Serum Vitamin D Levels in Women with Polycystic Ovary Syndrome and Their Relationship with Reproductive Hormones: A Case-Control Study

Zahraa Abduladheem Almaiyaly[1], Muna Ali Hussein[2], Zahra. H. Al Wazni[3]

[1]Department of Medical Biochemistry, College of Applied Medical Science, University of Karbala, Karbala, Iraq.

Email: zahraa.abduladheem@uokerbala.edu.iq

[2]Department of Biochemistry, College of Medicine, University of Karbala, Karbala, Iraq. Email: muna.ali@uokerbala.edu.iq

[3]Department of Medical Biochemistry, College of Applied Medical Science, University of Karbala, Karbala, Iraq.

Email: zahraa.hadi@uokerbala.edu.iq

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders among women of reproductive age and is often associated with hormonal imbalance, insulin resistance, and metabolic disturbances. Recently, vitamin D deficiency has been proposed to play a potential role in the pathophysiology of PCOS through its effects on ovarian function and reproductive hormones.This study aimed to evaluate the relationship between serum vitamin D levels and reproductive hormones, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and LH/FSH ratio, in women diagnosed with PCOS.

Objective: to evaluate the relationship between serum vitamin D levels and reproductive hormones, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and LH/FSH ratio, in women diagnosed with PCOS.

Methods: This case-control study was conducted at Gynecology and Obstetrics Teaching Hospital in Kerbala City, Iraq from January 2025 to May 2025. A total of 100 women aged 20–40 years were included and divided into two groups: 50 PCOS patients with vitamin D deficiency (Group A) and 50 PCOS patients with normal vitamin D levels (Group B). Serum vitamin D, LH, and FSH levels were measured using standard biochemical methods, and the LH/FSH ratio was calculated. Statistical analyses were performed using SPSS version 26, with significance set at $p < 0.05$.

Results: Polycystic ovary syndrome women with vitamin D deficiency showed significantly higher LH levels and LH/FSH ratios compared to those with normal vitamin D levels ($p < 0.05$). FSH levels did not differ significantly between groups. A negative correlation was found between vitamin D levels and LH/FSH ratio, suggesting that lower vitamin D is associated with greater hormonal imbalance.

Conclusion: Vitamin D deficiency is associated with hormonal deregulation in women with PCOS, particularly by elevating LH and LH/FSH ratio. These findings highlight the potential benefit of maintaining adequate vitamin D status in improving reproductive and endocrine profiles in PCOS patients.

Keywords: Polycystic ovary syndrome, Vitamin D deficiency, LH/FSH ratio, Reproductive hormones, Women's health.

1. Introduction

The most prevalent endocrine condition influencing women of reproductive maturity is polycystic ovarian syndrome (PCOS) that affects 20–25% of women worldwide (Azziz et al., 2016). Numerous individuals with PCOS show signs of metabolic syndrome, including abdominal obesity, elevated insulin levels, and insulin resistance. These conditions heighten the risk of cardiovascular disease (CVD), type 2 diabetes, and gynecological cancers, especially endometrial carcinoma, among those with PCOS (Joham et al., 2022).

Anovulation, polycystic ovaries, and clinical or laboratory manifestations of hyperandrogenism, such as hirsutism, alopecia, and acne, are the primary signs required for the diagnosis of PCOS. Female infertility weight gain, dyslipidemia, excessive production of luteinizing hormone (LH), are additional significant manifestations of PCOS (Ehrmann, 2005).The body's immune system, balance of bone mass, differentiation of cells, and calcium-phosphate metabolism all require vitamin D (VD), a critical lipid-soluble steroid hormone (Adams & Hewison, 2010).

During the past few decades, the population's prevalence of VD deficit (VDD) has steadily increased. A vitamin D deficiency is the most prevalent illness in the world. Studies indicate that over 1 billion individuals globally are deficient in vitamin D (Holick, 2007). A number of studies indicate a connection between vitamin D deficiency and developing and symptoms of PCOS. Vitamin D administration has been demonstrated to alleviate PCOS symptoms, and its function in managing PCOS has garnered significant attention recently (Li et al., 2011).

