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PREVALENCE OF VITAMIN D DEFICIENCY AND ITS ASSOCIATION WITH INSULIN RESISTANCE AMONG INDIAN WOMEN WITH POLYCYSTIC OVARY SYNDROME: EVIDENCE FROM A CROSS-SECTIONAL STUDY

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Keywords:

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ABSTRACT: The current study aims to assess whether circulating levels of 25(OH) D are related to insulin resistance in PCOS individuals, or whether this association is confounded by other pathophysiological factors related to the syndrome. This cross-sectional study included 151 women who were diagnosed with PCOS based on the Rotterdam consensus, recruited from a tertiary healthcare center in South Gujarat, India. Data collection encompassed anthropometric parameter (BMI and WC) and biochemical indices (fasting blood glucose, fasting insulin, HOMA-IR, and serum 25(OH)D levels). Vitamin D levels were used to classify participants into three distinct categories: deficiency (serum 25(OH) D level <20 ng/mL), insufficiency (serum 25(OH)D level 20–32 ng/mL), and sufficiency (serum 25(OH)D level>32 ng/mL). From the total participants, 115 (76.15%) exhibited suboptimal serum 25(OH)D levels. Insulin resistance is characterized by HOMA-IR value (>2.5) and QUICKI value (<0.349), was observed in 69.5% of the cohort. While a substantial proportion74.3% of individuals with low level of vitamin D also demonstrated insulin resistance. Kruskal-Wallis test revealed a non-significant difference in IR across vitamin D categories. In supporting to that correlation coefficient was found very weak and non-significant among serum 25(OH)D concentrations and insulin resistance parameters, including fasting insulin and HOMA-IR, QUICKI. Despite a higher prevalence of insulin resistance amid individuals with deficient vitamin D, this investigation did not demonstrate a significant relationship between circulating 25(OH)D concentrations and insulin resistance in females affected by polycystic ovary syndrome. Larger controlled studies with adjustment for adiposity and other confounders are needed to confirm these findings.

INTRODUCTION: Polycystic ovary syndrome is a complex endocrine disorder, impacting all age group but predominantly affects reproductive women age group [1].

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This condition is marked by the presence of elevated concentrations of androgens in body called as hyperandrogenism (HA), irregularities in the ovulation process considered as ovulatory dysfunction (OD) and multiples cysts form in the ovaries known as polycystic ovaries (PCO) [2].

A significant proportion of women diagnosed with polycystic ovary syndrome show insulin resistance, compensatory hyperinsulinemia and either overweight or obese. PCOS patients have complicated pathophysiology of insulin resistance

[3, 4]. Patients characterised with higher BMI, waist circumference and obesity with elevated insulin resistance [5]. PCOS patients have high prevalence of obesity which may show the association between obesity and insulin sensitivity [6]. PCOS women may have high risk of infertility and negative pregnancy outcomes, which may develop severe health issues. Various complications like cardiovascular disease, diabetes mellitus and other metabolic disorders are higher in PCOS women who have insulin resistance [7]. These circumstances significantly adversely affect the well-being of physical and mental health of women in their reproductive years, which in turn increases the social challenge and their burden [8].

Vitamin D is essential for basic metabolic processes, *i.e.,* it helps in calcium and phosphorus absorption for healthy bones and teeth. Also, it supports immune function, mood regulation and inflammation reduction. Insufficient vitamin D can resist these vital functions. Detection of vitamin D receptor (VDR) in various components of the female reproductive system indicates that there is an involvement of vitamin D inregulatory function and synthesis of hormones in ovary. In female ovaries have granulosa cells (GC) which contain VDR in both their nuclei and cytoplasm, suggesting that it may have role for the physiological processes in ovarian follicles. 25(OH)D activates into 1,25(OH)2D metabolite and inducing metabolic effects by expressing VDR in reproductive tissues [9].

