

Original Article

Serum uric acid levels in patients with different phenotypes of polycystic ovary syndrome

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Background & objectives: Polycystic ovary syndrome (PCOS) is a common endocrinopathy in reproductive-aged women, frequently associated with insulin resistance, obesity, and increased cardiovascular risk. Serum uric acid, linked to oxidative stress and endothelial dysfunction, may contribute to the cardiometabolic complications of PCOS. Although hyperuricemia is common in PCOS, differences in serum uric acid among phenotypes remain unclear. This study compared serum uric acid across PCOS phenotypes, hypothesising that classic, obese phenotypes would exhibit higher levels due to synergistic metabolic and hormonal effects.

Methods: A total of 180 women with PCOS [phenotypes A(n=96), B (n=19), C (n=35), and D (n=30)] and 51 age-matched controls were included. PCOS was diagnosed according to the revised 2003 Rotterdam criteria, and phenotyping was based on the presence of hyperandrogenism, oligo-anovulation, and polycystic ovarian morphology. Fasting serum uric acid levels were measured using the uricase method, and hyperuricemia was detected as ≥ 6.0 mg/dL. Analyses were performed using the Shapiro–Wilk test for normality, Levene’s test for homogeneity, and appropriate parametric or non-parametric tests with Holm correction for multiple comparisons.

Results: Among 231 women evaluated, age distributions were similar between groups. Median body mass index (BMI) and waist circumference (WC) were higher in the PCOS cohort, most prominently in Phenotype A (both $P < 0.001$). Serum uric acid was significantly higher in Phenotype A [4.8 (2.7–8.9) mg/dL] than in controls [4.1 (2.7–6.3) mg/dL] and Phenotype C [4.2 (2.6–5.8) mg/dL] (both $P < 0.001$). Hyperuricemia was more frequent in PCOS than in controls ($P = 0.035$), occurring predominantly in Phenotype A. Most hyperuricemic patients were obese; notably, no obese controls were available for comparison. Serum uric acid levels were positively associated with BMI (Spearman $\rho = 0.452$, +) and WC (0.412, $P < 0.001$), yet in BMI categories, no significant differences were found in serum uric acid among phenotypes.

Interpretation & conclusions: Among PCOS patients, elevated serum uric acid is significantly associated with the classic hyperandrogenic phenotype (Phenotype A) and may also be related to obesity, emphasising the importance of monitoring serum uric acid in obese classical PCOS females, particularly those presenting with Phenotype A.

Key words Body mass index - hyperuricemia - phenotype - polycystic ovary syndrome - uric acid

Polycystic ovary syndrome (PCOS) is an endocrinological disease that affects 5-20 per cent of women of reproductive age. PCOS is characterised by oligo-anovulation (OA), clinical or biochemical hyperandrogenemia (HA), and polycystic ovaries (PCO) appearing on ultrasonography (USG)^{1,2}. PCOS is also diagnosed under the revised 2003 Rotterdam criteria^{1,2}, and is often accompanied by the features of metabolic syndrome, such as dyslipidaemia, hypertension, insulin resistance (IR), obesity, and glucose intolerance³⁻⁵. Increased inflammation is also one of the main features of PCOS^{6,7}. Until recently, PCOS was viewed as a uniform entity; however, accumulating evidence indicates that the degree of metabolic dysregulation and inflammatory activation differs among its phenotypes⁸. For this reason, Phenotypes A (HA+OA+PCO), B (HA+OA), C (HA+PCO), and D (OA+PCO) are the four phenotypes of PCOS determined by combinations of HA, OA, and PCO. The severity and frequency of associated metabolic problems and inflammation in PCOS diminish as the phenotypic letters move from A to D⁸.

Uric acid, the final product of purine metabolism, is a major plasma antioxidant with dual biological effects, exerting protective antioxidant activity extracellularly, yet demonstrating pro-oxidant and potentially harmful actions once it enters cells^{9,10}. Elevated serum uric acid enhances oxidative stress through lipid peroxidation and low-density lipoprotein cholesterol oxidation, promoting cardiovascular disease^{9,11}. Moreover, serum uric acid levels correlate positively with insulin resistance, and hyperuricemia is regarded as a potential marker of insulin resistance¹².

