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Original Research Article

Vitamin D deficiency in women with polycystic ovarian syndrome- a cross-sectional observational study in Indian subcontinent

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ABSTRACT

Background: Polycystic Ovarian Syndrome (PCOS) affects up to 18% of reproductive-age women, presenting with menstrual dysfunction, hirsutism, and metabolic complications. Vitamin D deficiency (VDD) shares features such as insulin resistance and obesity. Although global studies link hypovitaminosis D to metabolic risk, evidence in Indian women remains limited and inconsistent. This study assessed the prevalence of VDD and its association with metabolic and endocrine parameters in Indian women with PCOS.

Methods: A cross-sectional study was conducted at Army Hospital Research and Referral, New Delhi, over 18 months (February 2022-February 2024). A total of 170 women (18-40 years) diagnosed with PCOS by Rotterdam criteria were enrolled. Participants were stratified by serum 25-hydroxyvitamin D (25(OH)D) levels: Group I (<30ng/ml) and Group II (≥30 ng/ml). Evaluations included anthropometry (BMI, waist circumference), hormonal profiles (LH, FSH, AMH, TSH), and metabolic markers (fasting glucose, insulin, HOMA-IR, HbA1c, cholesterol).

Results: Most participants (89.4%, n=152) had suboptimal Vitamin D levels, while only 10.6% (n=18) were sufficient. Comparative analysis showed no significant differences (p>0.05) between groups in age, menstrual irregularities, Ferriman-Gallway scores, ovarian morphology, or biochemical markers (LH, FSH, AMH, insulin, HOMA-IR, BMI). Spearman correlation confirmed no significant linear association between 25(OH)D levels and metabolic or hormonal variables.

Conclusions: Hypovitaminosis D is highly prevalent among Indian women with PCOS. However, Vitamin D status did not significantly influence metabolic or endocrine dysfunction in this cohort, suggesting it may represent a comorbid condition rather than a causal factor. Larger longitudinal studies are warranted to clarify causal pathways and therapeutic relevance.

Keywords: Polycystic ovarian syndrome, Vitamin D deficiency, Insulin resistance, Metabolic syndrome, Hyperandrogenism, HOMA-IR, Women health

INTRODUCTION

Polycystic Ovarian Syndrome (PCOS) is one of the most pervasive endocrine disorders, affecting 4-18% of women of the reproductive age worldwide.¹ It is a multi-systemic chronic condition presenting with the menstrual dysfunction, infertility, hirsutism, acne, and the obesity.^{2,3}

Beyond reproductive manifestations, PCOS carries a significant metabolic burden: nearly one-third of affected women develop metabolic syndrome, characterized by central obesity, hypertension, and insulin resistance, which predispose to type 2 diabetes, cardiovascular disease, and certain malignancies.⁴ An ideal management strategy would identify modifiable biochemical drivers to mitigate

these systemic risks. Vitamin D, a fat-soluble prohormone, has emerged as a candidate. Vitamin D receptors (VDR) are expressed in the ovaries and pancreatic beta-cells, and its active metabolite, 1,25-dihydroxyvitamin D₃, is hypothesized to influence reproductive physiology and glucose homeostasis.^{5,6} The Calcium-Mediated Insulin Release Theory further suggests Vitamin D regulates calcium levels essential for insulin-mediated glucose transport.⁷

However, the clinical reality falls far short of this ideal. Despite the high prevalence of both Vitamin D deficiency (VDD) and PCOS, our understanding of their synergistic impact remains fragmented and often contradictory. VDD intersects critically with PCOS. Vitamin D, beyond its classical role in calcium and bone metabolism, influences insulin sensitivity, ovarian follicular development, and cardiovascular regulation.⁸ In theory, adequate vitamin D levels should mitigate metabolic and reproductive dysfunctions, yet evidence consistently shows that women with PCOS have disproportionately high rates of deficiency compared to the general population.^{9,10} This raises questions about whether VDD is a driver, consequence, or cofactor in the pathophysiology of PCOS.

Evidence is conflicting Wehr et al (2009) and Li et al (2011) reported negative correlations between Vitamin D levels and insulin resistance markers such as HOMA-IR and BMI.^{11,12} Conversely, Kim et al (2014) and Mogili et al (2018) found no metabolic associations, suggesting Vitamin D may be a comorbid feature rather than a primary driver.^{10,13} These disparities often stem from variations in ethnic backgrounds, dietary habits, and the distinct phenotypic presentations of PCOS, leaving clinicians without a definitive consensus on the metabolic utility of Vitamin D. The consequences of unresolved evidence are significant. Persistent hypovitaminosis D may exacerbate hyperandrogenism and insulin resistance, perpetuating weight gain and ovulatory failure.¹⁴ Lack of clarity hinders guideline development, risking undertreatment of a metabolic risk factor or unnecessary medicalisation of a common deficiency.