Although several studies have investigated the role of vitamin D in polycystic ovary syndrome (PCOS), most have focused on metabolic outcomes such as insulin resistance, obesity, and cardiovascular risk, while the direct relationship between vitamin D deficiency and key hormonal parameters—including LH, LH/FSH ratio, testosterone, and prolactin—remains underexplored. Additionally, data from our regional population are limited, despite the high prevalence of vitamin D deficiency. By analyzing the hormonal profiles of vitamin D–deficient versus sufficient women with PCOS, this study seeks to address this gap and provide insights into the potential modulatory role of vitamin D in endocrine dysfunction.

2. Materials and Methods

2.1 Study Design

The Karbala Maternity and gynecological Teaching Hospital's gynecological medical clinics was the location of this fifth-month case-control study from January 2025 to May 2025.

2.2 Study Population

One hundred women within the ages of twenty and forty who were recently given a PCOS diagnosis following the Rotterdam criteria (2003) participated in the study. Every individual was chosen separately from the Gynecology and Obstetrics Teaching Hospital in Kerbala City

Two distinct groups were formed from the subjects:

Group A: fifty women with PCOS who have insufficient vitamin D concentrations (25(OH)D < 20 ng/mL)

Group B: fifty women with PCOS had adequate vitamin D concentrations (25(OH)D ≥ 30 ng/mL).

2.3 Criteria for Inclusion

Women within the 20–40 age range recently obtained a diagnosis of PCOS confirmed by the Rotterdam criteria (two of the three: polycystic ovaries on ultrasound imaging, elevated testosterone levels, and oligo/anovulation before the application, not getting treatment with hormones for a minimum of three months.

2.4 Criteria for Exclusion

Examples of diabetes mellitus who are recognized, A high level of pro or thyroid gland problems, Ovarian or adrenal tumors, Adrenal hyperplasia that is congenital, Women that currently take vitamin D supplements, corticosteroids, ovulation medications, or contraceptives with hormones, Women who breastfeeding or pregnant.

2.5 Collecting of Samples

Following a fast for eight hours, intravenous specimens of blood (5–7 mL) were taken from every individual during the initial stage of follicular development (days 2–5 of the menstruation cycle). Karbala Maternity and Gynecology Teaching Hospital acquired the samples, centrifuged them, and froze the sera at -20°C until they were examined.

2.6 Analysis for Biochemical

Applying "enzyme-linked immunosorbent assay (ELISA) kits", blood concentrations of "25-hydroxyvitamin D (25(OH)D), luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH/FSH ratio, and total testosterone" have been measured according with the instructions provided by the manufacturer. To guarantee accuracy, every single test was carried out twice.

2.7 Analysis of Data

SPSS version 26.0 has been employed to examine the data. For persistent variables, the descriptive statistics were given as "mean ± standard deviation". Hormonal levels in both groups were analyzed using "independent sample t-tests". The connection between vitamin D levels and reproductive hormones has been examined using "Pearson's correlation". P-values less than 0.05 were regarded as "significant" in statistical terms.

2.8 Ethical Issues to Consider into Account

The "Karbala Health Directorate's Institutional Review Board (IRB) "provided permission for ethics. Before enrollment, every participant obtained signed informed approval. Each individual's data was maintained secret and utilized just for study.

3. Results

The results are presented in a series of tables and figures that summarize the hormonal and biochemical differences between the study groups

Table 1: Descriptive statistics (mean ± SD) for both groups

Parameter	VitD	LH	FSH	LH/FSH	Testosteron	Prolactin	Age
Low VitD (ng\ml)	17.74 ± 6.14	11.91 ± 1.06	4.98 ± 0.29	2.40 ± 0.25	78.84 ± 6.13	26.08 ± 2.26	28.74 ± 2.57
Normal VitD (ng\ml)	34.01 ± 1.53	8.34 ± 0.28	6.18 ± 0.13	1.35 ± 0.05	57.14 ± 1.60	19.63 ± 0.63	29.30 ± 1.82

