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Association of Serum Vitamin D [25(OH)D] with Insulin Resistance Assessed by HOMA-IR in Newly Diagnosed Women with Polycystic Ovary Syndrome [PCOS / Polyendocrine Metabolic Ovarian Syndrome (PMOS)]: A Hospital-Based Cross-Sectional Study from Central India

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is one of the most prevalent endocrine disorders in reproductive-age women and carries a high burden of insulin resistance. Vitamin D deficiency, highly prevalent in Indian women, impairs pancreatic β-cell function and peripheral insulin receptor sensitivity, potentially worsening insulin resistance. Population-specific data from Central India examining this association in newly diagnosed PCOS women are scarce.
Objectives: To assess serum vitamin D [25(OH)D] levels in newly diagnosed PCOS women, determine the prevalence of vitamin D deficiency, and establish its association and correlation with insulin resistance as assessed by HOMA-IR.
Methods: A hospital-based, observational, cross-sectional study was conducted at Hamidia Hospital, Gandhi Medical College, Bhopal over 18 months. Sixty-six newly diagnosed PCOS women (Rotterdam criteria) were enrolled. Serum 25(OH)D was measured by competitive chemiluminescence immunoassay. HOMA-IR was calculated as [Fasting Insulin (μIU/mL) × Fasting Glucose (mg/dL)] / 405; values >2.5 indicated insulin resistance. Chi-square test, Student's t-test, and Pearson correlation were used.
Results: Insulin resistance was present in 71.2% of participants. Vitamin D deficiency (<20 ng/mL) was detected in 80.3%; combined suboptimal vitamin D status was present in 93.9%. Vitamin D deficiency was significantly more prevalent in insulin-resistant women (87.2% vs 63.2%; p=0.047). Mean serum vitamin D was significantly lower in insulin-resistant women (15.06 ± 7.25 ng/mL) than non-insulin-resistant women (19.54 ± 10.15 ng/mL; p=0.017). HOMA-IR correlated negatively with serum vitamin D (r = −0.248, p=0.045).
Conclusion: Vitamin D deficiency is near-universal in newly diagnosed PCOS women and is significantly associated with insulin resistance. Routine vitamin D screening and supplementation are warranted for early metabolic risk evaluation in PCOS.

Keywords: *Polycystic Ovary Syndrome; PCOS; PMOS; insulin resistance; HOMA-IR; vitamin D; 25-hydroxyvitamin D; India.*

INTRODUCTION

Polycystic Ovary Syndrome (PCOS)—also referred to in emerging literature as Polyendocrine Metabolic Ovarian Syndrome (PMOS), a term that better captures its multisystem endocrine and metabolic dimensions—is the most prevalent endocrine disorder in women of reproductive age, affecting 5–10% of women globally and an estimated 11–18% in India (Bharali et al., 2022; Gupta et al., 2017). Its hallmark triad—hyperandrogenism, ovulatory dysfunction, and polycystic

ovarian morphology, as defined by the Rotterdam criteria—extends well beyond the reproductive sphere to encompass significant metabolic comorbidities, most critically insulin resistance, dyslipidaemia, and substantially elevated lifetime risks of type 2 diabetes mellitus and cardiovascular disease (Azziz et al., 2009; Diamanti-Kandarakis & Dunaif, 2012).

Insulin resistance—the diminished ability of insulin to exert its biological effects on skeletal muscle, liver, and adipose tissue—affects 50–90% of women with PCOS irrespective of body mass index and serves as the central mechanistic link between PCOS and its long-term metabolic sequelae (Diamanti-Kandarakis & Dunaif, 2012). The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) is the most widely used and clinically practical surrogate marker, calculated from fasting glucose and insulin values (Abdesselam et al., 2021).

Vitamin D, a fat-soluble prohormone, exerts pleiotropic effects through its receptor (VDR), which is expressed in pancreatic β-cells, skeletal muscle, and adipose tissue, placing it in a direct regulatory role over insulin synthesis, secretion, and peripheral sensitivity (Grundmann & von Versen-Höynck, 2011). Vitamin D deficiency impairs glucose-stimulated insulin secretion, reduces insulin receptor expression, and upregulates pro-inflammatory cytokines TNF-α and IL-6—all of which independently worsen insulin resistance (Argano et al., 2023).