In South Asia, the issue is acute: approximately 80% of adults in India, Pakistan, and Bangladesh are Vitamin D deficient, yet data on its interplay with PCOS remains scarce.¹⁵ Despite the growing body of literature, gaps remain—particularly in understanding vitamin D status among Indian women with PCOS, where deficiency is widespread due to lifestyle and pigmentation factors. Our study addresses this gap by examining Indian women with PCOS at a tertiary care centre in New Delhi. While meta-analyses such as Chunla He et al (2015) suggest VDD may be a “minor pathway”.¹⁶

our research explores its role within a population with high baseline prevalence of both insulin resistance and VDD. Using Rotterdam Criteria for diagnosis and correlating absolute 25(OH)D levels with biochemical markers including LH, FSH, AMH, and HOMA-IR, we aim to test

the metabolic hypothesis in a real-world clinical setting.¹⁷ By critically evaluating Vitamin D status as a marker of metabolic severity in Indian women, this study builds upon prior work while addressing geographical and phenotypic gaps. Ultimately, it seeks to refine PCOS pathogenesis models and determine whether correcting Vitamin D deficiency should be a cornerstone of modern PCOS management.

Objectives of the study

The primary aim of this research is to determine the prevalence of vitamin D deficiency among women diagnosed with PCOS within an Indian clinical setting. Beyond simply establishing a baseline for deficiency, the study seeks to logically test the association between hypovitaminosis D and the core components of metabolic syndrome, specifically insulin resistance, Type 2 diabetes mellitus, dyslipidaemia, and obesity. By stratifying a cohort of 170 women into groups with sufficient and suboptimal vitamin D levels, the research aims to uncover whether low 25(OH)D concentrations act as a primary driver for the hormonal and metabolic dysregulation. Academically, this study contributes to an ongoing debate. Wehr et al (2009) reported strong negative correlations between vitamin D and insulin resistance, whereas Kim et al (2014) found no such link, leaving the role of the VDR in PCOS pathogenesis uncertain.¹¹⁻¹³ Practically, the stakes are considerable.

PCOS affects up to 18% of reproductive-age women and imposes significant health and financial burdens due to infertility and cardiovascular complications (Fauser et al, 2012).¹ With nearly 80% of Indian adults deficient in vitamin D, clarifying whether this deficiency exacerbates PCOS could inform cost-effective interventions such as supplementation. Using the CARS (Create a Research Space) framework, the study first establishes the territory: PCOS is a multifaceted endocrine disorder manifesting as menstrual dysfunction, hirsutism, and metabolic syndrome (Bani Mohammad and Majdi Seghinsara, 2017) it then identifies the niche: contradictory findings persist regarding vitamin D's metabolic role in Indian women, a population prone to both VDD and insulin resistance.^{10,11,13,18} Finally, the study occupies the niche by investigating vitamin D status and its metabolic associations in Indian women with PCOS, aiming to provide evidence that advances academic discourse and guides clinical practice.

METHODS

The present study employed a cross-sectional observational design to evaluate the prevalence of VDD and its correlations with metabolic and endocrine markers in Indian women diagnosed with PCOS. A cross-sectional approach was chosen to provide a snapshot of vitamin D status and explore associations with insulin resistance, obesity, and dyslipidemia, rather than establish causality. This design aligns with epidemiological guidelines where

prevalence and associations are best assessed through observational frameworks. Conducted at the Department of Obstetrics and Gynaecology, Army Hospital, Research and Referral, Delhi Cantt, New Delhi, the study spanned 18 months (February 2022-February 2024), generating data on hypovitaminosis D in Indian women with PCOS, a group underrepresented in global studies. Prior to data collection, the protocol underwent review and approval by the Institutional Ethics Committee (IEC). Ethical standards were strictly followed: participants received detailed explanations of study aims and procedures in English or Hindi, and written informed consent was obtained. Participation was voluntary, with the right to withdraw preserved. Confidentiality was maintained by restricting data use to research purposes and secure storage. The study population comprised 170 Asian women aged 18-40 years attending the outpatient department. Non-probability sampling was used to recruit patients meeting diagnostic requirements for PCOS.

