Available online at www.medicinescience.org

ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2026;15(3):1396-402

Relationship between vitamin D deficiency, polycystic ovary syndrome, and serum dehydroepiandrosterone levels

Sureyya Saridas Demir[1], Lale Vuslat Baki[2], Mehmet Nuri Duran[3], Sevilay Yavuz Dogu[4], Sule Birol Ince[5], Gul Karahan[6], Serem Kel Ilgin[7], Bulent Demir[8]

[1]*Private Clinic, Department of Perinatology, Çanakkale, Türkiye*
[2]*Memorial Şişli Hospital, Department of Obstetrics and Gynecology, İstanbul, Türkiye*
[3]*Ezine State Hospital, Department of Obstetrics and Gynaecology, Çanakkale, Türkiye*
[4]*Haseki Training and Research Hospital, Department of Obstetrics and Gynaecology, İstanbul, Türkiye*
[5]*Şişli Hamidiye Etfal Hospital, Department of Obstetrics and Gynecology, İstanbul, Türkiye*
[6]*Haydarpaşa Numune Hospital, Department of Obstetrics and Gynaecology, İstanbul, Türkiye*
[7]*Biga State Hospital, Department of Obstetrics and Gynaecology, Çanakkale, Türkiye*
[8]*Çanakkale Onsekiz Mart University, Faculty of Medicine, Department of Gynecology and Obstetrics, Çanakkale, Türkiye*

Received 26 March 2026; Accepted 20 May 2026
Available online 25 August 2026 with doi: 10.5455/medscience.2026.03.065

Abstract

Polycystic ovary syndrome (PCOS) is a multifaceted endocrine disorder characterized by heterogeneous clinical and metabolic features. Although previous research has frequently reported an association between vitamin D deficiency and PCOS, the clinical implications of this relationship remain uncertain. In particular, the potential interaction between vitamin D status, clinical manifestations, and dehydroepiandrosterone sulfate (DHEAS) levels has not been clearly established. In this study, we aimed to explore the relationship between serum vitamin D levels and DHEAS concentrations in women with PCOS, while also assessing their associations with key clinical parameters. A total of 230 women diagnosed according to the Rotterdam 2003 criteria were retrospectively evaluated. Patients were grouped according to vitamin D status as severe deficiency, deficiency, insufficiency, and sufficiency, and they were also classified according to normal or elevated DHEAS levels. Demographic characteristics, clinical findings, hormonal profiles, and biochemical parameters were obtained from electronic medical records, reflecting routine clinical practice. Statistical analyses were performed using appropriate parametric and non-parametric methods. Vitamin D deficiency was highly prevalent in the study population: severe deficiency was observed in 108 patients (47.0%), deficiency in 100 patients (43.5%), insufficiency in 13 patients (5.9%), and sufficiency in only 9 patients (4.1%). No statistically significant association was found between vitamin D categories and DHEAS levels ($p = 0.73$), infertility status ($p = 0.85$), hirsutism severity ($p = 0.68$), or menstrual interval ($p = 0.27$). Although reduced vitamin D levels were commonly observed, this did not translate into measurable differences in clinical outcomes. One possible explanation is the high baseline prevalence of vitamin D deficiency in the general population, which may obscure disease-specific effects. In addition, the deficiency lack of association between vitamin D status and DHEAS suggests that androgen excess in PCOS may be regulated by mechanisms independent of vitamin D.

Keywords: Polycystic ovary syndrome, vitamin D deficiency, dehydroepiandrosterone sulfate, hyperandrogenism, infertility, hirsutism

Introduction

Polycystic ovary syndrome (PCOS) is one of the most commonly encountered endocrine conditions in women of reproductive age. Rather than representing a single uniform disease, it reflects a complex and chronic disorder with a multifactorial background, which often poses challenges in routine clinical management. From a clinical perspective, PCOS should be considered a spectrum disorder, as patients may present with varying degrees of fertility, metabolic, and endocrine dysfunction. In routine clinical settings, the diagnosis is made by identifying a combination of key clinical features, including hyperandrogenism, menstrual cycle disturbances, chronic anovulation, and compromised

CITATION
Saridas Demir S, Baki LV, Duran MN, et al. Relationship between vitamin D deficiency, polycystic ovary syndrome, and serum dehydroepiandrosterone levels. Med Science. 2026;15(3):1396-402.