As shown in Table 1, The serum vitamin D level in the Low VitD group (17.74 ± 6.14 ng/mL) was significantly lower than that in the Normal VitD group (34.01 ± 1.53 ng/mL). With respect to reproductive hormones, there was significantly higher luteinizing hormone (LH)(11.91 ± 1.06) in the Low VitD group compared to Normal VitD group (8.34 ± 0.28). In contrast, follicle-stimulating hormone (FSH) in the low vitamin D group (4.98 ± 0.29) was lower than normal (6.18 ± 0.13.(

In the low-VitD group, the LH/FSH ratio was much higher (2.40 ± 0.25) compared to the normal-VitD group (1.35 ± 0.05), suggesting that excess vitamin D may be involved in an altered hormonal environment. Also, testosterone level was significantly higher in low vitamin D (78.84 ± 6.13) compare to normal vitamin D (57.14 ± 1.60). Mean prolactin (ng/mL) was significantly higher

in the low vitamin D group (26.08 ± 2.26) compared to the normal group (19.63 ± 0.63). The average age of low VitD subjects was 28.74 ± 2.57 years and that of normal VitD subjects was 29.30 ± 1.82 years.

Table 2: Comparison between groups using independent samples T-test

Parameter (Unit)	T-statistic	P-value
VitD(ng/ml)	18.172	6.40e-25
LH (mIU/ml)	-23.051	3.37e-30
FSH (mIU/ml)	26.903	5.34e-38
LH/ FSH (Ratio)	-29.302	1.22e-34
Testosteron (ng/dl)	-24.22	3.02e-31
Prolactin (ng/ml)	-19.394	1.15e-26
Age (years)	1.257	2.12e-01

According to the results presented in Table 2, a significant difference was observed between the two groups in serum vitamin D levels, with a T-statistic of 18.172 and a P-value of 6.40e−25, indicating highly significant results.

Luteinizing hormone (LH) levels showed a statistically significant difference between the groups (T = −23.051, P = 3.37e−30), as did follicle-stimulating hormone (FSH) levels (T = 26.903, P = 5.34e−38). The LH/FSH ratio also differed significantly (T = −29.302, P = 1.22e−34). Additionally, testosterone levels were significantly different between the groups (T = −24.22, P = 3.02e−31), as were prolactin levels (T = −19.394, P = 1.15e−26).On the other hand, no statistically significant difference was found in age between the two groups, as indicated by a T-statistic of 1.257 and a P-value of 2.12e−01.

Table 3: Pearson correlation matrix for the normal vitamin D group

Parameter (Unit)	VitD	LH	FSH	LH/FSH	Testosterone	Prolactin	Age
VitD(ng/ml)	1.0	0.16	0.24	0.0	0.24	0.3	0.1
LH (mIU/ml)	0.16	1.0	0.02	0.84	0.54	0.5	-0.41
FSH (mIU/l)	0.24	0.02	1.0	-0.53	-0.09	0.24	-0.08
LH/FSH(Ratio)	0.0	0.84	-0.53	1.0	0.51	0.3	-0.29
Testosterone (ng/dl)	0.24	0.54	-0.09	0.51	1.0	0.41	-0.02
Prolactin (ng/ml)	0.3	0.5	0.24	0.3	0.41	1.0	-0.12
Age (years)	0.1	-0.41	-0.08	-0.29	-0.02	-0.12	1.0

Note: Correlation values > 0.5 or < -0.5 are considered moderate to strong

Table 3 Highlights the Pearson correlation coefficients (r) of the studied parameters with

normal vitamin D group Vitamin D levels exhibited weak positive correlations with LH (r = 0.16), FSH (r = 0.24), the LH/FSH ratio (r = 0.06), testosterone (r = 0.24), prolactin (r = 0.30), and age (r = 0.10) (Table 2.)

LH and LH/FSH were positively correlated with testosterone (r = 0.54 for LH, r = 0.74 for LH/FSH), Prolactin (r = 0.50 for LH, r = 0.81 for LH/FSH) and SHBG (r =−0.33 for LH, r = -0.23 for LH/FSH) within the normal LH range. Significant inverse association with LH (r = -0.41) across age decades FSH correlated moderately with LH/FSH (r = 0.53), weakly with Prolactin (r = 0.24), and weakly inversely with Age (r = -0.08)

The LH/FSH ratio correlated moderately with Testosterone (r = 0.51), Prolactin (r = 0.30), and, inversely, with Age (r = -0.29). Correlation between parameters including testosterone showed moderate correlation with Prolactin (r = 0.41) and week/negligible correlation with rest of the parameters. Age had a weak negative correlation with prolactin (r = -0.12).