Previous studies investigating the relationship of PCOS with serum uric acid have revealed conflicting results. While some reported a markedly higher prevalence of hyperuricemia, up to 40 per cent, particularly with increasing obesity, others found serum uric acid levels comparable to those of healthy controls¹³⁻¹⁸. However, most studies were conducted in unclassified PCOS cohorts without considering phenotypic differences. This study aimed to compare serum uric acid levels across PCOS phenotypes to determine whether serum uric acid elevation is driven primarily by obesity and/or by phenotype-specific factors.

Materials & Methods

Designed prospectively as a case-controlled study, the present study was conducted in the departments of Internal Medicine, Endocrinology and metabolic diseases, and Obstetrics and Gynaecology, Usak

University, the training and research hospital between April 1, 2022, and July 20, 2023. The study was approved by the Ethics Committee of Usak University School of Medicine and also registered at ClinicalTrials.gov (Registration No: NCT06342180). After obtaining informed consent from *all individual participants*, women between 18–35 yr of age and with the diagnosis of PCOS under the 2003 Rotterdam criteria² were included in light of the biochemical, hormonal, and USG criteria. Participants with no known malignancy, hepatic or renal failure, gout, hypertension, active infection, or use of medications affecting serum uric acid or insulin resistance, and other endocrine diseases such as congenital adrenal hyperplasia, the existence of androgen-secreting tumours or Cushing syndrome, were excluded from the study².

Diagnostic criteria for polycystic ovary syndrome: The diagnostic criteria of PCOS consisted of the presence of two of these three criteria: (i) OA [oligomenorrhea (cycle length >35 days or <8 menstrual bleeding/year) or amenorrhea (absence of menses >3 months)], (ii) Clinical [hirsutism (the Ferriman–Gallwey scoring system was applied, with a total score ≥ 8 considered diagnostic for the presence of hirsutism) and/or acne] and/or biochemical [increased total testosterone levels or dehydroepiandrosterone sulfate (DHEAS)] signs of HA, and (iii) PCO appearance (the presence of 12 or more follicles in each ovary measuring 2-9 mm in diameter, and/or increased ovarian volume >10 mL) on USG. Biochemical hyperandrogenaemia was assessed by measuring DHEA-S (1.9–12.5 IU/L) using chemiluminescent immunoassays on the Snibe Maglumi 4000 Plus analyser (Shenzhen, China), and total testosterone (12–60 ng/dL) using the Abbott Advia Centaur system (Tarrytown, New York, USA).

Study population and sample size calculation: *A priori* sample size estimation was carried out through G*Power software (version 3.1.9.7). According to the study by Mu *et al*¹⁴, women with polycystic ovary syndrome (PCOS) exhibited significantly higher serum uric acid levels than controls (mean difference=0.64 mg/dL, $P<0.001$). Similarly, Yarali *et al*¹⁹ reported a comparable difference between PCOS and control groups (mean difference=0.70 mg/dL, $P=0.04$). Based on these data, the effect size (d) for the primary comparison between PCOS and control groups was determined as 0.7, corresponding to a large effect. Considering that PCOS cases were further categorised into four phenotypes according to the

Rotterdam criteria, an allocation ratio of 1:4 (control: PCOS) was applied. Using a two-tailed $\alpha=0.05$ and statistical power=0.80, minimum total sample size required was calculated to be 104 participants (21 controls and 83 PCOS cases). However, despite this sample size estimation, a larger number of patients and controls took part in the presented study to enhance the robustness of the analysis. Based on these criteria, patients were categorised into four phenotypes: Phenotypes A, B, C, and D, including 96, 19, 35, and 30 patients, respectively. A healthy control group of 51 age-matched and meeting the exclusion criteria women (18-35 yr) was included; specifically, they had no diagnosis of PCOS, and history of known cancers, or liver/kidney failures, on no medications to affect their serum uric acid and insulin resistance levels; and presented with no active infections, hirsutism, acne, polycystic ovaries (on USG), or irregular menstrual cycles.