Corresponding Author: Bulent Demir, Çanakkale Onsekiz Mart University, Faculty of Medicine, Department of Gynecology and Obstetrics, Çanakkale, Türkiye
Email: bulentdemirmd@hotmail.com

DOI: 10.5455/medscience.2026.03.065

reproductive potential, together with supportive clinical and biochemical evidence. Beyond its reproductive manifestations, PCOS is frequently associated with a spectrum of metabolic abnormalities, including insulin resistance, obesity, dyslipidemia, and an increased risk of developing type 2 diabetes mellitus [1]. Because PCOS affects both reproductive and metabolic systems, it is a patient group frequently encountered in both gynecology and endocrinology practice. For diagnosis, the Rotterdam 2003 criteria are still widely used worldwide. Based on these criteria, and after ruling out alternative etiologies, the diagnosis requires the presence of at least two of the following findings: (1) oligo- or anovulation, (2) clinical or biochemical hyperandrogenism, and (3) polycystic ovarian morphology [2]. In recent years, anti-Müllerian hormone (AMH) has also used as an alternative biomarker, as it may reflect an increased number of active or dominant follicles and can serve as a supportive diagnostic indicator [3]. Although the prevalence of PCOS differs depending on the population studied and the diagnostic criteria applied, it is generally reported to range between 8% and 13% [4]. Given its high prevalence and chronic nature, understanding the underlying mechanisms of PCOS and improving its management remain important. While the exact contribution of vitamin D to PCOS remains incompletely understood, emerging evidence suggests that it may play a contributory role in the underlying pathophysiological mechanisms of the syndrome [5]. Vitamin D deficiency is widely known as a global public health concern. Vitamin D deficiency appears to occur more frequently among women with PCOS than in the general population. This observation may be related to the involvement of vitamin D in key metabolic and endocrine functions, such as glucose regulation, insulin dynamics, and inflammatory signaling pathways [6]. Insulin resistance is considered a key property in the pathophysiology of PCOS [7]. Low vitamin D density have been associated with metabolic disorders such as insulin resistance, obesity, menstrual irregularities, and hyperandrogenism. Vitamin D deficiency has suggested to further aggravate both metabolic and reproductive disturbances in these patients [8]. At the same time, insulin resistance may enhance ovarian androgen production and suppress hepatic synthesis of sex hormone binding globulin (SHBG), ultimately leading to increased circulating free androgen levels. These hormonal alterations, together with potential changes in vitamin D state, may contribute to the development of hyperandrogenism and its clinical manifestations. Vitamin D may also be relevant to infertility in PCOS through its potential effects on follicular development, endometrial receptivity, implantation, and metabolic regulation. Recent studies have reported associations between serum vitamin D levels and assisted reproductive treatment outcomes in women with PCOS, although the evidence remains heterogeneous. Since dehydroepiandrosterone sulfate (DHEAS) reflects adrenal androgen activity, evaluating vitamin D status together with DHEAS and infertility may provide a broader view of the reproductive and endocrine profile of women with PCOS [9,10]. In clinical settings, however, these mechanisms rarely operate in isolation and often overlap with multiple patient-specific factors. In daily practice, however, these relationships are not always straightforward, and individual patient profiles may vary considerably. For this reason, there is still a need for studies that evaluate biochemical parameters together with clinical outcomes in an integrated manner. From a practical perspective, clinicians are often faced with heterogeneous patient profiles, making it difficult to draw uniform conclusions. Although several studies have examined vitamin D status and androgen-related parameters in PCOS, the specific relationship between vitamin D and DHEAS has not been fully clarified. This distinction is clinically relevant because DHEAS mainly reflects adrenal androgen activity, which may contribute differently to the heterogeneous presentation of PCOS. Therefore, we aimed to evaluate the association between vitamin D status and DHEAS levels in women with PCOS and to examine whether vitamin D categories were related to infertility, hirsutism, menstrual interval, and BMI in real-world clinical practice.

