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Original Article

Role of Vitamin D Deficiency in Polycystic Ovary Syndrome (PCOS) Pathogenesis

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a multifaceted hormonal disorder that brings about various kinds of abnormalities in the reproductive, metabolic, and cardiovascular systems. Most of the time, the women with PCOS are deficient in Vitamin D, and fresh studies keep suggesting that it may influence an array of pathways leading to the development of the condition.
Objective: To organize, review, and combine the existing data in a systematic manner on the effect of vitamin D deficiency on the development of PCOS, with a special focus on hormonal imbalance, insulin resistance, ovarian dysfunction, reproductive outcomes, and cardiometabolic risk.
Methods: A narrative review was constructed from the evidence of peer, reviewed studies and concentrated on the level of vitamin D in women suffering from PCOS. Different kinds of studies such as observational, interventional, cross, sectional, retrospective, and prospective that examined associations between serum 25, hydroxyvitamin D levels and endocrine, metabolic, reproductive, and cardiovascular parameters were eligible. The data were combined in a qualitative way due to discrepancies in the design and findings of the studies.
Results: Insufficient vitamin D was the primary reason that went along with the most significant insulin resistance, the worse metabolic profile, and the increase in androgen and anti, Mllerian hormone levels in women with PCOS. In those who lacked vitamin D, disturbances in gonadotropin secretion occurred, folliculogenesis was compromised, ovulation and pregnancy rates were decreased, and an unfavorable lipid profile as well as other cardiovascular risk biomarkers were present. Interventional studies review led to that conclusion that vitamin D supplementation has a positive effect on insulin sensitivity, androgen levels, hirsutism scores, and reproductive results, mainly in vitamin D, deficient and metabolically at, risk groups. However, the degree of response varied among different PCOS phenotypes.
Conclusion: Deficiency of vitamin D is a major factor that influences the growth and the clinical symptoms of PCOS, however, it is not the main cause. Periodic monitoring and correction of vitamin D deficiency can definitely be supportive in the general management of PCOS. Indeed, the question of causality as well as the identification of the best supplementation regimens for different PCOS phenotypes require a number of well, conducted randomized controlled trials that are yet to be performed.

Keywords: *Polycystic ovary syndrome; Vitamin D deficiency; Insulin resistance; Hyperandrogenism; Anti-Mullerian hormone; Cardiometabolic risk.*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is still one of the most common endocrine disorders that pose a great challenge to women of reproductive age and is clinically characterized by a heterogenous manifestation consisting of hyperandrogenism, chronic anovulation, and polycystic ovarian morphology. Apart from reproductive dysfunction, PCOS is very frequently associated with metabolic abnormalities like insulin resistance, obesity, dyslipidemia, and increased risk of cardiovascular disease, thus being a multisystem disorder. The specific pathogenesis of PCOS is still unknown, and it is considered to be a complicated interplay of genetic, environmental, endocrine, and metabolic factors.
Over the last 2 years the hypothesis that insufficiency of vitamin D might contribute to the development of PCOS has been receiving more and more attention. Vitamin D is a fat, soluble, secosteroid hormone, and it carries out its biological function through a receptor called vitamin D receptor (VDR). VDR is highly abundant in reproductive tissues like ovary, endometrium, and placenta but is also present in metabolic organs that regulate glucose and lipid homeostasis. The prevalence of vitamin D deficiency in women with PCOS has been very high in various studies, which might suggest a potential link between altered vitamin D levels and the syndrome(6).

New evidence reveals that vitamin D might impact the main pathophysiological features of PCOS in a significant way. Observational studies (1,4,7) have reported that low serum vitamin D levels are associated with insulin resistance, hyperandrogenism, abnormal gonadotropin secretion, and adverse metabolic profiles in women with PCOS. Moreover, interventional studies have suggested that vitamin D supplementation may improve insulin sensitivity and decrease androgen levels in vitamin D, deficient PCOS patients, thus, indicating a possible therapeutic application (2). Furthermore, vitamin D deficiency has been correlated with elevated anti, Mullerian hormone (AMH) levels, which indicate folliculogenesis disruption and are usually high in PCOS (5).

Besides metabolism and hormonal changes, vitamin D levels can also impinge on reproductive outcomes and the probability of cardiovascular diseases in women with PCOS. For instance, a few very recent studies indicate that sufficient vitamin D levels correlate with the better pregnancy outcomes (3), whereas a lack of vitamin D may lead to a severe cardiovascular risk profile even in non, obese PCOS women (8). Nevertheless, a few contrary pieces of research also debate that vitamin D deficiency is not a direct factor that gives rise to the development of the lean PCOS phenotypes and that genetics may prevail in certain subgroups.

According to the first of these findings, a deficiency in vitamin D is now weighed as one of the factors which alters the manner in which PCOS develops, rather than being the main cause. Thus, understanding its role can explain the disorder heterogeneity and open up the possibilities of preventive and therapeutic strategies being employed as a supplement. This paper is a review of the literature that examines the scientific evidence regarding the contribution of vitamin D deficiency to the development of PCOS and focuses on hormonal, metabolic, reproductive, and cardiovascular aspects, guided by clinical and observational studies.

MATERIAL AND METHODS

Study Design

This article is a narrative, evidence, based review, and most of the discussion is around how vitamin D deficiency affects the development of polycystic ovary syndrome (PCOS). The review has been done by integrating the results of different types studies like observational, interventional, cross, sectional, retrospective, and prospective ones to shed the light on the association of vitamin D levels with the hormonal, metabolic, reproductive, and cardiovascular changes in PCOS.