Vitamin D deficiency is disproportionately prevalent among Indian women due to high skin melanin content, prolonged indoor lifestyles, dietary insufficiency, and widespread use of sun-protective clothing (Holick, 2007). In women with PCOS, this baseline deficiency is further compounded by hyperandrogenism, chronic inflammation, and adiposity, which impair vitamin D activation (Morgante et al., 2022). Studies from Turkey, Egypt, and India have reported vitamin D deficiency in 86–90% of PCOS populations, with consistent inverse correlations between serum 25(OH)D and insulin resistance markers (Gokosmanoglu et al., 2020; Omran et al., 2020).

Despite a growing international literature, population-specific data from Central India examining the 25(OH)D–insulin resistance association in newly diagnosed PCOS women remain limited. The present study was therefore conducted to: (i) assess the prevalence and severity of vitamin D deficiency; (ii) compare vitamin D levels between insulin-resistant and non-insulin-resistant PCOS women; and (iii) determine the correlation between serum 25(OH)D and HOMA-IR in this understudied population.

MATERIALS AND METHODS

2.1 Study Design and Setting

An observational, hospital-based, cross-sectional study was conducted in the Department of Biochemistry, Gandhi Medical College (GMC), Bhopal, in collaboration with the Department of Obstetrics and Gynaecology, Hamidia Hospital, Bhopal, over 18 months. All biochemical analyses were performed at the Central Clinical Laboratory, Hamidia Hospital, Bhopal.

2.2 Ethical Approval

The study was approved by the Institutional Ethics Committee, Gandhi Medical College, Bhopal and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each participant in their preferred language prior to enrolment.

2.3 Participants

Newly diagnosed PCOS women attending the Gynaecology OPD, Hamidia Hospital, were consecutively recruited. PCOS was diagnosed using the Rotterdam criteria, requiring at least two of three features after exclusion of secondary causes: (1) oligo/anovulation; (2) clinical or biochemical hyperandrogenism; and (3) polycystic ovarian morphology (≥12 follicles 2–9 mm diameter and/or ovarian volume >10 mL in at least one ovary) (Rotterdam consensus, 2004). Exclusion criteria included: known diabetes mellitus, impaired renal function, ischaemic heart disease, cerebrovascular disease, hypertension, pregnancy or lactation, secondary androgen excess disorders, and current use of hormonal medications or vitamin D supplements.

2.4 Sample Size

Sample size was calculated using $n = z^2 \cdot p \cdot (1-p)/d^2$, with $z=1.96$ (95% confidence), $p=0.11$ (Indian PCOS prevalence) (Bharali et al., 2022), and $d=0.11$ (precision), yielding a minimum of 66 participants.

2.5 Biochemical Methods

Serum 25(OH)D was measured by competitive chemiluminescence immunoassay (CLIA) on the Beckman Coulter DXI 800 analyser. Serum 25(OH)D competes with alkaline-phosphatase-labelled 25(OH)D for binding to anti-25(OH)D antibodies on paramagnetic particles; the chemiluminescent signal is inversely proportional to 25(OH)D concentration (Burtis et al., 2012). Vitamin D status was classified as: deficient <20 ng/mL, insufficient 20–30 ng/mL, sufficient >30 ng/mL.

Fasting serum insulin was measured by two-step sandwich CLIA on the Beckman Coulter DXI 800 (reference range: 2.6–24.9 µIU/mL). Fasting serum glucose was estimated by the hexokinase method on a Biosystems BA 200 analyser (reference

range: 70–100 mg/dL). Blood samples (5 mL) were drawn after an overnight fast of ≥8 hours under strict aseptic conditions, centrifuged at 3,000 rpm for 15 minutes, and processed the same day. Internal quality controls were run daily per NCCLS guidelines (Burtis et al., 2012).

HOMA-IR = [Fasting Insulin (μIU/mL) × Fasting Glucose (mg/dL)] / 405. Values >2.5 indicated insulin resistance, consistent with Indian population references (Abdesselam et al., 2021).

2.6 Statistical Analysis

Data were analysed using Epi-info software. Continuous variables are expressed as mean ± standard deviation (SD); categorical variables as frequencies and percentages. Comparison of mean serum vitamin D between groups used Student's independent t-test; distribution of vitamin D categories used chi-square test. Bivariate association between serum 25(OH)D and HOMA-IR used Pearson correlation coefficient (r). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

3.1 Study Population and Clinical Profile

Sixty-six newly diagnosed PCOS women were enrolled. Baseline characteristics are presented in Table 1. The largest age group was 31–35 years (30.3%), followed by 21–25 years (27.3%). Oligomenorrhoea was the predominant menstrual pattern (47.0%), followed by amenorrhoea (33.3%); 93.9% of participants had some form of menstrual dysfunction. Hirsutism was the most frequent clinical manifestation (62.1%), followed by acne (42.4%), obesity (24.2%), and acanthosis nigricans (15.2%). Insulin resistance (HOMA-IR >2.5) was present in 71.2% (n=47) of participants.