A growing global health concern, deficiency of vitamin D may lead to various metabolic imbalances, including effect on glucose metabolism, cardiovascular disorder, autoimmune illnesses and psychological disease [10]. Findings indicate Hypovitaminosis D shifts cytokine balance to the proinflammatory levels [11]. Evidence suggests Vitamin D may activate the insulin receptor's expression, which improves insulin production and release, and inhibit proinflammatory cytokines [12]. Vitamin D is involved in insulin synthesis and release and contribute for glucose metabolism by regulating intracellular Ca^{2+} physiology required for the same [13]. In animal model research, it has been established that vitamin D reduces ROS increases antioxidant action which may preserve β cell function [14]. Activation of VDR alters gene expression which controls β cell viability and insulin secretion [15]. Likewise, vitamin D deficiency may cause metabolic and reproductive issues that are linked to PCOS. Low level of vitamin D may contribute to such dysfunctions through mechanisms that involve insulin resistance [16].

Various research has studied the relationship among low concentration vitamin D and women diagnosed with PCOS, also metabolic and clinical attributes of PCOS patients [17–19], but the exact nature of this link is still remaining unidentified in Indian population. Even though, there is an increasing number of intervention trials that are assessing the effect of vitamin D supplements in women diagnosed with PCOS, there are insufficient results which could assistance a connection among serum 25(OH)D concentrations and insulin resistance due to regional variations in lifestyle, sunlight exposure and genetic predisposition. Our research aimed to investigate the connection between a lack of vitamin D level and insulin resistance in individuals diagnosed with PCOS in an Indian clinical setting.

Patient and Method:

Patients: This cross-sectional analysis included 151 women diagnosed with PCOS from tertiary care Hospital, Navsari, Gujarat, India, between August 1, 2024, and January 31, 2025. Ethical clearance for the study was obtained from the Institutional Ethics Committee of Maliba Pharmacy College (Approval No: MPC/18/01/24-25), and informed consent was acquired from all participants prior to enrolment.

The assessment of PCOS was established based on the Rotterdam criteria (2003) [2]. In accordance with Rotterdam criteria; two out of the three characteristics; ovulatory dysfunction, which is indicated by irregular menstruation; hyperandrogenism as indicated by clinical and biochemical parameter; and polycystic ovaries which are indicated by the existence of 12 follicle per ovary an elevated mean ovarian volume exceeding 10 ml; are necessary diagnosis.

Sample Size: As such separate a priori sample-size calculation was not performed specifically for the association between vitamin D status and insulin resistance, as this analysis was conducted using

available data from the parent study. However, a post-hoc detectable-effect assessment indicated that a sample size of 151 participants would provide approximately 80% power at a two-sided α level of 0.05 to detect a correlation of about r = 0.23 or greater between vitamin D level and insulin-resistance indices.

Inclusion Criteria: Inclusion criteria were as follows: (1) Women aged 15-45 diagnosed with PCOS according to Rotterdam criteria (2003) (2) not receiving vitamin D supplements in any form from past three months (3) patients who are willing to provide informed consent.

Exclusion Criteria: Exclusion criteria were as follows: (1) already suffering from endocrine disorder like diabetes mellitus, thyroid disorders, or hyperprolactinemia (2) use of medications which altering glucose metabolism, insulin sensitivity, or hormone levels (3) at the time of enrolment either pregnant or lactation.

Anthropometric and Biochemical Assessments: Demographic data, including age, height in cm, weight in kg, and waist circumference in inches, were documented. Body mass index (BMI) was computed from these data. Blood samples were collected during the early follicular phase, between days 2 and 5 of menstrual bleeding, whenever spontaneous menstruation occurred. In women with oligomenorrhea, sampling was scheduled during days 2–5 following spontaneous menses where feasible. In women with amenorrhea, strict follicular-phase timing could not always be ensured, and sampling was performed at clinical evaluation. Fasting glucose and insulin were assessed using standard protocols. Insulin resistance indices were computed utilizing the homeostatic model assessment for insulin resistance (HOMA-IR) index and the quantitative insulin sensitivity check index (QUICKI) [20]. In this study, IR was considered as HOMA-IR>2.5 and QUICKI<0.349, based on previous epidemiological studies [21, 22].

