studies focusing on adolescents with PCOS are limited, despite the critical time of adolescence for early metabolic and cardiovascular risk assessment. Therefore, the aim of our study was to evaluate inflammatory markers in adolescents with PCOS, and to assess the associations between hyperandrogenism and systemic inflammation in this population.

Materials and Methods

Study Design and Patients

We retrospectively analyzed 108 patients with PCOS followed and treated at the Clinic of Pediatric Endocrinology, University of Health Sciences Türkiye, İzmir Dr. Behçet Uz Children's Diseases and Surgery Training and Research Hospital, between January 2023 and December 2025. Patients with missing data and those with chronic or other endocrine disorders were excluded, resulting in 89 patients included in the final analysis. This study was conducted in accordance with the Declaration of Helsinki, and approved by the University of Health Sciences Türkiye, İzmir Dr. Behçet Uz Children's Diseases and Surgery Training and Research Hospital Non-Interventional Research Ethics Committee (approval number: 2026/01-06, date: 15.01.2026). Informed consent was obtained from all subjects and parents involved in this study. Written informed consent was obtained from the patients and parents to publish this paper. All patients were followed up in our pediatric endocrinology clinic at a tertiary referral hospital in Western Türkiye. In accordance with international adolescent-specific guidelines, PCOS diagnosis required the presence of persistent menstrual irregularity for a period of at least 2 years from the time of menarche and hyperandrogenism (clinical or biochemical), following the exclusion of other causes of hyperandrogenism, such as Cushing syndrome or congenital adrenal hyperplasia. Ovarian morphology was excluded from the diagnostic criteria, as polycystic ovarian morphology is common during normal pubertal development and lacks diagnostic specificity in adolescents (1,16). A structured questionnaire was used to systematically evaluate all clinical and laboratory data. The standard deviation scores (SDS) of weight, height, and BMI were calculated based on Turkish children's reference values (17). Obesity was defined as a BMI-SDS >2, according to established pediatric growth references (18).

Hormonal and Biochemical Measurements

All laboratory assessments were performed before the initiation of any medical treatment for PCOS. Hormone analyses including luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, dehydroepiandrosterone sulfate (DHEA-S), 17-hydroxyprogesterone (17-OHP), 11-deoxycorticosterone (11-DOC), 21-deoxycorticosterone (21-DOC), 1,4-androstenedione (1,4-AS), total testosterone and sex hormone-binding globulin (SHBG) were performed in the follicular phase of the menstrual cycle after overnight fasting. The free androgen index (FAI) was calculated using the following formula: Total testosterone/SHBG×100. Hyperandrogenism was defined as a FAI >6 and/or DHEA-S >200 µg/dL and/or the presence of clinical hyperandrogenism, which is in accordance with previous recommendations and the published literature (1,16,19).

Based on peripheral blood cell counts, systemic inflammation markers were calculated; SII, NLR, and PLR. The calculations were as follows;

- SII = (neutrophils x platelets)/lymphocytes,
- NLR = neutrophils/lymphocytes,
- PLR = platelets/lymphocytes.

Statistical Analysis

Statistical analyses of the data were performed using the SPSS software package for Windows (Ver. 25.0; SPSS Inc., Chicago, IL, USA). The distribution of the data was evaluated using the Shapiro-Wilk test. For numerical comparisons, Student's t-test or Mann-Whitney U tests were used to assess differences between the two groups according to the normal distribution of the measured parameters. Categorical variables were analyzed with the chi-square (χ^2) test. Data are presented as mean±SD or median and interquartile range (IQR, 25th-75th percentiles). The relationship between inflammatory markers and androgens was analyzed using Spearman correlation tests. In all statistical tests, p values <0.05 were considered as statistically significant.

Results

A total of 108 patients with PCOS were initially evaluated. Patients with missing data and those with chronic or other endocrine disorders were excluded, resulting in 89 patients included in the final analysis. The mean age at presentation was 15.4±1.3 years. Irregular menstrual cycles were the most common presenting symptom (92.1%), followed by hirsutism (32.5%), obesity (4.4%), acne (2.2%), and dysmenorrhea (1.1%). The median BMI was 24.27 kg/m^2 (IQR: 21.52-29.36), with a median BMI-SDS of 1.21 (IQR: 0.11-2.41). Based on BMI-SDS criteria, 43.8% of the patients were classified as obese. The demographic, clinical, and laboratory characteristics of the patients at presentation are summarized in Table I.