Table 1: Baseline Clinical Characteristics of PCOS Participants (N=66)

Characteristic	N	Percentage (%)
Age group: ≤20 years	12	18.2
Age group: 21–25 years	18	27.3
Age group: 26–30 years	12	18.2
Age group: 31–35 years (largest group)	20	30.3
Age group: >35 years	4	6.1
Oligomenorrhoea	31	47.0
Amenorrhoea	22	33.3
Irregular menses	9	13.6
Eumenorrhoea (regular cycles)	4	6.1
Any menstrual dysfunction	62	93.9
Hirsutism	41	62.1
Acne	28	42.4
Obesity (clinical)	16	24.2
Acanthosis Nigricans	10	15.2
Insulin Resistance present (HOMA-IR >2.5)	47	71.2
Insulin Resistance absent (HOMA-IR ≤2.5)	19	28.8

N: Number; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance

3.2 Vitamin D Status in the PCOS Cohort

Vitamin D status distribution is presented in Table 2. Vitamin D deficiency (<20 ng/mL) was detected in 80.3% (n=53), insufficiency (20–30 ng/mL) in 13.6% (n=9), and only 6.1% (n=4) had sufficient levels (>30 ng/mL). Combined suboptimal vitamin D status (deficiency + insufficiency) was present in 93.9% of the PCOS cohort. The overall mean serum vitamin D was 17.34 ± 8.84 ng/mL, well below the sufficiency threshold.

Table 2: Distribution of Serum Vitamin D Status in PCOS Women (N=66)

Serum Vitamin D [25(OH)D] Status	N	%	Mean (ng/mL)
Deficient (<20 ng/mL)	53	80.3	—
Insufficient (20–30 ng/mL)	9	13.6	—

Sufficient (>30 ng/mL)	4	6.1	—
Suboptimal (deficient + insufficient)	62	93.9	—
Overall cohort	66	100	17.34 ± 8.84

Values expressed as n (%) or mean ± SD.

3.3 Vitamin D Status and Mean Vitamin D by Insulin Resistance Group

The comparison of vitamin D status and mean serum vitamin D between insulin-resistant and non-insulin-resistant PCOS women is shown in Table 3. Among insulin-resistant women (n=47), 87.2% were vitamin D-deficient, 8.5% insufficient, and only 4.3% sufficient. Among non-insulin-resistant women (n=19), 63.2% were deficient, 26.3% insufficient, and 10.5% sufficient. This distribution was statistically significant ($\chi^2 = 3.928$, p=0.047), confirming that vitamin D deficiency is significantly more prevalent among insulin-resistant PCOS women.

Mean serum vitamin D was significantly lower in insulin-resistant PCOS women (15.06 ± 7.25 ng/mL) compared with non-insulin-resistant women (19.54 ± 10.15 ng/mL; t = 6.013, p=0.017), representing a clinically meaningful difference of approximately 4.5 ng/mL, even though both groups remain in the deficient-to-insufficient range.

Table 3: Serum Vitamin D Status and Mean Vitamin D According to Insulin Resistance Status in PCOS Women

Vitamin D Category	IR Absent (n=19)	IR Present (n=47)	Test Statistic	p-value
<20 ng/mL (Deficient)	12 (63.2%)	41 (87.2%)	χ^2=3.928	0.047*
20–30 ng/mL (Insufficient)	5 (26.3%)	4 (8.5%)		
>30 ng/mL (Sufficient)	2 (10.5%)	2 (4.3%)		
Mean vitamin D (ng/mL)	19.54 ± 10.15	15.06 ± 7.25	t = 6.013	0.017*
Overall mean vitamin D: 17.34 ± 8.84 ng/mL				

IR: Insulin Resistance (HOMA-IR >2.5); *Statistically significant (p<0.05); Values as n (%) or mean ± SD.

3.4 Correlation between Serum Vitamin D and HOMA-IR

Pearson correlation analysis revealed a statistically significant negative correlation between serum 25(OH)D and HOMA-IR (r = −0.248, p=0.045) as presented in Table 4. As vitamin D levels decreased, HOMA-IR increased, confirming an inverse linear relationship between these two parameters across the full range of values in the PCOS cohort.