A commercially available rapid chromatographic immunoassay kit (AccuTest Vitamin D, Accurex Biomedical Pvt. Ltd., India) was used to test serum 25-hydroxyvitamin D[25(OH)D].Deficiency of vitamin D is characterized by a 25(OH)D concentration of below 20ng/ml, a level ranging between 20 to 32 ng/ml indicates an insufficiency of vitamin D, and sufficient vitamin D levels are detected with the concentration exceeds 32ng/ml; provided by assay manufacturer. These ranges were used as an assay-specific operational threshold to maintain consistency with the calibration and interpretation recommended for the diagnostic kit.

Statistical Analysis: Demographic values were presented as mean (standard deviation). Categorical variables were reported as frequencies and percentages. To identify the distinctions among the categories, first we applied the Kolmogorov-Smirnov test to check normality.

Comparisons among vitamin D categories were conducted Kruskal-Wallis (non-parametric) test. Spearman's correlations were employed to ascertain relationship among variables. A data analysis was performed using SPSS version 27 software. Differences were considered significant when the p-value was less than 0.05.

RESULT:

Demographics and Clinical Characteristics: Overall, 151 PCOS patients were involved in this research. The mean BMI of the research cohort was 25.72 kg/m^2 with standard deviation 3.91kg/m^2. Likewise, mean fasting insulin and HOMA-IR value were found higher than their normal range **Table 1.**

TABLE 1: BASELINE FEATURES OF STUDY PARTICIPANTS

Variable	Mean ± SD
Age (years)	28.35 ± 6.01
Weight (kg)	63.49 ± 10.65
Height (cm)	157.02 ± 5.62
BMI (kg/m^2)	25.72 ± 3.91
Waist Circumference (in)	35.37 ± 3.08
Fasting Blood Sugar (mg/dL)	91.60 ± 13.72
Fasting Insulin (µU/mL)	16.67 ± 10.86
HOMA-IR	3.71 ± 2.26
QUICKI	0.31 ± 0.02

Tests of Normality: Kolmogorov-Smirnov test was used to assess normality of data. Fasting blood sugar, fasting insulin, HOMA-IR and QUICKI values did not follow normal distribution (p<0.05). Hence, non-parametric methods like Kruskal-Wallis test is used to find out difference among categories.

Comparison across Vitamin D Categories: About 45.7% PCOS patients were having vitamin D deficient level, while 30.46% patients were having vitamin D insufficient level. Comparison of clinical and biochemical parameters across vitamin D categories (sufficient, insufficient, deficient) revealed no statistically significant differences in BMI, fasting glucose, fasting insulin, HOMA-IR, or QUICKI indices ($p > 0.05$, Kruskal–Wallis test). Although women with vitamin D deficiency tended to have slightly higher insulin levels and HOMA-IR values, these differences did not reach statistical significance **Table 2.**

TABLE 2: METABOLIC PARAMETERS BY VITAMIN D LEVEL

Variable	Vitamin D deficient (n=69)	Vitamin D insufficient (n=46)	Vitamin D sufficient (n=36)	Kruskal-Wallis (H)	p value
BMI	25.204	25.307	25.267	0.439	0.803
Waist Circumferences	34	35	36	3.693	0.158
Fasting insulin	14.32	14.95	14.61	0.049	0.976
HOMA-IR	3.12	3.64	3.10	0.119	0.942
QUICKI	0.32	0.31	0.32	0.99	0.952

Data are presented as median [IQR] because variables were non-normally distributed. Between-group comparisons were performed using the Kruskal–Wallis test. H = Kruskal–Wallis test statistic; IQR = interquartile range.

Box plot was generated to find out the distribution of HOMA-IR and to find out distinct among categories. **Fig. 1** shows a box plot of HOMA-IR values among Vitamin D categories.

Women with insufficient group showed median close to the center of the box of HOMA-IR values compared to sufficient and deficient group. In contrast, the box plot for deficient group shows a longer upper whisker, indicating a right skewed distribution. Each category shows outliers, but insufficient group had extreme outlier.

While in **Fig. 2** QUICKI value of deficient group displays symmetric distribution with balanced whiskers; while insufficient group showed a right-skewed distribution and sufficient group showed a left skewed distribution.