Table I. The demographic, clinical, and laboratory characteristics of the patients

Age (years)	15.41±1.33
Weight, SDS*	1.21±1.91
Height, SDS*	-0.07±1.03
BMI	24.27 (21.52-29.36)
BMI-SDS	1.21 (0.11-2.41)
Obesity (%)	43.8
Glucose (mg/dL)	89 (86-92)
Insulin	15.59 (11.91-21.53)
HOMA-IR	3.03 (1.67-4.32)
Triglyceride (mg/dL)	87.55 (63-122)
Total cholesterol (mg/dL)	154.20 (137.65-171.82)
LDL (mg/dL)	80 (67-100)
HDL (mg/dL)	52.00 (43.50-58.75)

*Normal distribution (Shapiro-Wilk test)
Data are given as mean±SD or median (IQR: 25th-75th percentile).
SDS: Standard deviation score; BMI: Body mass index; HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, LDL: Low-density lipoprotein HDL: high-density lipoprotein, IQR: Interquartile range

The hormonal characteristics of the study population at presentation are summarized in Table II. Gonadotropin levels showed a median LH concentration of 8.10 U/L (IQR: 5.58-11.90) and a mean FSH concentration of 5.65±1.40 U/L, with a median LH/FSH ratio of 1.43 (IQR: 0.99-2.02). Median levels of total testosterone, FAI, and DHEA-S were 37.52 ng/dL (IQR: 29.20-56.00), 7.63 (IQR: 0-12.94), and 263.50 µg/dL (IQR: 198.12-325.50), respectively. Levels of adrenal androgens, including 17-OHP, 11-DOC, and 21-DOC, are also presented in Table II. Hyperandrogenism was identified in 73% of the adolescents with PCOS.

The median values of inflammatory markers SII and NLR were 520.80 (IQR: 364.57-659.70) and 1.62 (IQR: 1.28-2.24), respectively, while the mean PLR was 121.88±30.54. Spearman correlation analysis revealed no statistically significant associations between androgen levels and inflammatory markers, including NLR, PLR, and SII (all p>0.05) (Table III). In addition, inflammatory markers did not differ significantly between those adolescents with and those without hyperandrogenism (all p>0.05) (Table IV).

No significant correlations were observed between BMI and BMI-SDS and inflammatory markers, including NLR, PLR, and SII (all p>0.05). Moreover, inflammatory markers did not differ significantly between adolescents with PCOS according to obesity status (all p>0.05) (Table V). homeostasis model assessment of insulin resistance

Table II. Hormonal data of the patients

LH (U/L)	8.10 (5.58-11.90)
FSH* (U/L)	5.65±1.40
LH/FSH ratio	1.43 (0.99-2.02)
Estradiol (ng/dL)	36.15 (29.85-47.55)
Total testosterone (ng/dL)	37.52 (29.20-56.00)
DHEA-S (µg/dL)	263.50 (198.12-325.50)
11-DOC (ng/dL)	37.92 (22.79-74.67)
21-DOC (ng/dL)	2.38 (1.69-3.70)
17-OHP (ng/mL)	0.71 (0.40-1.39)
1,4-AS (ng/mL)	3.29 (2.19-4.93)
FAI	10.58 (5.5-16.68)

*Normal distribution (Shapiro-Wilk test)
Data are given as mean±SD or median (IQR: 25th-75th percentile)
LH: Luteinizing hormone; FSH: Follicle-stimulating hormone; DHEA-S: Dehydroepiandrosterone sulfate; 11-DOC: 11-deoxycorticosterone; 21-DOC: 21-deoxycorticosterone; 17-OHP: 17-Hydroxyprogesterone; 1,4-AS: 1,4-androstenedione; FAI: Free androgen index, SD: Standard deviation, IQR: Interquartile range

Table III. Spearman correlation coefficients between inflammation markers and androgen levels