Table 4: Pearson Correlation of HOMA-IR with Serum Vitamin D [25(OH)D] in PCOS Women

Parameters	Pearson r	p-value
HOMA-IR vs Serum Vitamin D [25(OH)D]	−0.248	0.045*
Direction of correlation	Negative (inverse)	
Interpretation	As serum vitamin D decreases, HOMA-IR increases	

r: Pearson correlation coefficient; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; *p<0.05

DISCUSSION

The present study establishes three principal findings in newly diagnosed PCOS women from Central India. First, vitamin D deficiency is near-universal—80.3% were deficient and 93.9% had suboptimal vitamin D status, with an overall mean of 17.34 ± 8.84 ng/mL. Second, vitamin D levels were significantly lower in insulin-resistant PCOS women than in non-insulin-resistant women (15.06 vs 19.54 ng/mL, p=0.017), and vitamin D deficiency was significantly more prevalent in the insulin-resistant group (87.2% vs 63.2%, p=0.047). Third, HOMA-IR correlated significantly and negatively with serum 25(OH)D (r = −0.248, p=0.045).

The near-universal vitamin D deficiency in our PCOS cohort aligns with published reports from similar populations. Gokosmanoglu et al. (2020) documented 86% vitamin D deficiency in Turkish PCOS women, with significantly elevated HOMA-IR, BMI, and fasting glucose in the deficient group. Omran et al. (2020) reported 90% deficiency in an Egyptian PCOS cohort with none achieving sufficient vitamin D status. In the Indian context, Aghade (2024) found significantly lower vitamin D and higher HOMA-IR in PCOS women in Maharashtra, with a significant negative correlation between the two—directly paralleling our findings. The comprehensive review by Mohan et al. (2023) confirmed that vitamin D deficiency exacerbates insulin resistance in PCOS through impaired calcium homeostasis, reduced VDR-mediated insulin receptor activity, and heightened inflammatory cytokine production.

The moderate magnitude of the negative correlation ($r = -0.248$) is consistent with the broader literature and reflects the multifactorial pathogenesis of insulin resistance in PCOS. Vitamin D operates through several parallel pathways—direct VDR-mediated enhancement of insulin receptor gene expression, calcium-dependent improvement of insulin-stimulated glucose transport, and anti-inflammatory suppression of TNF-α and IL-6 (Argano et al., 2023). Adiposity acts as a significant confounder, simultaneously sequestering vitamin D in fat tissue (reducing bioavailability) and independently worsening insulin resistance in PCOS women, which explains why the correlation strength is moderate rather than strong. The significantly lower mean vitamin D in insulin-resistant PCOS women (15.06 vs 19.54 ng/mL), even though both groups remain in the deficient range, has important clinical implications. It suggests that within a universally deficient Indian PCOS population, progressively lower vitamin D reflects—or contributes to—progressively worsening insulin signalling. Bharti and Mitra (2025) similarly documented 68% insulin resistance in an Indian PCOS cohort and emphasised the need for routine metabolic screening, reinforcing the importance of affordable biochemical markers in resource-limited settings.

Vitamin D supplementation (20,000–50,000 IU weekly or biweekly) has demonstrated improvements in insulin sensitivity, menstrual regularity, and androgen profiles in PCOS women in randomised trials (Mohan et al., 2023; Asemi et al., 2015). Given the universality of deficiency in the present cohort and the low cost of supplementation, empiric vitamin D repletion in all newly diagnosed PCOS women is clinically justifiable, pending confirmatory evidence from adequately powered Indian randomised controlled trials.

Strengths of this study include validated CLIA methodology for 25(OH)D measurement, a rigorously defined PCOS cohort with strict exclusion of confounders including vitamin D supplement users, standardised sample collection and same-day processing, and a focus on a Central Indian PCOS population that is under-represented in the global literature. Limitations include the cross-sectional design precluding causal inference, modest sample size (n=66), absence of a healthy control group and PCOS phenotype subclassification, single urban tertiary care centre setting limiting generalisability to rural or lean-PCOS populations, and non-adjustment for season of blood sampling.