Material and Methods

This multicenter retrospective study was conducted using data from women diagnosed with PCOS who were evaluated during the study period. Given the retrospective design, all eligible patients meeting the inclusion criteria during the study period were included in the analysis. Demographic and clinical information, including age, body mass index (BMI), presenting complaints, family history, menstrual characteristics, and ultrasonographic findings, was retrieved from the hospital's electronic medical record system. This approach enabled the analysis of real-world clinical data rather than controlled experimental conditions. Hormonal profiling was performed by evaluating prolactin, thyroid-stimulating hormone (TSH), and gonadotropins (FSH and LH), together with androgen-related markers, including total and free testosterone and DHEAS. Serum vitamin D levels were assessed separately as part of the metabolic evaluation. In addition, biochemical parameters were analyzed using data from the same database, including lipid profile components (LDL, HDL, triglycerides, and total cholesterol) as well as fasting blood glucose levels. Patients with systemic diseases or endocrine disorders that could affect hormonal status, including congenital adrenal hyperplasia, Cushing's syndrome, and adrenal tumors, were excluded from the study. In addition, individuals who had used hormonal therapy prior to the study period or medications known to influence biochemical parameters were not included in the analysis. Patients with documented use of vitamin D supplements, insulin-sensitizing agents, corticosteroids, anti-androgen treatments, or drugs affecting hormonal/metabolic parameters were excluded. Due to the retrospective design of the study and the use of anonymized patient records, written informed consent was waived by the ethics committee. Clinical hyperandrogenism was evaluated with the modified Ferriman-Gallwey (mFG) scores and scores of more than 8 were considered as hirsutism. Hirsutism grades were classified as normal (mFG score < 8), mild (mFG score 8 – 15), moderate (mFG score 16 – 29), severe (mFG score ≥ 30).

DOI: 10.5455/medscience.2026.03.065

BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²). Ultrasonographic assessments were performed using a Voluson P6 system (GE Medical Systems). Venous blood samples were collected between 08:00 and 09:00 a.m. during the early follicular phase (days 2–5 of the menstrual cycle) following an overnight fast of 10–12 hours. Hormonal analyses were conducted using a chemiluminescent microparticle immunoassay system (Architect analyzer, module C). Free testosterone levels were measured by radioimmunoassay using commercially available kits (DIAsource). Biochemical parameters—including fasting glucose, fasting insulin, and lipid profile components were measured using an automated clinical chemistry analyzer (Beckman Coulter AU5800). Following initial analyses, serum samples were centrifuged and stored at −40°C for subsequent vitamin D measurement. Vitamin D concentrations were determined using liquid chromatography–tandem mass spectrometry (LC–MS/MS). Based on serum levels, vitamin D status was categorized as severe deficiency (< 10 ng/mL), deficiency (11–20 ng/mL), insufficiency (21–30 ng/mL), and sufficiency (> 30 ng/mL). Age-specific reference ranges for DHEAS in females were defined as 45–320 µg/dL for ages 18–29 years, 40–325 µg/dL for ages 30–39 years, and 25–220 µg/dL for ages 40–49 years [11,12].

All statistical analyses were carried using IBM SPSS Statistics version 26. Continuous variables were presented as mean ± standard deviation or median with minimum–maximum values, according to their distribution. Categorical variables were presented as numbers and percentages. Normality was evaluated with the Shapiro Wilk test. For non-normally distributed data, MannWhitney U tests were applied, while categorical data was analyzed using the Chi-square test. Comparisons of vitamin D levels with BMI were performed using ANOVA. A two-tailed p value < 0.05 was considered statistically significant. In addition to the main group comparisons, univariate logistic regression analyses were performed for selected categorical outcomes in order to provide a clearer estimate of effect size. Crude odds ratios with 95% confidence intervals were calculated. For vitamin D-related analyses, severe vitamin D deficiency was accepted as the reference category. The evaluated outcomes included elevated DHEAS status, infertility, and moderate/severe hirsutism.