	NLR		PLR		SII	
	Spearman r'	p value	Spearman r'	p value	Spearman r'	p value
Total testosterone	-0.010	0.931	-0.019	0.872	0.041	0.722
DHEA-S	-0.054	0.651	-0.007	0.955	-0.024	0.840
11-DOC	-0.154	0.350	0.141	0.392	-0.100	0.544
21-DOC	-0.277	0.088	-0.108	0.511	-0.271	0.095
17-OHP	0.029	0.813	0.005	0.965	0.007	0.955
1,4-AS	-0.039	0.741	-0.021	0.862	0.028	0.817
FAI	-0.133	0.272	-0.106	0.384	-0.235	0.051

p<0.05 indicates a statistically significant correlation
DHEA-S: Dehydroepiandrosterone sulfate; 11-DOC: 11-deoxycorticosterone; 21-DOC: 21-deoxycorticosterone; 17-OHP: 17-hydroxyprogesterone; 1,4-AS: 1,4-androstenedione; FAI: Free androgen index, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SII: Systemic immune-inflammation index

Table IV. Inflammatory markers in adolescents with PCOS, comparison according to hyperandrogenism

	Total	Hyperandrogenism (+) (n=65)	Hyperandrogenism (-) (n=24)	p value
SII	520.80 (364.57-659.70)	486.16 (373.15-652.08)	556.59 (340.94-657.36)	0.900
NLR	1.62 (1.28-2.24)	1.61 (1.27-2.23)	1.87 (1.28-2.23)	0.707
PLR*	121.88±30.54	122.51±29.82	120.37±32.48	0.597

*Normal distribution (Shapiro-Wilk test)
p<0.05 indicates statistically significant
SII: Systemic immune-inflammation index, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, PCOS: Polycystic ovary syndrome

Table V. Inflammatory markers in adolescents with PCOS: comparison between patients with and without obesity

	Total	Obese (n=39)	Non-obese (n=50)	p value
SII	520.80 (364.57-659.70)	513.60 (402.51-718.50)	534.01 (341.55-651.67)	0.372
NLR	1.62 (1.28-2.24)	1.66 (1.35-2.39)	1.61 (1.24-2.13)	0.260
PLR*	121.88±30.54	117.89±28.95	125.69±31.03	0.268

*Normal distribution (Shapiro-Wilk test)
p<0.05 indicates statistically significant
SII: Systemic immune-inflammation index, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, PCOS: Polycystic ovary syndrome

(HOMA-IR) showed a weak but statistically significant positive correlation with SII (r=0.234, p=0.038), whereas no significant correlations were observed between HOMA-IR and NLR or PLR.

Discussion

In this study, we investigated the relationship between androgen levels and inflammatory markers in adolescents with PCOS. Our findings demonstrate that inflammatory markers, including NLR, PLR, and SII, were not significantly associated with androgen levels. Furthermore, these markers did not differ according to the presence of obesity or hyperandrogenism.

Low-grade chronic inflammation has been increasingly recognized as a component of PCOS pathophysiology in adult populations (4,11,12,14,15). Previous studies have reported elevated levels of inflammatory markers, including hs-CRP, NLR, PLR, MPV, SII, SIRI and cytokines in women with PCOS compared to healthy controls, supporting the concept of an inflammatory environment of the disease (4,5,11,13,14). However, most data are from adult cohorts, in whom long-term metabolic abnormalities, obesity, and IR are more prevalent.

In the largest population-based study evaluating SII in children and adolescents, which included 4,134 children aged 6-19 years, the mean SII was reported as 355.71 (IQR:

255.15-492.00), while children with obesity exhibited higher SII values (10). Notably, the median SII observed in our cohort of adolescents with PCOS [528 (IQR: 366.88-655.69)] was higher than the general pediatric population and it was comparable to the levels reported in children with obesity. This finding suggests that adolescents with PCOS may exhibit a degree of low-grade systemic inflammation similar to that observed in pediatric obesity.

In previous studies, BMI has been consistently shown to be associated with inflammatory markers, particularly the SII (10,12,20). Furthermore, elevated inflammatory markers have also been reported in children with obesity, especially among those exhibiting features of metabolic syndrome (21). In contrast, we found no significant associations between BMI or BMI-SDS and inflammatory markers. Moreover, there were no significant differences in NLR, PLR, or SII between adolescents with PCOS according to their obesity status. This suggest that obesity alone may not be enough to trigger systemic inflammation in adolescence. It is likely that the duration of obesity and metabolic stress plays a critical role, with inflammatory consequences becoming more significant the longer the disease is present in adulthood.

