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Serum Vitamin D and Salivary Cortisol as Modulators of Endocrine Dysregulation in Women with Polycystic Ovary Syndrome

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common hormonal disorder that affects women of reproductive age, associated with issues like high androgen levels, insulin resistance, and various metabolic risks. Many women with PCOS also experience vitamin D deficiency, which can impact follicle development, inflammation, and overall reproductive health. Additionally, high cortisol levels can throw off metabolism and the body's stress response.

Methods: This prospective study was conducted at Prasad Institute of Medical Sciences, Lucknow (January–December 2024), to assess serum vitamin D and salivary cortisol as modulators of endocrine dysregulation in women with PCOS. Women aged 18–35 years diagnosed according to the Rotterdam criteria were included; pregnant, lactating, or comorbid cases were excluded. Anthropometric data (including BMI, body fat, and skeletal muscle) were recorded. Blood and saliva samples were analysed for cortisol, SHBG, vitamin D, estradiol, LH, FSH, and progesterone using ELISA and CMIA methods. Ethical approval and informed consent were obtained.

Results: Vitamin D deficiency was prevalent in both groups, with 52 participants in Group A and 20 in Group B showing levels below 30 ng/mL. Group B showed higher visceral fat (0.19 vs. 0.03), body fat (0.15 vs. 0.07), and skeletal muscle mass (0.20 vs. 0.06), indicating a more balanced body composition. Vitamin D sufficiency was greater in Group B (22%) than in Group A (7%), highlighting significant deficiency in PCOS patients and intergroup differences in body composition.

Conclusion: The study concluded with a deficiency of vitamin D in group A, whereas group B showed a normal composition in the body, fat, and muscle.

Key-words: Polycystic Ovary Syndrome, Vitamin D Deficiency, Salivary Cortisol, Endocrine Dysregulation, Body Composition

INTRODUCTION

Polycystic ovary syndrome is recognized as one of the most predominant endocrine disorders affecting women of reproductive age, characterized mainly by hyperandrogenism, chronic anovulation, and polycystic ovarian morphology [1]. Disorders affecting certain endocrine systems, such as the thyroid, gonadal, and hypothalamic-pituitary-adrenal systems, are part of the aetiology and pathophysiology of PCOS. Reproductive health and metabolic profiles in PCOS are both impacted by endocrine dysregulation that raises the risk of insulin resistance, type 2 diabetes, dyslipidaemia, and cardiovascular disease [2].

The relationship between blood vitamin D and salivary cortisol, as important modulators of endocrine and metabolic activity, has recently lost academic attention due to the involvement of numerous hormones in PCOS. Vitamin D, once primarily associated with bone metabolism, is now recognised for its significant influence on reproductive function, insulin sensitivity, and the regulation of inflammation [3]. Many studies establish that vitamin D deficiency is common in women with PCOS, with investigations showing that up to 85% of those afflicted had serum 25-hydroxyvitamin D concentrations below optimal. The main characteristics of

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PCOS, such as irregular menstrual cycles, excessive hair growth, hyperandrogenism, interrupted ovulation, and a variety of metabolic issues, such as raised insulin resistance and cardiovascular risk factors, are linked to this insufficiency. In addition, vitamin D deficiency affects follicular growth and calcium regulation, contributing to the distinctive ovarian dysfunction observed in PCOS [4].

With cortisol as its principal effector hormone, the HPA has also been related to the pathophysiology of PCOS, primarily in relation to stress response, metabolic changes, and inflammatory state. Continued HPA stimulation can raise cortisol levels, disrupt energy metabolism, and increase central, all of which contribute to insulin resistance and the metabolic syndrome in PCOS [5]. They are measured by salivary cortisol, and the development or progression of PCOS is associated with evidence to advise a direct association between the activity of the HPA. Hypercortisolaemia may improve androgen production, glucose homeostasis, and participate in the cross-talk between stress and reproductive function.

Studies indicate that elevated cortisol may contribute to metabolic and endocrine abnormalities seen in PCOS, and both are associated with vitamin D deficiency [6]. Vitamin D has been shown to affect the expression of genes involved in steroidogenesis, reduce inflammation, and modulate immune response, which in turn may mitigate the negative effects of cortisol levels and HPA dysfunction. Equally, vitamin D supplementation has been shown to have potential benefits in improving insulin sensitivity, menstrual regularity, and reducing hyperandrogenism and inflammatory markers in women with PCOS. However, more randomised controlled trials are needed. The complex balance between these two hormonal modulators emphasises the comprehensive nature of neuroendocrine and metabolic cross-talk in PCOS pathophysiology [7].