Weight, height, waist circumference (WC), and presence of hirsutism was reassessed by the same investigator (CD). Fasting blood samples for serum uric acid measurement were collected, centrifuged, and stored at -70°C till all samples were analysed. The serum uric acid levels (2.5-6.2 mg/dL) were evaluated using the uricase method and the Architect C series (Abbott Co., Lake Forrest, IL, USA), and the level of serum uric acid ≥ 6 mg/dL was considered hyperuricemia²⁰. Furthermore, overweight and obesity were acquired under the formula suggested by WHO (BMI 25.0–29.9 kg/m² and ≥ 30.0 kg/m², respectively), and patients were categorised into corresponding groups based on these definitions. All participants were re-evaluated by the same researcher (OO) for ovarian PCO morphology using a Mindray DC-7 device with a 3–6.6 MHz transabdominal and/or 3–5 MHz transvaginal transducer (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China).

Statistical analysis: The data were analysed with the Statistical Package for Social Sciences for Windows, v22.0 (IBM Inc., Chicago, IL, USA). The normally distributed data and variance homogeneity were investigated with the Shapiro–Wilk and Levene’s tests, respectively. Continuous variables were summarised as median [Q1, Q3]; mean \pm standard deviation was also provided when assumptions were met. For comparisons between two groups, the student’s t-test (or Welch’s t-test when variances were unequal) was applied if assumptions were satisfied, and the Mann-Whitney U

test otherwise. For comparisons among more than two groups, one-way ANOVA (or Welch’s ANOVA when appropriate) was performed when assumptions were met, and the Kruskal–Wallis test otherwise. When omnibus tests were significant, pairwise comparisons were conducted using Tukey HSD or Games–Howell following ANOVA, and Dunn’s test after Kruskal–Wallis, with *P*-values adjusted by the Holm method. For categorical variables, Pearson’s chi-square test was used when expected frequencies were adequate, and exact methods (*e.g.*, Fisher’s test) when they were not. All tests were two-tailed, with a statistically significant value of $P \leq 0.05$.

Results

A total of 231 women were evaluated (180 PCOS, 51 controls). Age did not differ between groups. Median BMI and WC values were found significantly elevated in the PCOS cohort than in the control group ($P < 0.001$). Among phenotypes, elevations were most pronounced in Phenotype A. For BMI, Phenotype A exceeded controls ($P < 0.001$), Phenotype C and D ($P < 0.001$ and $P = 0.050$, respectively), whereas A-B and B-C comparisons were not significant. For WC, Phenotype A exceeded controls and Phenotype C (both $P < 0.001$) and was also higher than Phenotype D ($P = 0.044$). Regarding serum uric acid, the overall PCOS group had higher levels than the controls ($P < 0.001$); only Phenotype A differed from the controls ($P < 0.001$). Serum uric acid in Phenotype A was higher than in C ($P < 0.001$) but not different from B or D (Table I).

Consistent with these findings, levels of serum uric acid revealed no significant difference across PCOS phenotypes within individual BMI categories (Supplementary Table). Obesity distribution analysis showed that obese individuals were markedly overrepresented among women with PCOS, particularly in Phenotype A (44.8%) and Phenotype B (36.8%), whereas normal-weight individuals were relatively underrepresented. When stratified by BMI, most hyperuricemic cases in PCOS were concentrated in the obese subgroup. In Phenotype A, 15 of 21 hyperuricemic patients (71.4%) were obese, while only one case occurred in the normal-weight subgroup. Similar patterns were observed in Phenotypes B and D, where all hyperuricemic patients were either overweight or obese. No obese participants were present in the control group, precluding direct comparison (Supplementary Table).

Table I. Demographic and laboratory data in controls and PCOS phenotypes (with adjusted pairwise comparisons)