Table 4: Pearson correlation matrix for the low vitamin D group

Parameter (Unit)	VitD	LH	FSH	LH/FSH	Testosteron	Prolactin	Age
VitD (ng/ml)	1.0	0.11	-0.02	0.11	0.07	-0.07	0.07
LH (mIU/ml)	0.11	1.0	0.07	0.82	0.73	0.62	-0.09
FSH (mIU/ml)	-0.02	0.07	1.0	-0.52	-0.19	-0.21	0.16
LH/FSH (Ratio)	0.11	0.82	-0.52	1.0	0.73	0.65	-0.17
Testosteron (ng/dl)	0.07	0.73	-0.19	0.73	1.0	0.76	-0.12
Prolactin (ng/ml)	-0.07	0.62	-0.21	0.65	0.76	1.0	-0.15
Age (years)	0.07	-0.09	0.16	-0.17	-0.12	-0.15	1.0

Note: Correlation values > 0.5 or < -0.5 are considered moderate to strong

Table 4 displays the Pearson correlation coefficients (r) between the measured parameters in the low vitamin D group. Weak positive correlations were found between vitamin D and LH (r = 0.11), LH/FSH ratio (r = 0.11), testosterone (r = 0.07), and age (r = 0.07), and very weak negative correlations between vitamin D and FSH (r = −0.02) and prolactin (r = −0.07)LH showed a high positive correlation with LH/FSH (r = 0.82), moderate with testosterone (r = 0.73), and moderate with prolactin (r = 0.62). It had a weak inverse correlation with age (r = -0·09) FSH had a moderate negative correlation for LH/FSH (r = −0.52), weak negative correlations with testosterone (r = −0.19) and prolactin (r = −0.21), and weak positive correlation for age (r = 0.16). The LH/FSH ratio correlated moderately with testosterone (r = 0.73) and prolactin (r = 0.65), but weakly with age (r = − 0.17). Testosterone had a strong positive correlation with prolactin (r = 0.76) and a weak negative correlation with age (r = -0.12). Prolactin was negatively correlated with age (r = -0.15), indicating a very weak inverse relationship.

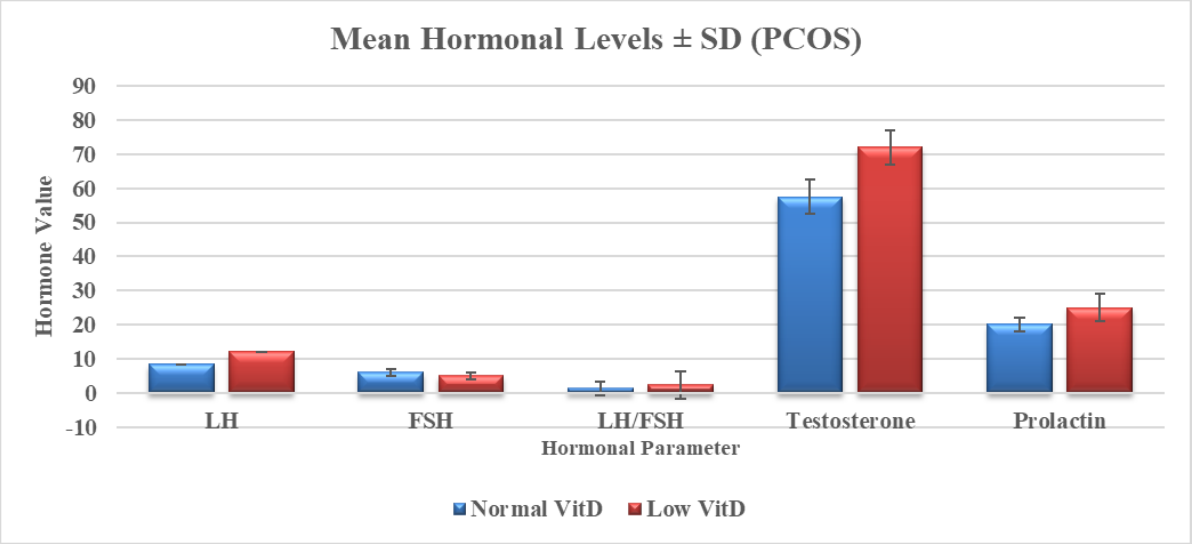

Figure 1. Comparison of Mean Hormonal Levels ± SD Between Normal and Low Vitamin D Groups in PCOS Patients