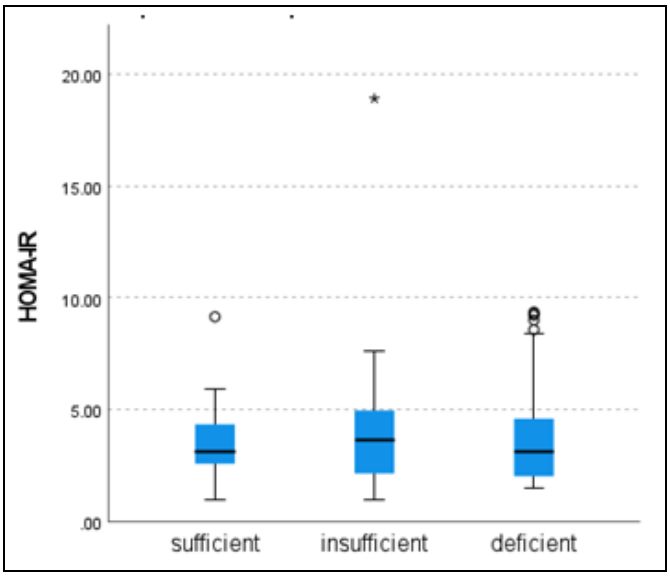

FIG. 1: BOX PLOT OF HOMA-IR VALUES ACROSS VITAMIN D CATEGORIES IN WOMEN WITH PCOS

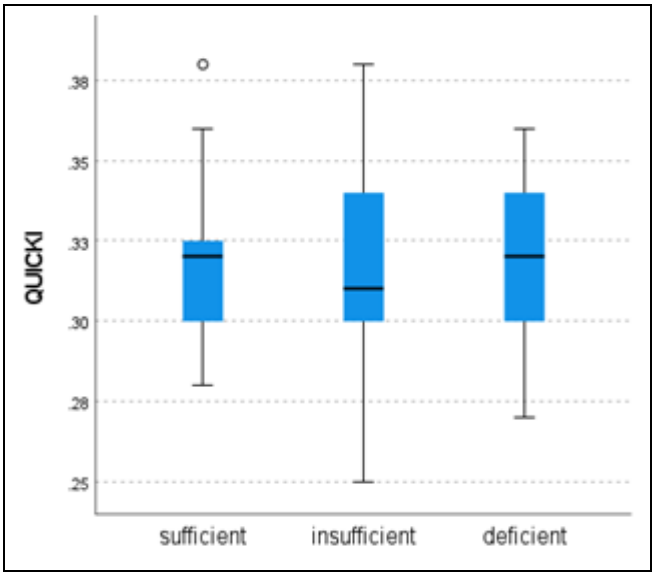

FIG. 2: BOX PLOT OF QUICKI VALUES ACROSS VITAMIN D CATEGORIES IN WOMEN WITH PCOS

Correlation Analysis: Spearman's rank correlation presents weak correlation analysis of vitamin D with insulin resistance indicators, including BMI, waist circumference, fasting plasma insulin and HOMA-IR **Table 3.** Specifically, vitamin D showed a negative correlation with fasting blood sugar ($r = –0.003$, $p > 0.05$) and a positive correlation with fasting insulin ($r = 0.002$, $p > 0.05$), HOMA-IR ($r = 0.004$, $p > 0.05$) and QUICKI ($r = 0.018$, $p > 0.05$).

TABLE 3: RELATIONSHIP BETWEEN CIRCULATING 25(OH)D AND BIOMARKERS OF INSULIN RESISTANCE

Variables	r	p value
BMI	0.012	0.887
Waist Circumference	-0.154	0.059
Fasting Glucose	-0.003	0.967
Fasting Insulin	0.002	0.981
HOMA-IR	0.004	0.965
QUICKI	0.018	0.831

A statistically significant and strong positive correlation was identified between body mass index (BMI) and insulin resistance, indicated by HOMA-IR ($r = 0.881$, $p < 0.001$), implying increases in BMI are closely associated with elevated insulin resistance. Additionally, a moderate positive association was detected among BMI and fasting plasma glucose levels ($r = 0.464$, $p < 0.001$), implying a relationship between adiposity and glucose dysregulation. Furthermore, a strong positive relationship was found among BMI and fasting insulin concentrations ($r = 0.864$, $p < 0.001$), reinforcing the link between obesity and hyperinsulinemia. These reports suggest the important role of adiposity in modulating insulin sensitivity and glucose metabolism in women with PCOS **Table 4.**