CONCLUSION

In newly diagnosed PCOS women from Central India, vitamin D deficiency is near-universal (80.3% deficient; 93.9% suboptimal) and is significantly associated with insulin resistance. A significant negative correlation between serum 25(OH)D and HOMA-IR ($r = -0.248$, $p=0.045$) confirms that lower vitamin D tracks greater insulin resistance in PCOS. These findings highlight the urgent need for routine vitamin D screening and targeted supplementation as part of the metabolic evaluation of all newly diagnosed PCOS women in India. This study contributes population-specific evidence to the growing literature on PCOS metabolic risk stratification and supports the clinical utility of this affordable, widely available biomarker in resource-limited settings. Prospective, adequately powered randomised controlled trials examining whether vitamin D correction reduces HOMA-IR in Indian PCOS women are warranted.

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REFERENCES

1. Abdesselam, A., Zidoum, H., Zadjali, F., Hedjam, R., Al-Ansari, A., & Bayoumi, R. (2021). Estimate of the HOMA-IR cut-off value identifying subjects at risk of insulin resistance using a machine learning approach. Sultan Qaboos University Medical Journal, 21(4), 604–612.
2. Aghade, S. M. (2024). Evaluation of serum vitamin D and ferritin level and their correlation with insulin resistance and adiposity in women with polycystic ovarian syndrome: A hospital-based study in Maharashtra, India. Indian Journal of Health Sciences and Biomedical Research KLEU, 17(3), 217–223.
3. Argano, C., Mirarchi, L., Amodeo, S., Orlando, V., Torres, A., & Corrao, S. (2023). The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: State of the art. International Journal of Molecular Sciences, 24(20), 15485.
4. Asemi, Z., Foroozanfard, F., Hashemi, T., Bahmani, F., Jamilian, M., & Esmaillzadeh, A. (2015). Calcium plus vitamin D supplementation affects glucose metabolism and lipid concentrations in overweight and obese vitamin D-deficient women with polycystic ovary syndrome. Clinical Nutrition, 34(4), 586–592.
5. Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale, H. F., & Futterweit, W. (2009). The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: The complete task force report. Fertility and Sterility, 91(2), 456–488.
6. Bharali, M. D., Rajendran, R., Goswami, J., Singal, K., & Rajendran, V. (2022). Prevalence of polycystic ovarian syndrome in India: A systematic review and meta-analysis. Cureus.

7. Bharti, S. S., & Mitra, D. (2025). Evaluation of fasting insulin and HOMA-IR in PCOS patients: A cross-sectional biochemistry study. International Journal of Current Pharmaceutical Review and Research, 17(7), 1720–1725.
8. Burtis, C. A., Ashwood, E. R., & Bruns, D. E. (2012). Tietz textbook of clinical chemistry and molecular diagnosis (5th ed.). Elsevier.
9. Diamanti-Kandarakis, E., & Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. Endocrine Reviews, 33(6), 981–1030.
10. Gokosmanoglu, F., Onmez, A., & Ergenc, H. (2020). The relationship between vitamin D deficiency and polycystic ovary syndrome. African Health Sciences, 20(4), 1880–1886.
11. Grundmann, M., & von Versen-Höynck, F. (2011). Vitamin D — roles in women's reproductive health? Reproductive Biology and Endocrinology, 9(1), 146.
12. Gupta, M., Singh, D., Toppo, M., Priya, A., Sethia, S., & Gupta, P. (2017). A cross sectional study of polycystic ovarian syndrome among young women in Bhopal, Central India. International Journal of Community Medicine and Public Health, 5(1), 95.
13. Holick, M. F. (2007). Vitamin D deficiency. New England Journal of Medicine, 357(3), 266–281.
14. Mohan, A., Haider, R., Fakhor, H., Hina, F., Kumar, V., & Jawed, A. (2023). Vitamin D and polycystic ovary syndrome (PCOS): A review. Annals of Medicine and Surgery, 85(7), 3506–3511.
15. Morgante, G., Darino, I., Spanò, A., Luisi, S., Luddi, A., & Piomboni, P. (2022). PCOS physiopathology and vitamin D deficiency: Biological insights and perspectives for treatment. Journal of Clinical Medicine, 11(15), 4509.
16. Omran, E. F., Ramzy, A., Shohayeb, A., Farouk, N., Soliman, M., & Baz, H. (2020). Relation of serum vitamin D level in polycystic ovarian syndrome (PCOS) patients to ICSI outcome. Middle East Fertility Society Journal, 25(1), 22.
17. Rotterdam ESHRE/ASRM-sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Human Reproduction, 19(1), 41–47.