Results

A total of 230 women diagnosed with PCOS were included in the study. The mean age of the participants was 26.7 ± 5.6 years (median 26; range 15–40). Baseline anthropometric, metabolic, and endocrine characteristics are summarized in Table 1. The mean BMI was 28.9 ± 6 kg/m², and the average menstrual interval was 65.8 ± 49 days. The mean fasting glucose level was 96.3 ± 17.3 mg/dL, and the median fasting insulin level was 10 µIU/mL (range, 2–1987). Lipid profile parameters showed mean HDL, LDL, and total cholesterol levels of 47.6 ± 12.8 mg/dL, 105.4 ± 30.4 mg/dL, and 174.6 ± 38.2 mg/dL. The mean DHEAS level was 251.8 ± 97.9 µg/dL, while total and free testosterone levels were 1.3 ± 8.4 ng/mL and 2.8 ± 1.2 pg/mL, respectively. The mean serum vitamin D level was 13 ± 9.6 ng/mL. Mean levels of FSH, LH, estradiol, TSH, and prolactin were 6.7 ± 1.7 mIU/mL, 11 ± 5.4 mIU/mL, 59.8 ± 37.7 pg/mL, 2.3 ± 1.4 mIU/L, and 14 ± 5.9 ng/mL, respectively.

Table 1. Anthropometric, metabolic and endocrine characteristics of PCOS patients

Variable	Mean ± SD	Median (Min. – Max.)
Age	26.7 ± 5.6	26 (15 - 40)
BMI	28.9 ± 6	28.4 (17.6 – 47)
Menstrual interval	65.8 ± 49	45 (20 – 270)
Fasting glucose	96.3 ± 17.3	93 (70 – 264)
Insulin	19.8 ± 132.2	10 (2 – 1987)
HDL	47.6 ± 12.8	45 (8.4 – 146)
LDL	105.4 ± 30.4	102.5 (43.8 – 236)
Total cholesterol	174.6 ± 38.2	173 (0.6 – 318)
DHEAS	251.8 ± 97.9	235.3 (47 – 588)
Total testosterone	1.3 ± 8.4	0.6 (0.1 – 117)
Free testosterone	2.8 ± 1.2	2.5 (0.8 – 8.2)
Vitamin D	13 ± 9.6	10.6 (3 – 83.3)
FSH	6.7 ± 1.7	6.7 (2 – 11.1)
LH	11 ± 5.4	10.6 (1.1 – 37.7)
Estradiol	59.8 ± 37.7	54 (1 – 350)
TSH	2.3 ± 1.4	2.1 (0.3 – 13.9)
Prolactin	14 ± 5.9	13 (4.4 – 49.9)

BMI: body mass index, HDL: high-density lipoprotein, LDL: low-density lipoprotein, DHEAS: dehydroepiandrosterone sulfate, FSH: follicle-stimulating hormone, LH: luteinizing hormone, TSH: thyroid-stimulating hormone

The distribution of vitamin D status and DHEAS categories is presented in Table 2. Based on vitamin D classification, severe deficiency was observed in 108 patients (47.0%), while 100 patients (43.5%) had deficiency. Vitamin D insufficiency and sufficiency were identified in 13 (5.9%) and 9 (4.1%) individuals, respectively. Regarding adrenal androgen status, 163 patients (70.9%) had DHEAS levels within age-adjusted normal ranges, whereas 67 patients (29.1%) showed elevated levels.

Table 2. Numbers of PCOS patients according to Vitamin D classification and normal or elevated DHEAS

Classification	*n*	%
Vitamin D level		
Severe deficiency (≤ 10 ng/mL)	108	47.0
Deficiency (11 – 20 ng/mL)	100	43.5
Insufficiency (21 – 30 ng/mL)	13	5.9
Sufficiency (> 30 ng/mL)	9	4.1
DHEAS level		
Normal	163	70.9
Elevated	67	29.1

DHEAS: Dehydroepiandrosterone sulfate

DOI: 10.5455/medscience.2026.03.065

Analyses examining the relationship between vitamin D categories and clinical variables revealed no statistically significant associations with DHEAS levels (p = 0.73), infertility status (p = 0.85), hirsutism severity (p = 0.68), or menstrual interval (p = 0.27). Similarly, when patients were grouped according to normal versus elevated DHEAS levels, no significant differences were observed in infertility (p = 0.49), hirsutism (p = 0.77), BMI (p = 0.20), or menstrual interval (p = 0.09) (Table 3).