TABLE 4: CORRELATION MATRIX OF BMI WITH MARKERS OF INSULIN RESISTANCE

Variables	r	p value
Waist Circumference (inches)	0.257	0.015
Fasting Glucose (mg/dL)	0.464	<.001
Fasting Insulin (µU/mL)	0.864	<.001
HOMA-IR	0.881	<.001

TABLE 5: REGRESSION ANALYSIS

Variable	B (Unstandardized)	SE	Beta (Standardized)	p value	95% CI
Age	0.018	0.031	0.047	0.575	-0.045-0.080
BMI	0.175	0.059	0.302	0.003	0.058-0.291
Waist Circumference	-0.179	0.073	-0.244	0.015	-0.3223- -0.036
Vitamin D	0.002	0.230	0.001	0.992	-0.453-0.458

R^2=0.075, Adjusted R^2=0.050, F=2.971, p=0.021

Multiple linear regression analysis **Table 5** demonstrated that BMI ($\beta = 0.302$, p=0.003) and waist circumference ($\beta = -0.244$, $p = 0.015$) were significant predictors of HOMA-IR. After adjusting for confounders, participants with vitamin D levels were not significantly ($p = 0.992$). Age was not significantly associated with HOMA-IR ($p = 0.575$). The overall model was significant ($F = 2.971$, $p = 0.021$) and explained 5% of the variance (adjusted $R^2 = 0.050$).

DISCUSSION: Nowadays, polycystic ovary syndrome (PCOS) is a widespread endocrine disorder which influence all age group women, may develop serious consequences like cardiovascular or metabolic disorder. A prevalent metabolic dysfunction observed in such conditions is insulin resistance. There is still uncertainty regarding molecular pathways that causes resistance of insulin in PCOS.

Recently considerable focus has been given on vitamin D status as a probable aspect in metabolic disorder. One of vitamin D deficiency pleiotropic effect may be consider as insulin resistance to its involvement in several cellular systems [5].

From our study it was determined that 30.46% of subjects had vitamin D insufficiency while 45.7% subjects had vitamin D deficiency. Numerous studies have demonstrated an association among vitamin D deficiency and women with PCOS [23]. Yildizhan *et al.* reported 67% patient of PCOS were suffering from vitamin D [24]. Our study revealed a significant vitamin D deficiency in PCOS patients, align to the previous research.

Our data also observed; in comparison to patients having insufficiency and sufficiency group respectively, patients having deficiency group had the highest HOMA-IR levels. Epidemiological

findings report inverse correlation among 25(OH)D concentrations and insulin resistance and glucose intolerance [25]. Despite of numerous researches among vitamin D deficiency and insulin resistance, mechanism is unclear due to undiscovered link at cellular level of these two parameters in PCOS. One of cohort study reported chronic inflammation with low severity; induces vitamin D deficiency and insulin resistance which might be absent in acute inflammation [26]. At molecular level endocrine effects of vitamin D depend on tissue expression activated by enzymes and VDR; so directly systemic 25(OH)D may not capture local hormone or adipose signalling. Metabolic disorder could decrease vitamin D that do not reflect a causal vitamin D-IR pathway which raise question for interpreting of null hypothesis [27].

Although the correlation among vitamin D deficiency and insulin resistance has been well investigated, limited data are available from the Indian population, particularly from hospital-based cohorts. Our findings reported that there is no significant connection among vitamin D deficiency with insulin resistance in patients with polycystic ovary syndrome in this region. Several epidemiological studies have observed a positive association of glucose metabolism with vitamin D deficiency [28, 29]. Hahn *et al.* found that lower levels of 25-OH vitamin D are linked with insulin resistance and obesity in patients with PCOS [30]. Kotsa *et al* reported beneficial impact of cholecalciferol on glucose metabolism parameters in PCOS patients [31]. But genetic variation in population alter vitamin D status and its metabolic consequences because of heterogeneity in cohorts. There are variants in the VDR and metabolising genes modify responses and may change the link between circulating 25(OH)D concentrations and insulin sensitivity [32]. PCOS phenotypes may show vitamin D-IR relationships stronger but no correlation found in overall. So heterogenicity of phenotypes may reduce overall correlations [33].