Diagnosis followed the Rotterdam Criteria (2004, ESHRE/ASRM), requiring at least two of three features: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology.¹⁷ Morphology was assessed via high-resolution pelvic ultrasonography (Philips Affinity 70), with ≥ 12 follicles (2–9 mm) in either ovary considered diagnostic. Stringent exclusion criteria ensured validity by eliminating confounding conditions. Women with endocrine or reproductive disorders mimicking PCOS (endometriosis, congenital adrenal hyperplasia, hyperprolactinemia, thyroid disease, Cushing syndrome) were excluded, as were those with pre-existing diabetes, recent hormonal therapy, smoking, drug abuse, or psychiatric illness. These measures isolated the effects of PCOS and vitamin D status on metabolic parameters.

Clinical and anthropometric evaluations were performed at baseline. Weight, height, and waist circumference (WC) were measured by trained personnel, with WC taken at the iliac processes and umbilicus. Body Mass Index (BMI) was calculated to assess adiposity. Biochemical assessments were conducted on fasting blood samples collected at 08:00 hours.

Analyses included fasting glucose, fasting insulin, Luteinizing Hormone (LH), Follicle-Stimulating Hormone (FSH), Prolactin (PRL), Anti-Müllerian Hormone (AMH), and Thyroid Stimulating Hormone (TSH), using automated chemiluminescence immunoassay (ICMA) or euglobulin clot lysis assay (ECLA) kits. Insulin resistance was quantified using the Homeostasis Model Assessment (HOMA-IR). The central variable, serum 25-hydroxyvitamin D (25(OH)D₃), was measured to determine vitamin D status. Following Endocrine Society criteria, participants were stratified into two groups: Group I with suboptimal levels (<30ng/ml, deficiency/insufficiency) and Group II with sufficient levels (≥ 30 ng/ml). This threshold delineated those requiring clinical attention from those with adequate

status. Statistical analysis was performed using SPSS version 28.0. Continuous variables were expressed as mean \pm standard deviation (SD) or medians with interquartile ranges, while categorical variables were reported as absolute numbers and percentages. To determine the significance of differences between the two vitamin D groups, the study employed Student's t-tests for normally distributed data and the Mann-Whitney U test for non-normally distributed variables. Categorical data were compared using Chi-squared or Fisher's exact tests. Finally, the relationship between absolute 25(OH)D levels and other clinical or biochemical parameters was explored using Spearman correlation analysis, with a p-value of <0.05 considered the threshold for statistical significance.

RESULTS

Vitamin D prevalence and participant demographics

This cross-sectional study evaluated 170 Asian women (18-40 years) with PCOS, stratified by serum 25-hydroxyvitamin D (25(OH)D) levels: Group I (<30ng/ml) and Group II (≥ 30 ng/ml). Suboptimal vitamin D was highly prevalent, affecting 89.4% (n=152), while sufficiency was observed in only 10.6% (n=18). Mean ages were 30.13 \pm 4.13 (Group I) and 30.44 \pm 3.87 (Group II), with no significant difference between groups (p=0.760).

Clinical and ultrasonographic observations

Clinical features, including menstrual irregularities and hirsutism, were compared across both vitamin D cohorts. In Group I (<30ng/ml), 61.8% had oligomenorrhea and 38.2% amenorrhea, while Group II (≥ 30 ng/ml) showed 66.7% and 33.3%, respectively (p=0.690). Hyperandrogenism via Ferriman-Gallwey scores averaged 9.95 \pm 3.74 (Group I) and 9.50 \pm 4.06 (Group II) (p=0.737). Polycystic ovarian morphology was observed in 74.3% of Group I versus 66.7% of Group II (p=0.485).

Endocrine and metabolic profiles

The relationship between vitamin D status and various biochemical markers was evaluated, as summarised in the professional table below. No statistically significant differences (p < 0.05) were observed between Group I and Group II across all tested endocrine and metabolic parameters.

Correlation analysis

Spearman correlation analysis was performed to identify potential linear relationships between absolute 25(OH)D levels and metabolic or hormonal variables. No significant correlations were identified. For instance, vitamin D levels did not significantly correlate with LH (rho=-0.102, p=0.184), HOMA-IR (rho=0.047, p=0.542), or BMI (rho =0.02, p=0.796).