	Control (n=51)	Phenotype A (n=96)	Phenotype B (n=19)	Phenotype C (n=35)	Phenotype D (n=30)	Total PCOS (n=180)	<i>p</i> (Kruskal-Wallis)	<i>p</i> 1 (Control-A)	<i>p</i> 2 (Control-B)	<i>p</i> 3 (Control-C)	<i>p</i> 4 (Control-D)	<i>p</i> 5 (A-B)	<i>p</i> 6 (A-C)	<i>p</i> 7 (A-D)	<i>p</i> 8 (B-C)	<i>p</i> 9 (B-D)	<i>p</i> 10 (C-D)	<i>p</i> 11 (Control-Total PCOS)	
Age (yr)	23.0 (23.0, 24.5)	25.0 (22.0, 29.3)	22.0 (20.0, 28.0)	25.0 (22.0, 28.0)	24.0 (23.0, 28.8)	24.0 (22.0, 29.0)	0.337	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	0.458
BMI (kg/m ²)	21.5 (20.0, 24.1)	27.2 (23.3, 35.9)	24.8 (21.1, 32.5)	22.2 (20.9, 25.1)	24.9 (21.2, 27.2)	25.5 (21.6, 32.4)	<0.001	<0.001	0.201	0.587	0.050	0.560	<0.001	0.201	0.587	0.773	0.587	0.587	<0.001
Waist circumference (cm)	75.0 (71.0, 81.0)	89.5 (78.0, 103.3)	82.0 (74.5, 94.00)	77.0 (70.5, 88.0)	78.0 (70.3, 93.8)	83.5 (74.8, 99.3)	<0.001	<0.001	0.292	0.953	0.807	0.396	<0.001	0.044	0.953	1.0	1.0	1.0	<0.001
serum uric acid (mg/dL)	4.1 (3.6, 4.6)	4.8 (4.1, 5.8)	4.7 (4.1, 5.6)	4.2 (3.7, 4.9)	4.3 (3.9, 5.1)	4.6 (4.0, 5.5)	<0.001	<0.001	0.159	1.0	0.489	1.0	<0.001	0.258	0.385	1.0	1.0	1.0	<0.001

Values are presented as median [Q1, Q3]. For each variable, 'Global *P*' indicates the results of ANOVA or Kruskal-Wallis, depending on assumption suitability. Pairwise *P*-values (*P*₁ to *P*₁₁) are reported with Holm correction (adj. *p*) applied to Mann-Whitney U, BMI, body mass index; Control, control group; A-D, PCOS phenotypes

The prevalence of hyperuricemia (≥ 6 mg/dL) was significantly higher in women with PCOS than in controls (14.4% vs 2.0%, $P=0.035$). Among phenotypes, the highest rate was observed in Phenotype A (22.9%, $P=0.005$ vs controls), followed by Phenotypes B (10.5%) and D (6.7%), whereas no hyperuricemia was detected in Phenotype C (Table II).

Correlation analyses supported these findings: Serum uric acid levels were not significantly associated with age (Spearman rho = 0.035, $P = 0.601$), whereas significant positive correlations were observed with BMI (Spearman rho = 0.452, $P < 0.001$) and WC (Spearman rho = 0.412, $P < 0.001$).

Discussion

We found that the frequency of hyperuricemia was higher among women with PCOS than in the controls. Serum uric acid levels were elevated, particularly in Phenotype A compared with the controls and Phenotype C, while Phenotypes B and D showed no statistically significant difference. The prevalence of hyperuricemia was greater in Phenotype A, as compared to the control group and Phenotype C. Most hyperuricemic patients in our cohort were clustered within the obese and overweight subgroups of Phenotype A.

Hyperuricemia is a common metabolic disorder encountered in PCOS patients¹³⁻¹⁵. In prevalence studies, the hyperuricemia frequency has been shown as 2-3 times higher in PCOS patients than in the controls, depending on the degree of obesity^{14,15}. Consistent with these observations, the present study demonstrated that hyperuricemia was predominantly clustered within the obese subgroup, increasing progressively with obesity severity. However, these studies were conducted in unstratified PCOS populations, whereas the current study provides a more refined, phenotype-specific analysis.

In our study, no correlation was detected between serum uric acid levels and age, whereas serum uric acid concentrations were positively associated with both BMI and waist circumference, consistent with previous findings^{16,21}. In one of our previous studies stratified by obesity status, serum uric acid levels in obese PCOS patients were comparable to those in obese controls, whereas non-obese PCOS patients exhibited higher serum uric acid concentrations than non-obese controls¹⁶. Although obesity is widely recognised as a major determinant of hyperuricemia,

Table II. Frequency of hyperuricemic patients in PCOS phenotypes and the control group