Vitamin D-IR relationship is affected by co existing exposures and comorbidities which may hinder associations. Numerous observational studies report correlation is sensitive to adiposity, lifestyle and other covariates; therefore, null hypothesis for correlation between these two can be accepted if residual confounding factor is not properly controlled [34]. According to studies, the majority of obese PCOS patients exhibit insulin resistance (IR). Furthermore, studies concluded that in obese patients, insulin resistance (IR) and low 25 (OH) D concentration are inversely correlated [12, 18]. In compared to the control group for BMI, women who have been diagnosed with PCOS showed a tendency to have larger fat cells, as well as a higher waist to hip ratio [35]. This indicates that women with PCOS not only have higher body fat distribution but also have its consequences like obesity and metabolic disorders. These changes are strongly related to lower insulin sensitivity. The major cause for insulin resistance in PCOS patient is declined level of serum adiponectin; a hormone that increase insulin sensitivity [36]. In adipose tissue, reduction in adiponectin levels causes a rise in inflammatory cytokine which further contribute to insulin resistance [37].

Our result suggests vitamin D deficiency does not have direct link in insulin resistance development in this hospital based cross sectional sample using the present analytical approach. Prevalence of hypovitaminosis D, skin diseases, different sun exposure, for tified food intake and lifestyle changes in population may alter associations. These confounding factors may vary with vitamin D status and insulin tolerance which may fail to control bias result towards null hypothesis. Similarly, disease like metabolic syndrome or liver disease and drugs could change the insulin sensitivity or vitamin D concentrations by altering pharmacokinetic; can confound the correlation [32, 34].

The present findings should be viewed within the framework of the 2023 International Evidence-based Guideline for PCOS. The guideline emphasizes the increased cardiometabolic vulnerability of women with PCOS and supports assessment of cardiovascular risk factors and glycaemic status in this population. In this context, the frequent presence of insulin resistance in our study participants, particularly its relationship with BMI, underlines the clinical relevance of metabolic risk evaluation in PCOS. However, while insulin resistance is recognized as an important contributor to PCOS pathophysiology, the guideline cautions that available insulin assays have limited applicability in routine clinical care. Similarly,

vitamin D status is not identified in the guideline as a diagnostic criterion or primary treatment target for PCOS [38]. There were few limitations to our investigation. We solely examined PCOS patients; in lack of a control group in our research. This may have limited the depth of our analysis and the validation of our results because the control group could provide a comparative baseline to strengthen our results. Vitamin D thresholds vary among studies and clinical guidelines, and there is no universally accepted cut-off for sufficiency. In this study, a value of >32 ng/mL was selected because it was the reference threshold provided by the diagnostic kit used for serum 25(OH)D estimation. Therefore, comparisons with studies using alternative cut-offs, such as ≥20 ng/mL or ≥30 ng/mL, should be made carefully. Future studies may consider applying standardized or multiple threshold definitions to improve comparability across populations. Future studies should incorporate mechanistic analyses, such as proteomics or gene polymorphism evaluation, and Future research should include mechanistic studies, such as the assessment of proteomics or gene polymorphisms, along with randomized controlled trials that have larger participant groups.

CONCLUSION: In conclusion, no direct correlation was found among serum 25(OH)D concentrations and hyperinsulinemia in women with PCOS. Overall, vitamin D deficiency and insulin resistance were identified among the women with PCOS included in this study. However, due to the absence of a non-PCOS comparator group, no definitive conclusion can be drawn regarding whether these abnormalities are uniquely increased in PCOS. Further controlled studies are needed to confirm these observations.

Ethics Approval: The study was authorized by institutional ethics committee of Maliba Pharmacy College (Ref No. MPC/18/01/24-25)

CONFLICT OF INTEREST: The authors have no conflicts of interest regarding this investigation.

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