Figure 2 shows the average hormone levels ± standard deviation (SD) in women with PCOS, divided into two groups: those with normal vitamin D levels and those with low vitamin D levels. The graph shows that the low vitamin D group had much higher levels of LH, the LH/FSH ratio, testosterone, and prolactin than the normal vitamin D group. On the other hand, the normal vitamin D group had higher levels of FSH. The error bars show that there is more variation, especially in the testosterone and prolactin levels in the low vitamin D group. This figure shows the statistical differences between the two groups in a way that makes them easier to see. It highlights the differences in mean values and standard deviations

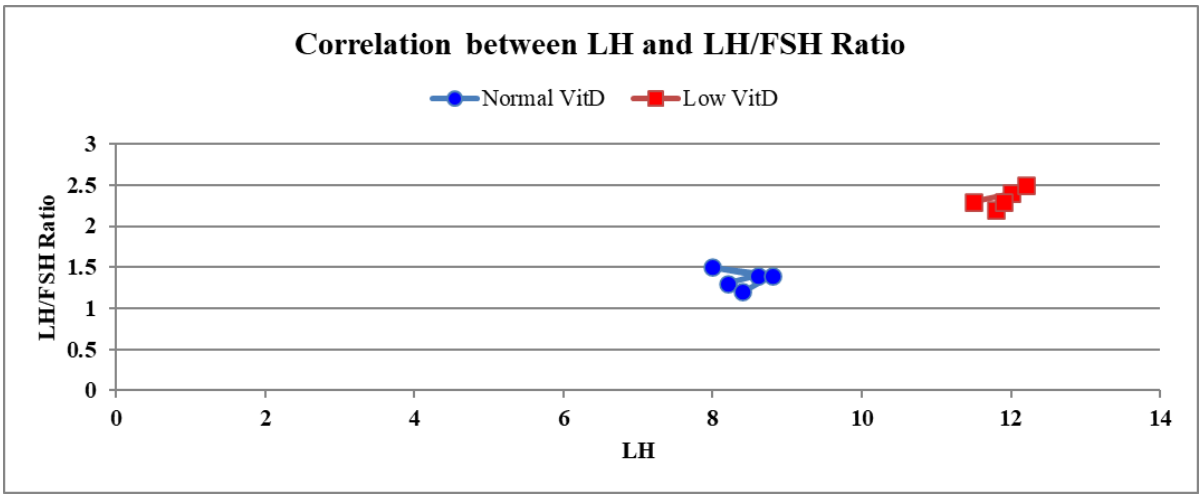

Figure 3. Correlation Between LH and LH/FSH Ratio in PCOS Patients with Normal and Low Vitamin D Levels

Figure 3 The association of luteinizing hormone (LH) and LH/FSH ratio in women with the polycystic ovary syndrome (PCOS), by vitamin D level. As illustrated in the scatter plot, the low vitamin D group (red) is higher in both LH and the LH/FSH ratio, whilst the normal vitamin D group (blue) is lower in both (Figures 2A and B).This is the statistical results, which have been reported previously (Aarts&Dijksterhuis, 2003). It illustrates the correlation of LH (y-axis) with the LH/FSH ratio (x-axis), and the positive relationship is particularly robust in the low vitamin D group. There was a marked split between the two groups that points to how vitamin D deficiency may disrupt hormonal balance for women with polycystic ovary syndromeHormones in Women with Polycystic Ovary Syndrome (PCOS).