Table 3. Association of vitamin D categories with DHEAS status, infertility, hirsutism, and menstrual interval

Clinical characteristics		Vitamin D classification				p
		Severe deficiency	Deficiency	Insufficiency	Sufficiency	
DHEAS, n (%)	**Normal**	70 (75.3)	62 (68.9)	7 (63.6)	5 (71.4)	0.73
	Elevated	23 (24.7)	28 (31.1)	4 (36.4)	2 (28.6)	
Infertility, n (%)	**Positive**	15 (17.2)	16 (21.6)	2 (25)	2 (25)	0.85
	Negative	72 (82.8)	58 (78.4)	6 (75)	6 (75)	
Hirsutism, n (%)	**Normal**	6 (5.8)	6 (6.3)	1 (7.7)	0 (0)	0.68
	Mild	45 (43.7)	39 (40.6)	6 (46.2)	6 (66.7)	
	Moderate	44 (42.7)	47 (49)	4 (30.8)	3 (33.3)	
	Severe	8 (7.8)	4 (4.2)	2 (15.4)	0 (0)	
Menstrual Intervalmedian (min - max)		45 (22 – 240)	47.5 (20 – 270)	60 (20 – 240)	45 (28 – 60)	0.27

DHEAS: Dehydroepiandrosterone sulfate

Further evaluation of DHEAS classification also showed no significant differences between groups in terms of infertility, hirsutism severity, BMI categories, or menstrual characteristics (Table 4). Overall, the distribution of these clinical parameters was comparable between patients with normal and elevated DHEAS levels.

Table 4. Comparing classification of DHEAS levels with infertility, hirsutism, BMI and menstrual interval

Clinical characteristics		Classification of DHEAS		p
		Normal	Elevated	
Infertility, n (%)	**Positive**	22 (17.9)	10 (24.4)	0.49
	Negative	101 (82.1)	31 (75.6)	
Hirsutism, n (%)	**Normal**	8 (5.5)	2 (3.3)	0.77
	Mild	61 (41.8)	22 (36.7)	
	Moderate	68 (46.6)	32 (53.3)	
	Severe	9 (6.2)	4 (6.7)	
BMI, n (%)	**< 25**	43 (75.4)	14 (24.6)	0.20
	25 – 30	41 (63.1)	24 (36.9)	
	≥ 30	57 (75)	19 (25)	
Menstrual Interval median (min – max)		45 (20 – 270)	60 (22 – 240)	0.09

BMI: Body mass index

In contrast, BMI differed significantly across vitamin D categories (one-way ANOVA, p = 0.048) (Table 5). Post hoc analysis (Tukey HSD) demonstrated that patients with vitamin D levels > 20 ng/mL had higher BMI values compared to those with levels between 10–20 ng/mL (p = 0.049).

The results of the univariate logistic regression analyses are shown in Table 6. Vitamin D category was not significantly associated with elevated DHEAS status, infertility, or moderate/severe hirsutism. Likewise, elevated DHEAS status did not significantly increase the odds of infertility or moderate/severe hirsutism. These findings were consistent with the initial group comparisons and suggested that the observed associations were weak and statistically non-significant.

DOI: 10.5455/medscience.2026.03.065

Table 5. Comparison of BMI across vitamin D categories

Variable	Vitamin D level			p
	< 10 Mean ± SD	10 – 20 Mean ± SD	> 20 Mean ± SD	
BMI (kg/m²)	28.36 ± 6.31	27.03 ± 7.16	31.69 ± 8.99	0.04*
One-way ANOVA test				

Table 6. Univariate logistic regression analyses for selected outcomes

Outcome	Predictor	Crude OR	95% CI	p-value
Elevated DHEAS	Vitamin D category	1.21	0.76–1.94	0.423
Infertility	Vitamin D category	1.16	0.70–1.91	0.560
Moderate/severe hirsutism	Vitamin D category	0.98	0.67–1.44	0.920
Infertility	Elevated DHEAS	1.48	0.63–3.46	0.365
Moderate/severe hirsutism	Elevated DHEAS	1.34	0.73–2.47	0.342