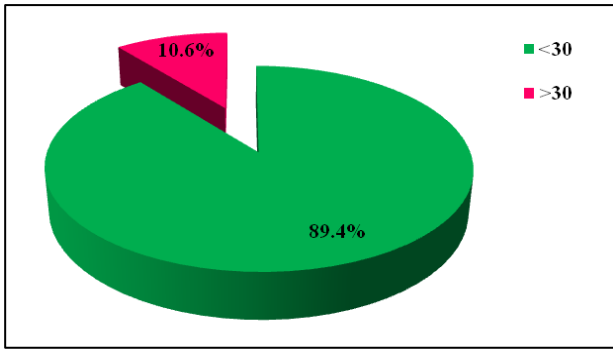


Figure 1: Distribution of patients into two groups on the basis of 25 (OH) D level i.e., <30 ng/ml and >30 ng/ml. It was observed that 89.4% PCOS patients had 25 (OH) D level <30 ng/ml and 10.6% had 25 (OH) D level >30 ng/ml.

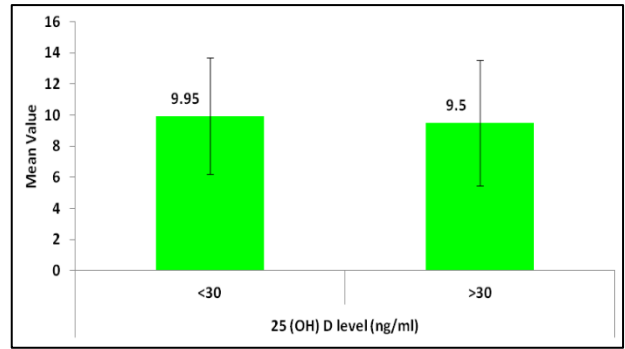


Figure 3: Shows mean ferriman-gallwey scores: 9.95 ± 3.74 (<30ng/ml) vs. 9.50 ± 4.06 (≥ 30 ng/ml), with no significant difference ($p=0.737$).

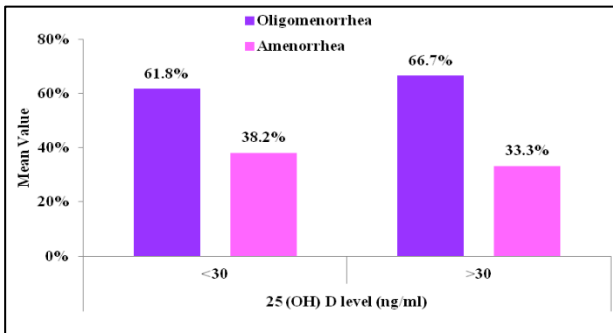


Figure 2: Shows oligomenorrhea (61.8% vs. 66.7%) and amenorrhea (38.2% vs. 33.3%) across vitamin D groups, with no significant difference ($p=0.790$).

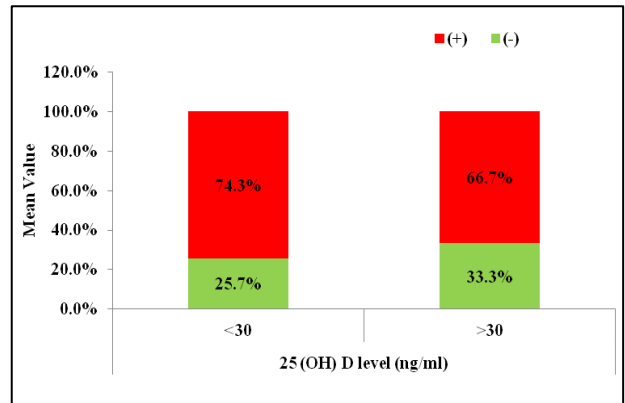


Figure 4: Shows polycystic ovaries in 74.3% (<30ng/ml) vs. 66.7% (≥ 30 ng/ml), with no significant distribution difference between groups ($p=0.485$).

Table 1: Distribution of 25(OH)D Status in the Study Population (n=170).

Vitamin D status 25(OH)D	Serum level (ng/ml)	Frequency (N)	Percentage (%)
Deficient/insufficient	<30	152	89.4
Sufficient	>30	18	10.6
Total		170	100

Table 2: Comparison of Clinical and Ultrasound Findings by Vitamin D Status.