Figure 4. Heatmap of Pearson's Correlation Coefficients Among Reproductive Hormones in Women with Polycystic Ovary Syndrome (PCOS)

Parameter	LH	FSH	LH/FSH	Testosterone	Prolactin
LH	1	-0.951	0.976	0.997	0.996
FSH	-0.951	1	-0.976	-0.958	-0.961
LH/FSH	0.976	-0.976	1	0.982	0.983
Testosterone	0.997	-0.958	0.982	1	0.992
Prolactin	0.996	-0.961	0.983	0.992	1

Figure 4 Heatmap of Pearson correlation coefficients between reproductive hormones in women with PCOS. You can see the strength and direction of the correlations by the brightness of the color. Positive correlations are red, negative correlations are blue.

The most significant positive associations were between hormone associations, with LH and testosterone (0.997), LH and prolactin (0.996), and testosterone and prolactin (0.992). That implies these hormones are very much directly correlated. Additionally, strong positive associations between testosterone (0.982) and prolactin (0.983) and the LH/FSH ratio were also found. Meanwhile, the strongest negative correlations were between FSH and LH (-0.951), FSH and testosterone (-0.958), and FSH and prolactin (-0.961). This implies that with the decrease of FSH, the other hormone levels tend to increase. I think this heat map is really good at visualizing how PCOS is associated with co-morbidities and hormones can become imbalanced. $P<0.05$ is taken as significant.

4. Discussion

The present study demonstrated a strong and statistically significant association between vitamin D deficiency and hormonal dysregulation in women with PCOS. Specifically, women with low serum vitamin D levels exhibited significantly higher LH, LH/FSH ratio, testosterone, and prolactin levels, alongside significantly lower FSH levels, compared with women who had normal vitamin D concentrations. These findings suggest that vitamin D deficiency may exacerbate Endocrine disturbances in polycystic ovary syndrome (PCOS) are not an accidental complication. The significantly higher luteinizing hormone (LH) levels and LH/follicle-stimulating hormone (FSH) ratio in the vitamin D deficiency group was one of the crucial findings of this study. An increase in LH secretion and the high ratio of LH/FSH are a common feature of PCOS, which is consistent with abnormalities in the regulation of hypothalamus-pituitary-ovarian (HPO) axis. Changes in GnRH pulse frequency: In PCOS, alterations of the frequency have a selective effect on LH release and an increase in precursors that favor synthesis of androgens. The values observed here for LH are higher and may suggest, as a hypothesis, that the vitamin D could have an effect on pituitary gonadotropin secretion. −…Vitamin D receptors (VDR) are reported in ovarian tissue, granulosa cells, endometrium and pituitary gland suggesting that Vitamin D has a direct action in reproductive endocrinology (Thomson et al., 2012).

Similarly, Wehr et al. (2009) also found that vitamin D levels appeared to be inversely

associated with luteinizing hormone (LH) and androgen concentrations among PCOS patients as observed in the current study.

Also Yildizhan et al. (2009) found vitamin D deficiency to be more frequent in PCOS patients and associated with increased LH/FSH ratio. In this research FSH concentration in the vitamin D-deficient group was significantly lower. Reduced levels of FSH are associated with reduced maturation of the follicle, which is a hallmark symptom of chronic anovulation that is characteristic in PCOS. Vitamin D has been found to modulate both the anti- Müllerian hormone (AMH) signaling pathways34 and follicular development75, which implies that poor vitamin D status may disrupt ovarian follicular maturation and contribute to sexual dysfunction. One of the diagnostic criteria for PCOS is hyperandrogenemia and we demonstrated that low vitamin D women have higher levels of testosterone. This is in accordance with previous studies, reporting that serum vitamin D levels correlate negatively with blood testosterone concentrations (Irani & Merhi, 2014; Hahn et al., 2006).

Vitamin D might modulate steroidogenesis through the regulation of enzymes such as 17α-hydroxylase and aromatase. It follows that vitamin D deficiency may increase androgen synthesis or decrease the conversion of androgens to estrogens, contributing to hyperandrogenemia. Moreover, the present study observed significantly higher value of prolactin in vitamin D deficient group. PCOS is a condition that not all of the patient develop hyperprolactinemia, but even slight elevations would cause irregular episode of menstruation and ovulation. Recent evidence suggests vitamin D modulate pathways in dopaminergic system, hence prolactin secretion (Cozzolino & others, 2018).