Severe vitamin D deficiency was used as the reference category in categorical comparisons. OR: odds ratio; CI: confidence interval; DHEAS: dehydroepiandrosterone sulfate

Discussion

In the present study, we aimed to comprehensively evaluate the interplay between vitamin D status, clinical features, and DHEAS levels in a cohort of women diagnosed with PCOS. Our findings demonstrated that vitamin D deficiency was remarkably frequent within this population, which is in line with previously published data [13,14]. However, despite this high prevalence, no meaningful association was identified between vitamin D levels and adrenal androgen status, nor with key clinical parameters including infertility, hirsutism, and menstrual irregularities. Interestingly, these results do not fully align with certain reports suggesting a more pronounced clinical impact of vitamin D in PCOS. While the majority of studies consistently report a high frequency of vitamin D deficiency in this patient group, the extent to which this deficiency translates into clinically relevant outcomes appears to be inconsistent across different populations [15,16]. From a clinical perspective, this variability may reflect underlying heterogeneity in patient characteristics, environmental exposures, and metabolic profiles. Taken together, our findings support the notion that although vitamin D deficiency is commonly observed in women with PCOS, its direct contribution to clinical manifestations may be limited or context-dependent rather than universally applicable [12]. This interpretation is compatible with the multifactorial nature of PCOS, where complex and overlapping regulatory mechanisms - rather than a single isolated factor - are likely responsible for the observed clinical phenotype.

A substantial body of evidence has indicated that reduced vitamin D levels are frequently encountered among women diagnosed with PCOS. For instance, Bashir et al. demonstrated that vitamin D deficiency was more pronounced in PCOS patients when compared to healthy control populations [17]. However, it remains unclear whether this represents a causal factor, a coincidental finding, or a consequence of PCOS itself. What is more consistently reported is that the gap in vitamin D levels between healthy controls and women with PCOS is not always clearly demonstrated [18]. These inconsistent signs propose that vitamin D status may be influenced more by environmental and population-related factors rather than PCOS itself. Factors such as sunlight exposure, dietary habits, clothing style, seasonal variation, and geographic latitude can significantly affect vitamin D levels and may also influence study outcomes. In addition, variations in study design, sample size, and population characteristics may further contribute to the variability observed across studies. From a clinical perspective, these variations may significantly influence study outcomes; therefore, any observed association between vitamin D deficiency and PCOS should be interpreted within the context of population-specific and environmental factors. In this regard, population-specific characteristics must not be overlooked when interpreting study findings. In our cohort of 230 patients, only 4.1% of the participants had enough vitamin D levels. We also found that nearly 90% of the patients had vitamin D levels under 20 ng/mL. In our study, no significant association was observed among vitamin D levels and clinical features of PCOS, including infertility, hirsutism, and menstrual irregularities. It is well known that vitamin D deficiency of is quite prevalent in our country across different seasons and affects a large proportion of otherwise healthy individuals [19]. Therefore, the high frequency of vitamin D deficiency observed in our study population may not be specific to PCOS. Rather, it may reflect a broader population-based pattern. This perspective may help explain why biochemical findings do not always translate into clinical differences. This situation may partly explain why we did not determine a significant relationship between vitamin D levels and clinical findings in our cohort. In this context, the

DOI: 10.5455/medscience.2026.03.065

high background prevalence of vitamin D deficiency of should be considered when interpretation our results. The association among vitamin D status and increased androgen levels has not yet been clearly established [20]. Although this topic has been widely investigated, the available evidence remains inconsistent and does not allow for a definitive conclusion. Some studies have suggested an inversely correlated between vitamin D levels and androgen markers, indicating that lower vitamin D levels may be correlated with higher androgen concentrations. This has led to the hypothesis that vitamin D might play a modulatory role in androgen metabolism [21]. Taken together, the available literature does not present a consistent pattern, and substantial heterogeneity persists across studies examining this association. In our study, we did not detect any correlation between vitamin D levels and DHEAS concentrations. This finding suggests that adrenal androgen production, at least in our cohort, may not be directly influenced by vitamin D status. It also raises the possibility that other regulatory mechanisms may play a more dominant role in determining androgen levels in women with PCOS [22].