A comprehensive assessment of serum vitamin D and salivary cortisol levels suggests a method to better consider the level of endocrine dysregulation in PCOS and for predicting disease progression and therapeutic response. Such information, personalised treatment methods, and mechanisms primarily determine and heterogeneity of PCOS clinical phenotypes; biomarkers may facilitate the earlier identification of women at risk for metabolic difficulties [8]. Developing investigations will continue to explore the potential modulatory roles of these hormones to improve results for women affected by PCOS through targeted involvement, prevention of metabolic and optimisation of reproductive health.

MATERIALS AND METHODS

Research design- This is a prospective study aimed at determining vitamin D levels in serum and the salivary cortisol enzyme, which acts as a modulator in the dysregulation of women suffering from PCOS. The study was conducted in the Prasad Institute of Medical Sciences, situated in Lucknow (UP). The study was conducted for a period of one year, from January 2024 to December 2024. All the patients aged 18 to 35 years were diagnosed with PCOS, and some with menstrual complications were included in the study. All participants were selected based on written and verbal consent from the patient and their family, and the study was conducted in accordance with proper ethical norms, as approved by the committee. Saliva samples were collected for further assessment of the study.

Inclusion criteria

- Patients aged 18 to 35 years are considered for the study.
- The patients who are diagnosed with PCOS are included in the study.
- Proper written and verbal consent and approval is necessary for the study.

Exclusion criteria

- Mainly pregnant women or those who are in the lactation period are not allowed for the study.
- The disabilities associated are excluded.
- Any diagnosed Tumors, prolactinoma, CAH, Cushing's syndrome are not considered for the study.
- Any pre-history of cancer or medications is not allowed.
- Severe calorie intake or malnutrition is not permitted for the study.

Subject Selection

Case group or Group A- The Rotterdam criteria were used for the confirmation of PCOS in the study participants. There were 2 or 3 criteria for the diagnosis, the first one is the oligomenorrheic, which is ≥35 days and the > 3months of amenorrheic intermenstrual interval, the second one is the use of the ultrasound for the PCOM, where the ovarian volume should be >10cm

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and the third one is the clinical hyperandrogenism. Apart from these, the intellectual or the presence of disabilities, or the case of pregnancy or a woman in the period of lactation, was not considered for the study. Other etiologies, like tumors or prolactinoma, along with congenital adrenal hyperplasia (CAH) and the Cushing disorder, which can trigger the PCOS condition, are also not allowed for the study. Patients with a pre-history of cancer and its associated medication or a history of severe calorie intake were also not considered for the study.

Control group- There is another group, designated as Group B, which is referred to as the control group. This group consists of healthy women with no menstrual abnormalities, no clinical evidence of hyperandrogenism, and a normal endocrine profile, without any diagnostic evidence of PCOS. These women were considered for this group to facilitate better comparative outcomes.

Evaluation of Anthropometric Indices- The height of barefoot individuals was measured in centimetres using an ultrasonic-based digital stadiometer (model: EasyCare EC1800, India). Also, the BMI (kg/m^2) was measured. The fat content of the body was measured in percentages, and the muscle mass of the skeletal portion of the whole body was measured in percentages, both of which were observed using bioelectrical impedance evaluation with the help of a body composition monitor (model: OMRON-HBF-375, Karada Scan, Kyoto, Japan).

Biochemical Assay and Collection of Saliva- The biochemical tests were conducted, and all participants were asked to rinse their mouths with fresh water and refrain from consuming food or beverages for at least one hour before saliva collection. The individuals were advised to seat in a straight and upright position and to tilt their heads in a slight forward direction for the collection of the saliva at the specific interval of time by the use of the passive drool procedure and were stored in the ice chilled cryovials of 1.80 ml and the saliva was collected by the salimetrics (Item No. 5016.02, Carlsbad, United States). The cryovials with the saliva were stored in the mini-cooler at a temperature of -20°C until future use.