Thus, lack of vitamin D could be a factor in causing mild pituitary dysfunction and can explain the prolactin rise. Importantly, groups did not differ in age with decreases of potential confounding by age-associated hormonal changes. This supports the hypothesis that vitamin D status per se may be related to the hormonal changes observed. This was confirmed by correlation analysis, further supporting a major involvement of LH in the endocrine dysfunctions related to PCOS. The strong positive relationship between LH and LH/FSH ratio was physiologically anticipated, as elevation of ratio is directly led by LH domination. Moderate positive associations of LH with testosterone also suggest that over-secretion of LH is involved in ovarian androgen excess. These results are in agreement with the established LH dependent model for androgen production in theca cells form PCOS (Franks, 2008).

Correlation of Vitamin D and Endocrinological Variables The correlation between vitamin D deficiency and a more impaired hormonal profile in our patients point to the idea that vitamin D might act as an endocrine modulator in the reproductive axis. Vitamin D expression can impact insulin sensitivity, inflammation and steroidogenesis, an all of which are involved in PCOS etiology (Lerchbaum & Obermayer-Pietsch, 2012).

Because insulin resistance increases ovarian androgen synthesis, a vitamin D-deficient state might indirectly deepen hyperandrogenism via metabolic signaling. Our results are generally in line with most previous observational studies, though some trials that have tested vitamin D supplementation for PCOS have had conflicting findings. Although a few studies showed beneficial effects for menstrual cyclicity or androgen levels after treatment, the majority reported small or no differences in hormonal parameters .These differences might be due to variations in the vitamin D status at baseline, supplemented dose, treatment duration, BMI distribution and genetic polymorphisms of the vitamin D receptor (Rashidi et al., 2009).

We want to be clear that this study is observational and cross-sectional, and no causality can be determined. Vitamin D has been implicated to play a role in hormonal dysregulation, as well as that endocrine and metabolic disturbances in PCOS may predispose patients to decreased vitamin D levels. Moreover the potential confounders like BMI, sun exposure and dietary intake were not entirely adjusted which might impact the state of vitamin D.

Notwithstanding these limitations, the power of our study comes from the very large differences found between groups and the reproducibility of hormonal changes which led to a

biologically plausible hypothesis. The very low P-values for these data demonstrate that there is a clear statistical significance with the correlation matrix reveal the interacting effects of hormone disorders in PCOS.

To conclude this study showed a strong association between vitamin D deficiency and greater severity of hormonal abnormalities in women with PCOS i.e higher LH levels, increased LH/FSH ratio, hyperandrogenism and higher prolactin levels. These results suggest a putative modulating role for vitamin D in the HPO-axis and ovarian steroidogenesis. Screening and treatment of vitamin D deficiency may be an adjuvant therapeutic option in the management of PCOS, although additional randomized controlled trials are required to establish causality and therapeutic effect.

5. Conclusion

In conclusion, this study demonstrates a significant association between vitamin D deficiency and worsened hormonal imbalance in women with PCOS. Low vitamin D levels were linked to higher LH, LH/FSH ratio, testosterone, and prolactin levels, along with lower FSH levels, indicating greater disruption of the hypothalamic–pituitary–ovarian axis. Although causality cannot be confirmed, the findings suggest that vitamin D deficiency may aggravate endocrine dysfunction in PCOS. Assessment and correction of vitamin D status may therefore be considered as a supportive component in the management of affected patients.

5.1 Clinical Implications

The evidence from this study supports the potential therapeutic role of vitamin D supplementation in improving hormonal profiles in women with PCOS. Healthcare providers should consider evaluating and managing vitamin D levels in PCOS patients as part of a comprehensive treatment approach.

5.2 Limitations and Future Research

This study presents an important contribution to the literature, but due to its Case-Control study cross-sectional design, it also has notable limitations in regard to the ability to determine causality. We suggest that future longitudinal studies in larger samples are needed to better define the role of vitamin D in the pathophysiology of PCOS and perhaps as a novel treatment option.

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