These findings suggest that androgen excess in PCOS is unlikely to be explained by a single mechanism and is more plausibly driven by the interaction of multiple regulatory pathways. Another notable observation in our study was the absence of an inverse association between vitamin D levels and BMI. In contrast to the commonly held assumption that obesity is linked to lower vitamin D levels, our data showed higher BMI values in individuals with relatively higher vitamin D levels (> 20 ng/mL). Although a meta-analysis by Pereira-Santos et al. reported a high prevalence of vitamin D deficiency among obese women with PCOS, other studies have yielded inconsistent or only weak associations between BMI and vitamin D status [23]. One possible description for our findings is that vitamin D deficiency was uniformly high across all BMI categories in our population, thereby masking potential differences between subgroups. In other words, when deficiency is highly prevalent in the entire cohort, the expected inverse relationship may not be clearly detectable. Additionally, variations in lifestyle factors, dietary habits, and sun exposure may have influenced both BMI and vitamin D levels in a similar manner, further reducing the likelihood of observing a clear association. This may partially account for the unexpected direction of the relationship observed in our data. In Türkiye, vitamin D levels may be affected by seasonal variation, sunlight exposure, lifestyle factors, and supplementation practices. Therefore, unrecorded vitamin D supplementation may partly explain the relatively higher vitamin D levels observed in patients with higher BMI, particularly in those receiving closer metabolic follow-up. The main strengths of this study include the use of real-world clinical data from a well-defined PCOS cohort and the simultaneous evaluation of vitamin D status, DHEAS levels, and clinically relevant parameters such as infertility, hirsutism, menstrual interval, and BMI.

Limitations

When interpreting our findings, certain methodological constraints should be taken into account. First, the retrospective design inherently carries a risk of selection bias and precludes definitive conclusions regarding causality. Second, the absence of a healthy control group is an important limitation. Therefore, our findings cannot determine whether vitamin D deficiency is specific to PCOS or reflects a broader population-level pattern. Furthermore, the single-center design may limit the generalizability of the findings. Vitamin D levels may also be affected by season, sunlight exposure, supplementation history, lifestyle, dietary habits, and physical activity, which could not be fully evaluated in this retrospective dataset. In addition, detailed insulin resistance indices such as HOMA-IR were not included. Therefore, residual confounding related to BMI, insulin resistance, age, androgen profile, LH/FSH ratio, and environmental factors cannot be excluded.

Conclusion

Overall, vitamin D deficiency seems to be quite common in women with PCOS. In our cohort, however, we did not observe any clear relationship between vitamin D levels and clinical findings or DHEAS concentrations. These findings suggest that, in our cohort, vitamin D status was not clearly associated with DHEAS levels or selected clinical findings. However, this result should be interpreted cautiously because of the retrospective design, absence of a healthy control group, and potential residual confounding. Further prospective studies with well-defined populations are required to better understand the clinical relevance of vitamin D in women with PCOS.

Ethical Approval

This study was conducted Clinical Research Ethics Committee approval no: 2026-37; meeting no: 2026-03/03-22; date: 25 February 2026. The committee reviewed the study protocol, including its rationale, objectives, and methodology, and concluded that there were no ethical or scientific concerns regarding the conduct of the study at the specified centers.

Patient Consent

As this study was based on a retrospective review of archived data, informed consent was not required.

Conflict of Interest

The authors confirm the absence of any conflicts associated with this work.

Funding

The authors declare that they have received no financial support for the study.

References

1. Escobar-Morreale HF. Polycystic ovary syndrome: definition, aetiology, diagnosis and treatment. Nat Rev Endocrinol. 2018;14:270-84.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod. 2004;19:41-7.
3. Balen AH, Morley LC, Misso M, et al. The management of anovulatory infertility in women with polycystic ovary syndrome: an analysis of the evidence to support the development of global WHO guidance. Hum Reprod Update. 2016;22:687-708.