	Hyperuricemia		<i>p</i> 1	<i>p</i> 2	<i>p</i> 3	<i>p</i> 4
	No (%)	Yes (%)				
The control group	50 (98)	1 (2)				
Phenotype A	74 (77.1)	22 (22.9)	0.005			
Phenotype B	17 (89.5)	2 (10.5)	0.197	0.530		
Phenotype C	35 (100)	0 (0)	1	0.004	0.136	
Phenotype D	28 (93.3)	2 (6.7)	0.555	0.107	1	0.224
Total PCOS cases	154 (85.6)	26 (14.4)	0.035			

PCOS, Polycystic ovary syndrome; *p*1, Comparisons between the control group and PCOS Phenotypes; *p*2, Comparisons between Phenotype A, and Phenotypes B, C, and D; *p*3, Comparisons between Phenotype B, and Phenotypes C and D; *p*4, Comparisons between Phenotypes C and D

these findings suggest that PCOS itself may exert an independent effect on serum uric acid elevation. In our study, obese women were overrepresented across all PCOS phenotypes, particularly in Phenotypes A and B. Hyperuricemia was observed predominantly among obese PCOS patients, less frequently in overweight individuals, and rarely in those of normal weight. When serum uric acid was compared between PCOS and controls within underweight, normal-weight, and overweight subgroups, no significant differences were observed, and no obese controls were available for direct comparison. Furthermore, serum uric acid levels did not differ significantly across PCOS phenotypes within each BMI category, indicating that inter-phenotypic variations were not solely driven by adiposity. This observation aligns with previous studies reporting higher serum uric acid levels in lean PCOS women than BMI-matched controls and suggests that, beyond adiposity, intrinsic hormonal or metabolic disturbances may contribute to serum uric acid elevation in PCOS²². However, these findings contrast with studies reporting comparable serum uric acid levels between PCOS and control groups after adjustment for obesity^{17,18}.

Although previous studies have reported conflicting results regarding whether obesity or PCOS itself is a more dominant feature in the development of hyperuricemia, our findings indicate that serum uric acid elevation is most evident in the classical PCOS phenotype and is positively associated with adiposity. These results suggest that both metabolic and hormonal factors may contribute to increased serum uric acid levels in PCOS.

Androgen excess may partly explain the phenotype-specific differences observed²³⁻²⁵. Experimental data indicate that androgens enhance hepatic purine synthesis and renal uric acid reabsorption, whereas

anti-androgen therapy can reduce serum uric acid levels^{18,26,27}. Accordingly, the elevated serum uric acid levels seen in Phenotype A likely reflect the impact of biochemical or clinical hyperandrogenism, suggesting that androgen excess contributes to the increased uric acid burden characteristic of this phenotype.

Inflammation, a hallmark of PCOS, varies among phenotypes and is most prominent in the classic hyperandrogenic form (Phenotype A). Considering that inflammation may exacerbate oxidative and endothelial stress, both linked to uric acid metabolism²⁸⁻³⁰, and that hyperandrogenaemia may also elevate serum uric acid^{14,18}, the higher serum uric acid levels observed in Phenotype A may be related to the combined effects of inflammation and androgen excess. As both inflammatory activity and hyperandrogenism tend to decrease progressively from Phenotype A to D, the predominance of hyperuricemia in Phenotype A may represent a biologically plausible association rather than a causal relationship.

As there were no obese individuals in the control group, preventing direct comparison with obese PCOS patients, this represents one of the main limitations of our study. The absence of data regarding anti-androgen treatment represents the limitation of the present study, as its potential impact on serum uric acid concentrations could not be evaluated. Another limitation of the study is the unequal phenotype distribution, with particularly small sample sizes for Phenotypes B and D. Nevertheless, Phenotype A constituted the largest and most metabolically active subgroup, consistent with a prior meta-analysis reporting that classic Phenotypes A and B collectively account for approximately 66 per cent of all PCOS cases³¹. Although the overall sample size was sufficient for phenotype-based comparisons, subgroup sizes

decreased substantially after stratification by obesity categories, which may have reduced the reliability of these subgroup analyses. Additional limitations include the lack of control for dietary confounders such as purine, fructose, or alcohol intake. Furthermore, the absence of biochemical assessment of insulin resistance limits the interpretation of the metabolic mechanisms underlying elevated serum uric acid levels in PCOS. We consider that novel studies with larger populations will provide more accurate findings.

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Conflicts of Interest: None.

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