ELISA of Free Cortisol and Sex-Hormone-Binding-Globulin (SHBG)- The quantification of the cortisol in the saliva was done by the use of an ELISA kit **(**LDN; Germany, SA E-6000). The normal range in the morning was 1.6 to 9.2(ng/ml). During the mid of the midday, the range was 0.9 to 6.9 and during the afternoon, the range was 0.6 to 3.6. The intra- and inter-assay coefficients of variation (CV%) revealed values of 4.1 to 7.1 and 4.2 to 9.1, respectively. The concentration of SHBG in the saliva was measured using a sandwich ELISA kit (MyBioSource, MBS2701743; San Diego, USA). The range for the deterioration was 31.2-2000 pg/ml, also the CV% is <10 and <12. The centrifugation of the saliva was performed for about 20 minutes at 4°C at 1000×g and the rest of the supernatants were stored for future use in the assay. Spectrophotometer measured the concentrations of the cortisol and the SHBG at the absorbance of 450nm wavelength [make: Bio-rad, Model No. iMark (Microplate) reader, SL No. 10095, California, United States.

Estimation of haematological parameters- The blood samples were collected by the puncture of the vein after vigorous fasting for a period of 12 hours during the day of the follicular stage, that is, on the second or the third day of the menstrual cycle. The concentration of the 25-hydroxy-vitamin D [25(OH)D] in blood was <20 (VDD), vitamin D insufficiency (VDI)] was in the range of 20 to <30 and vitamin D sufficiency (VDS) is ≥30, this all are assessed by the chemiluminescent microparticle immunoassay (CMIA) procedure and by the Alinityi (Abbott) system [assay CV (intra=3.66–6.56% and inter=4.19–7.01%)]. Additionally, the concentrations of estradiol, JH, and FISH, as well as the levels of the progesterone hormone, were measured in the blood samples collected from the patients. Additionally, the ratio of LH and FSH was calculated using the standard formula.

Statistical Analysis- Data were analyzed using SPSS v27. Shapiro–Wilk and Kolmogorov–Smirnov tests were used to assess data normality. An independent t-test was used to compare body composition parameters, and Pearson's correlation evaluated the relationships between 25(OH)D, anthropometric, and hormonal factors. Graphs and charts were prepared in Microsoft Excel 2007. A $p<0.05$ was considered statistically significant.

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RESULTS

Table 1 shows that most of the groups were deficient in vitamin D, with 52 out of 58 participants in group A and 20 out of 58 participants in group B deficient in vitamin D, while only a few individuals in each group had an adequate amount of vitamin D.

Table 1: The exposure level of the variants of vitamin D according to both groups

Vitamin D variants	Exposure level	Group A	Group B
VDD/VDI <30 ng/mL	Exposed	52	20
VDS ≥30 ng/mL	Not exposed	6	2

Table 2 demonstrates the distribution of obesity-related parameters across Group A and Group B. Visceral fat levels were lower in Group A (0.03) compared to Group B (0.19), suggesting greater central adiposity in the latter. Similarly, body fat percentage was marginally higher in Group B (0.15) than in Group A (0.07). Body mass index (BMI) values showed a slight difference, being lower in Group B (0.006) than in Group A (0.01). Interestingly, skeletal muscle percentage was higher in Group B (0.209) compared to Group A (0.06), indicating relatively better muscle mass preservation in Group B. Overall, Group B exhibited higher fat distribution as well as better skeletal muscle proportion.

Table 2: Different obesity parameters are distributed according to the groups

Obesity indices	Group A (n = 58)	Group B (n = 22)
Visceral fat	0.03	0.19
BMI (kg/m²)	0.01	0.006
Body fat (%)	0.07	0.15
Skeletal muscle whole body (%)	0.06	0.20

Table 3 compares vitamin D status between Group A and Group B. In Group A, the majority of participants (81%) had vitamin D deficiency (VDD), whereas this proportion was slightly lower in Group B (64%). Vitamin D insufficiency (VDI) was observed in 12% of Group A and 14% of Group B, showing nearly similar distribution. Notably, vitamin D sufficiency (VDS) was more frequent in Group B (22%) compared to Group A (7%). These findings suggest that Group B had a comparatively better vitamin D profile, with a higher proportion achieving sufficiency and fewer individuals showing deficiency.

Table 3: The level of vitamin D, the values of the VDD, VDI and VDS according to the group A and Group B

Vitamin D level	Group A (n = 58)	%	Group B (n = 22)	%
VDD (<20 ng/mL)	47	81%	14	64%
VDI (20–30 ng/mL)	7	12%	3	14%
VDS (≥30 ng/mL)	4	7%	5	22%

DISCUSSION

The roles of serum vitamin D and salivary cortisol as modulators of endocrine dysregulation in women with PCOS continue to be discovered, with increasing evidence of their impacts on reproductive, metabolic, and psychological health. Recent studies have compared these markers in women with PCOS versus controls and assessed the effects of supplementation and stress physiology on disease development and symptomatology [9].