DOI: 10.5455/medscience.2026.03.065

4. Deswal R, Narwal V, Dang A, Pundir CS. The prevalence of polycystic ovary syndrome: a brief systematic review. J Hum Reprod Sci. 2020;13:261-71.
5. Teede HJ, Tay CT, Laven JJE, et al. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. J Clin Endocrinol Metab. 2023;108:2447-69.
6. Lei X, Zhou Q, Wang Y, et al. Serum and supplemental vitamin D levels and insulin resistance in T2DM populations: a meta-analysis and systematic review. Sci Rep. 2023;13:12343.
7. Zhao H, Zhang J, Cheng X, et al. Insulin resistance in polycystic ovary syndrome across various tissues: an updated review of pathogenesis, evaluation, and treatment. J Ovarian Res. 2023;16:9.
8. Muscogiuri G, Altieri B, Annweiler C, et al. Vitamin D and chronic diseases: the current state of the art. Arch Toxicol. 2017;91:97-107.
9. Yu L, Hu B, Kong J, et al. Effect of vitamin D deficiency on clinical pregnancy outcomes in women with polycystic ovary syndrome undergoing in vitro fertilization. Front Endocrinol (Lausanne). 2025;16:1710004.
10. Guo L, Zheng X, Gu C, et al. Associations between serum 25-hydroxyvitamin D concentrations and assisted reproductive technology outcomes in women with polycystic ovary syndrome: a cohort study. Front Endocrinol (Lausanne). 2025;16:1639137.
11. Hundemer GL, Leung AA, Kline GA, et al. Biomarkers to guide medical therapy in primary aldosteronism. Endocr Rev. 2024;45:69-94.
12. Cui A, Zhang T, Xiao P, et al. Global and regional prevalence of vitamin D deficiency in population-based studies from 2000 to 2022: a pooled analysis of 7.9 million participants. Front Nutr. 2023;10:1070808.
13. Kim JJ, Choi YM, Chae SJ, et al. Vitamin D deficiency in women with polycystic ovary syndrome. Clin Exp Reprod Med. 2014;41:80-5.
14. Wehr E, Pilz S, Schweighofer N, et al. Association of hypovitaminosis D with metabolic disturbances in polycystic ovary syndrome. Eur J Endocrinol. 2009;161:575-82.
15. He C, Lin Z, Robb SW, Ezeamama AE. Serum vitamin D levels and polycystic ovary syndrome: a systematic review and meta-analysis. Nutrients. 2015;7:4555-77.
16. Mohan A, Haider R, Fakhor H, et al. Vitamin D and polycystic ovary syndrome (PCOS): a review. Ann Med Surg (Lond). 2023;85:3506-11.
17. Bashir M, Mukhtar S, Ikram M, Javed A. Vitamin D deficiency and PCOS; association between vitamin D deficiency and PCOS in females presenting in a tertiary care hospital. Professional Med J. 2019;26:40-4.
18. Ng BK, Lee CL, Lim PS, et al. Comparison of 25-hydroxyvitamin D and metabolic parameters between women with and without polycystic ovarian syndrome. Horm Mol Biol Clin Investig. 2017;31:20160057.
19. Cinar N, Harmanci A, Yildiz BO, Bayraktar M. Vitamin D status and seasonal changes in plasma concentrations of 25-hydroxyvitamin D in office workers in Ankara, Turkey. Eur J Intern Med. 2014;25:197-201.
20. Kazeminia M, Rajati F, Rasulehvandi R, Rajati M. The effect of vitamin D on the hormonal profile of women with polycystic ovarian syndrome: a systematic review and meta-analysis. Middle East Fertil Soc J. 2024;29:45.
21. Lerchbaum E, Obermayer-Pietsch B. Mechanisms in endocrinology: vitamin D and fertility: a systematic review. Eur J Endocrinol. 2012;166:765-78.
22. Goodarzi MO, Carmina E, Azziz R. DHEA, DHEAS and PCOS. J Steroid Biochem Mol Biol. 2015;145:213-25.
23. Pereira-Santos M, Costa PRF, Assis AMO, et al. Obesity and vitamin D deficiency: a systematic review and meta-analysis. Obes Rev. 2015;16:341-9.