Parameter	Group I (<30 ng/ml)	Group II (>30 ng/ml)	P value
Menstrual pattern			0.690
- oligomenorrhea n (%)	94 (61.8%)	12 (66.7%)	
- amenorrhea n (%)	58 (38.2%)	6 (33.3%)	
Hirsutism (FG score)	9.95 ± 3.74	9.50 ± 4.06	0.737
Polycystic ovaries (+)	113 (74.3%)	12 (66.7%)	0.485

Table 3: Comparative Analysis of Endocrine and Metabolic Markers (Mean \pm SD).

Parameter	Group I (<30 ng/ml)	Group II (>30 ng/ml)	P value
Luteinizing hormone (miu/ml)	7.13 ± 3.01	6.82 ± 2.96	0.522
Follicle-stimulating hormone (miu/ml)	7.53 ± 2.23	7.37 ± 3.30	0.415
Anti-müllerian hormone (ng/ml)	4.39 ± 1.54	4.86 ± 2.10	0.409
Thyroid stimulating hormone (TSH)	4.13 ± 1.93	3.38 ± 1.44	0.092
Fasting blood sugar (mg/dl)	86.15 ± 8.61	86.44 ± 9.44	0.632

Continued.

Parameter	Group I (<30 ng/ml)	Group II (>30 ng/ml)	P value
Fasting insulin (mu/ml)	9.82±2.88	10.73±3.22	0.290
Homa-IR	2.09±0.63	2.30±0.87	0.409
HbA1c (%)	5.49±0.80	5.76±0.73	0.136
Body mass index (BMI, kg/m ²)	23.00±3.21	23.95±4.09	0.558
Total cholesterol (mg/dl)	169.48±26.77	173.33±39.00	0.277

Table 4: Spearman correlation coefficients (RHO) for vitamin D levels.

Variable	RHO	P value
LH (miu/ml)	-0.102	0.184
FSH (miu/ml)	0.004	0.957
AMH	0.026	0.735
Fasting insulin	0.035	0.653
Homa-IR	0.047	0.542
Total cholesterol	0.092	0.233

DISCUSSION

The current study highlights a striking prevalence of suboptimal vitamin D levels among Indian women with PCOS, with 89.4% of participants exhibiting serum 25(OH)D levels below 30 ng/ml. This finding is consistent with broader epidemiological data identifying hypovitaminosis D as a global public health crisis, particularly in South Asia where deficiency rates often exceed 80%. In the specific context of PCOS, our results parallel those of Neelaveni et al, who reported an 85.2% prevalence of deficiency, and Wehr et al, who observed insufficiency in 72.8% of their cohort.¹¹⁻¹⁹ Such high rates suggest that vitamin D status may represent either a comorbid feature of PCOS or a significant environmental contributor to its manifestation. Despite this profound prevalence, our analysis revealed no statistically significant differences in clinical, metabolic, or endocrine parameters between women with serum 25(OH)D levels above and below 30 ng/ml. This lack of association extended to primary markers of insulin resistance, including HOMA-IR ($p=0.409$) and fasting insulin ($p=0.290$), as well as clinical markers such as the Ferriman-Gallwey score ($p=0.737$) and polycystic ovarian morphology ($p=0.485$). These findings contrast with several landmark studies. Wang et al and Wehr et al reported significant negative correlations between vitamin D levels and HOMA-IR, BMI, and cholesterol.¹¹⁻²⁰ Similarly, Gokosmanoglu et al found higher LH and testosterone levels in vitamin D-deficient patients, whereas our cohort showed comparable LH levels across groups ($p=0.522$).²¹

The disparity between our findings and prior literature may be explained by the anthropometric profile of our participants. The mean BMI (23.0-23.9 kg/m²) was relatively lean compared to obese cohorts in other studies. Obesity is a well-recognized risk factor for vitamin D deficiency, likely due to sequestration of this fat-soluble vitamin in adipose tissue. In leaner PCOS populations, the metabolic impact of deficiency may be attenuated, suggesting that the “Vitamin D–Insulin” axis could be

BMI-dependent. Our results align with Kim et al (2014), who also found no significant associations in a Korean cohort, indicating that vitamin D’s role in PCOS pathogenesis may vary across ethnic and phenotypic subgroups.¹³

This study contributes novelty by focusing on Indian women with PCOS, a population underrepresented in global research. While Western studies have linked vitamin D deficiency to metabolic risk, few have examined South Asian contexts. Our findings suggest that the burden of hypovitaminosis D in Indian PCOS women is even greater than reported elsewhere, with prevalence exceeding 70%. This underscores the interplay of cultural, environmental, and biological factors in shaping disease risk.