IR plays a central role in the pathophysiology of PCOS and is strongly associated with its clinical manifestations. Consequently, women with PCOS are at increased risk of metabolic syndrome, type 2 diabetes mellitus, cardiovascular disease, and possibly higher cardiovascular mortality (1,4). In our study, in contrast to the lack of association with obesity, HOMA-IR demonstrated a weak but statistically significant positive correlation with SII. This suggests that IR may be a key factor in the early development of systemic inflammation in adolescents with PCOS.

While Bulu et al. (11) reported a significant positive association between PLR and HOMA-IR, we did not observe a similar relationship in our adolescent PCOS cohort, suggesting potential differences in inflammatory responses according to age.

Studies conducted in adult populations have suggested inflammation could be correlated with circulating androgens (5,22,23). In their review, Dey et al. (5) highlighted the association of both hematological inflammatory markers and pro-inflammatory cytokines with hyperandrogenism. In a retrospective cross-sectional study including women aged 18-40 years diagnosed with PCOS, inflammatory markers, particularly SII differed significantly across PCOS phenotypes with most elevated in phenotypes characterized by hyperandrogenism. In addition, correlation analyses indicated positive associations of NLR and SII with total testosterone and FAI (13). Similarly, Padder et al. (24) reported that hyperandrogenic PCOS phenotypes exhibited significantly higher levels of inflammatory cytokines. In another study, NLR was identified as an inflammation marker in patients with PCOS, exhibiting a positive correlation with free testosterone and 1,4-AS levels (25). In contrast, a study evaluating 392 women with hirsutism found no significant relationship between circulating androgen levels and NLR values (26). Similarly, our findings demonstrate that inflammatory markers were not significantly associated with hyperandrogenism or androgen levels. The lack of significant differences in inflammatory markers between adolescents with and those without hyperandrogenism further underscores the complexity of PCOS pathophysiology during adolescence. Pubertal hormonal fluctuations and physiological changes in androgen production may prevent the identification of the relationship between androgen concentrations and systemic inflammation at this age. This may partly explain why associations observed in adult women with PCOS are not consistent in the adolescent cohort.

Study Limitations

This study had certain limitations. Firstly, it was a retrospective, single-center study, which may limit its generalizability. Secondly, the absence of a healthy control group limits comparisons with those adolescents without PCOS. Although our sample size was comparable to previous adolescent PCOS studies, limited statistical power may have reduced the ability to detect subtle associations. In addition, age-related hormonal variability during adolescence, including pubertal fluctuations in androgen production and dynamic changes in sex hormone-binding globulin levels, may further obscure potential relationships between circulating androgen concentrations and systemic inflammation. Additionally, inflammatory status was assessed using hematological inflammatory markers rather than cytokine-based markers.

Conclusion

In conclusion, inflammatory markers in adolescents with PCOS appear to be independent of androgen excess and obesity, while IR may represent an early driver of low-grade systemic inflammation. Although elevated inflammation markers have been linked to systemic inflammation in chronic diseases and shown to be increased in women with PCOS in previous studies, its clinical relevance in adolescents with PCOS remains less clear. Early identification and diagnosis of PCOS during adolescence are of critical

importance, given the strong association of PCOS with long-term metabolic abnormalities.

Ethics

Ethics Committee Approval: This study was approved by the University of Health Sciences Türkiye, İzmir Dr. Behçet Uz Children's Diseases and Surgery Training and Research Hospital Non-Interventional Research Ethics Committee (approval number: 2026/01-06, date: 15.01.2026).

Informed Consent: Informed consent was obtained from all subjects and parents involved in this study. Written informed consent was obtained from the patients and parents to publish this paper.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ö.K., G.A.K., İ.M.E., Ö.N., H.A.K., B.Ö., Concept: Ö.K., B.Ö., Design: Ö.K., Data Collection or Processing: Ö.K., N.P., G.A.K., İ.M.E., Ö.N., H.A.K., Analysis or Interpretation: Ö.K., Literature Search: Ö.K., Writing: Ö.K., B.Ö.

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