Most studies advise an essential deficiency of vitamin D in women with PCOS, compared with healthy controls, and its association with ovulatory dysfunction, hyperandrogenism, and metabolic imbalances. For example, Akinola *et al.* found that PCOS subjects had decreased vitamin D levels and hormonal imbalances, including elevated testosterone and AMH [2]. Mohan *et al*. and others have demonstrated a reproducible association between low serum vitamin D levels and impaired folliculogenesis, dysregulation of AMH, and increased infertility [10]. The therapeutic potential of vitamin D supplementation was supported by Miao *et al*., who reported improved insulin resistance, decreased serum testosterone, and better lipid profiles in PCOS after supplementation, paralleling findings in other randomised controlled trials [11].

However, some meta-analyses question whether vitamin D supplementation translates to improved metabolic and hormonal outcomes in all patients. He *et al*. performed a meta-analysis, which concluded that while vitamin D deficiency correlated with PCOS clinical severity,

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supplementation did not always reverse metabolic or hormonal disruptions. This discrepancy may be attributed to individual differences, study duration, and baseline vitamin D status [5]. Another result is that the benefit of high-dose supplementation plateaus, indicating minimal added advantage in some studies. Compared to other studies, the remains that correcting hypovitaminosis D is beneficial in PCOS; until now, optimal dosing and population stratification need additional investigation [12].

Cortisol, a portion as a biomarker of HPA axis activation, has been identified as raised in multiple PCOS populations, dependent on physiological stress and possible chronic neuroendocrine disruption [13]. Benjamin et al.'s systematic review and meta-analysis of 41 studies exposed that pooled cortisol levels were higher in women with PCOS compared to healthy controls, with blood cortisol displaying the highest effect size. The impact of chronic stress and hormonal imbalance may contribute to altered reproductive physiology, insulin resistance, and metabolic syndrome in affected women [14]. In contrast, Dumesic et al. found no difference in serum cortisol or cortisone between PCOS women and controls, suggesting that individual variability and variable PCOS phenotypes may influence HPA axis results [15].

When assessing cortisol metabolism, especially the increased clearance and altered A-ring reduction pathways, other studies have revealed independent associations with adrenal hyperandrogenism. Some investigators even point out that altered cortisol metabolism may exist regardless of BMI, again emphasising PCOS as a heterogeneous disorder. The role of cortisol as a stress marker and its interaction with androgen production is increasingly recognised in the literature, with evidence suggesting that interventions aimed at reducing stress and improving stress may positively affect metabolic and reproductive outcomes [16].

Direct comparison of vitamin D and cortisol research in PCOS shows some different results. Both markers exhibit abnormalities in a majority of PCOS studies, implicating their roles in metabolic and reproductive dysfunction. Where studies separate in establishing therapeutic efficacy, while vitamin D supplementation dependably increases serum levels and correlates with improved metabolic markers in most but not all studies, the relationship with direct hormonal improvements remains less uniform [17]. Similarly, while cortisol indicates HPA axis hyperactivity in many PCOS subjects, not all studies show strong relationships to symptom severity or successful intervention.

A comparative study requires longer-term and larger-scale involvement. The duration of treatment, baseline deficiencies, genetic factors, and comorbidities all likely modify the response and overall disease outcomes [18]. Meta-analyses and systematic reviews agree that both markers, serum vitamin D and salivary/serum cortisol, serve as indicators of primary endocrine disturbance; however, they advise caution in the complete application of findings across diverse populations [19]. More personalised methods and prospective trials are recommended to optimise results in PCOS management.

CONCLUSIONS

The study concluded the prevalence of the vitamin D levels for both of the groups, where the rate of deficiency is high in case of the group A, which shows that 90% of the individuals are deficient while Group B has a small amount of people who are adequate in vitamin D, less deficiency and highly sufficient in vitamin D. The correlation analysis also revealed that the weak positive associations have been observed in case of the obesity and group B had strong positive relationship with the percentage of the body fat and the muscle mass, which suggests that the composition of the body can affect the vitamin D level. Thus, the findings provide the burden of the deficiency of the said vitamin and its association with different body parameters.

CONTRIBUTION OF AUTHORS

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