From a theoretical perspective, our results challenge the clinical application of the Calcium-Mediated Insulin Release Theory, which posits that vitamin D regulates calcium levels essential for insulin-mediated glucose transport and pancreatic β -cell secretion.⁷ The presence of VDR in the ovaries and the insulin gene’s Vitamin D Response Element (VDRE) suggests genomic roles in reproduction and glucose homeostasis. Yet, our data imply that these mechanisms may be overshadowed by other dominant factors in PCOS, such as genetic predisposition or hormonal imbalances. Chunla He et al’s meta-analysis supports this, suggesting vitamin D deficiency may represent a minor pathway or comorbid symptom rather than a primary driver of hormonal dysregulation in PCOS.¹⁶ The Endocrine Crosstalk Model further proposes that vitamin D influences ovarian steroidogenesis and androgen production. Supplementation trials with vitamin D, such as Pal et al (2012), have shown reductions in androgen levels and improved reproductive outcomes.²² While our study did not directly assess reproductive endpoints, correlations with metabolic markers indirectly support this model’s plausibility.

The limitations of this study must be acknowledged as they may have influenced the findings. First, the cross-sectional

design precludes causal inference; we cannot determine whether PCOS leads to lower vitamin D or vice versa. Second, the sufficient vitamin D group (>30 ng/ml) was small (n=18, 10.6%), reducing statistical power to detect subtle differences. Third, confounding variables such as seasonal variation, dietary intake, and sunlight exposure were not controlled, though they are critical determinants of vitamin D synthesis. Reliance on single-point serum 25(OH)D measurements introduce variability, as levels fluctuate with lifestyle and season. Finally, excluding women on supplementation or hormonal therapy, while methodologically necessary, may have biased the sample toward more severe cases.

In conclusion, vitamin D deficiency is nearly ubiquitous among Indian women with PCOS but does not appear to be a primary determinant of metabolic or endocrine severity in this relatively lean cohort. These findings support a conservative view of vitamin D as a general health marker rather than a central therapeutic target in PCOS. Future research should prioritize large-scale, longitudinal studies stratified by BMI and phenotype to identify subgroups that may benefit from supplementation. Randomised controlled trials assessing vitamin D replacement on long-term outcomes—such as live birth rates and type 2 diabetes prevention—are essential to move beyond cross-sectional associations and establish clinical guidelines, particularly in South Asia. Investigations into gene–nutrient interactions, including VDR polymorphisms, may clarify susceptibility to deficiency and its metabolic consequences. Finally, multi-centre collaborations are needed to disentangle contextual factors such as diet, sunlight exposure, and cultural practices that modulate the relationship between vitamin D and PCOS.

CONCLUSION

This cross-sectional observational study investigated the prevalence of VDD among Indian women with PCOS and explored associations between serum 25(OH)D levels and metabolic or endocrine risk factors. Findings revealed a strikingly high prevalence of suboptimal vitamin D status, with 89.4% of participants exhibiting levels below 30 ng/ml. Despite this widespread deficiency, no statistically significant differences were observed between vitamin D-deficient and sufficient groups across clinical, hormonal, or metabolic parameters, including HOMA-IR, fasting insulin, BMI, and LH levels. Correlation analyses similarly failed to identify linear relationships between absolute vitamin D concentrations and PCOS severity.

These results carry important theoretical implications. While prior literature emphasizes the “Calcium-Mediated Insulin Release Theory” and the role of VDR in glucose metabolism and ovarian function, our findings suggest that these pathways may exert limited clinical impact in certain populations. VDD may represent a comorbid feature of PCOS rather than a primary driver of hormonal or metabolic dysregulation. This highlights the need to

account for ethnic and phenotypic diversity, as vitamin D’s role may be modulated by BMI or genetic predisposition. Future research should move beyond cross-sectional associations toward large-scale, longitudinal studies and multi-centre randomized controlled trials (RCTs) to establish causality and therapeutic thresholds. Investigations should also assess whether supplementation improves long-term outcomes such as live birth rates or diabetes prevention, even in the absence of immediate metabolic changes.

Limitations include the cross-sectional design, which restricts causal inference, and the small vitamin D-sufficient group (n=18), reducing statistical power. Larger, balanced cohorts with control for confounders such as seasonal sunlight exposure and diet are needed. Ultimately, this study underscores a disconnect between high deficiency rates and clinical severity in South Asian women with PCOS, challenging the universality of vitamin D’s metabolic influence and supporting a more nuanced, personalized approach to PCOS management.

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