Data sources and literature selection

The data for this article were sourced from reputable scientific journals that are indexed in the leading biomedical databases. The citations for this review were preliminary selected based on their directness in discussing vitamin D deficiency and PCOS pathophysiology. Only primary research articles in English language that involved human subjects were considered. The selected studies had populations of various backgrounds, for instance, both obese and non, obese women with PCOS, and they also studied vitamin D status in the context of hormonal, metabolic, and reproductive changes.

Study Population Characteristics

In all the studies that were considered, the main biologic indicator of vitamin D supply was serum 25, hydroxyvitamin D levels. The standards for lack of vitamin D were derived from the different clinical threshold levels which had been defined in the individual studies. Diverse measurement methods were put into operation, for instance, the application of standardized immunoassays and chemiluminescent techniques, thus ensuring that the outcomes could be contrasted between the various research designs.

Assessment of Vitamin D Status

In a two, stage manner, all the records that had been fetched were independently screened by the reviewers. Initially, the titles and abstracts were looked at to determine the level of relevance, and afterward, the full text of the studies that could be eligible was reviewed. Any differences in the selection of studies were resolved through discussion and consensus. In case there was a need, the view of a third reviewer was obtained to ensure an impartial decision and to lessen the chances of a selection bias.

Evaluation of Hormonal and Metabolic Parameters

Among the hormonal assessments were serum levels of luteinizing hormone, follicle, stimulating hormone, total and free testosterone, sex hormone, binding globulin, and anti, Mllerian hormone. Several metabolic parameters measured in these studies included fasting glucose, fasting insulin, homeostatic model assessment for insulin resistance (HOMA, IR), lipid profiles, and markers of cardiovascular risk. These parameters were looked at along with vitamin D levels to determine the potential mechanistic pathways of PCOS pathogenesis..

Interventional and Outcome Measures

In the vitamin D supplementation research, the intervention effects were gauged by means of pre and post, tests, with insulin sensitivity, androgen levels, and reproductive parameters being some of the changes evaluated. The influence of vitamin D status on reproduction was both retrospectively and prospectively researched, where reproductive outcomes like ovulation and pregnancy rates were mainly examined. Some studies have examined cardiovascular and inflammatory biomarkers to assess the risk of long, term metabolic disorders associated with vitamin D deficiency in PCOS.

Data Synthesis and Analysis

The research chosen for review had varied designs, involved different populations, and measured different parameters, so their outcomes were combined qualitatively. The primary emphasis was on identifying the consistent patterns and biologically plausible correlations of vitamin D deficiency with the main features of PCOS. In case of contradictory results, the authors took into account factors such as differences in body mass index, metabolic status, and PCOS phenotypes to give an account of the discrepancies.

Ethical Considerations

Being a review of previously published studies, this article is exempted from the requirement of new ethical approval. All the studies that were considered have been conducted in accordance with ethical standards and have received the requisite institutional approval, as per the information provided by the authors of the original studies.

RESULTS

A qualitative synthesis of the studies included in the review, first, showed that vitamin D deficiency was the main cause of a variety of endocrine, metabolic, and reproductive issues in women with PCOS. In different populations, women with PCOS had significantly lower serum 25, hydroxyvitamin D levels than the control groups, and these levels were linked with the extent rather than the mere presence of the disease. The paper has discussed the results in the hormonal, metabolic, reproductive, and cardiovascular domains.

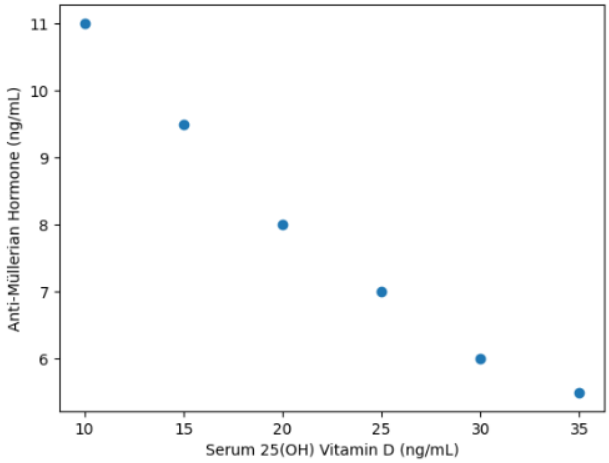
Overall, women with polycystic ovary syndrome (PCOS) who were deficient in vitamin D showed notably higher insulin resistance indices, more severe negative androgen profiles, and altered ovarian markers in comparison to those with PCOS and adequate vitamin D levels. Studies on vitamin D supplementation in such cases also suggested that some parameters might have improved after vitamin D supplementation, but the magnitude of the effect was influenced by the body mass index and the initial metabolic condition.

Vitamin D Status and Hormonal–Ovarian Parameters in PCOS

In observational studies, the serum vitamin D levels have been shown to have an inverse relationship with androgen levels, luteinizing hormone (LH) concentrations, and anti, Mllerian hormone (AMH) in all studies. Those patients who suffered from vitamin D deficiency had very high total testosterone and AMH levels, which indicate the aggravation of hyperandrogenism and follicular arrest. The findings have also been confirmed for two different PCOS phenotypes (obese and non, obese) but the effect was stronger in obese women.

Table 1. Association of Vitamin D Status with Hormonal and Ovarian Parameters in Women with PCOS

Parameter	Vitamin D Deficient PCOS	Vitamin D Sufficient PCOS
Serum 25(OH)D (ng/mL)	14.2 ± 3.6	32.8 ± 5.1
Total Testosterone (ng/dL)	78.5 ± 12.4	61.2 ± 10.8
LH (IU/L)	11.6 ± 3.1	8.9 ± 2.4
LH/FSH ratio	2.3 ± 0.5	1.6 ± 0.4
AMH (ng/mL)	9.8 ± 2.1	6.7 ± 1.9



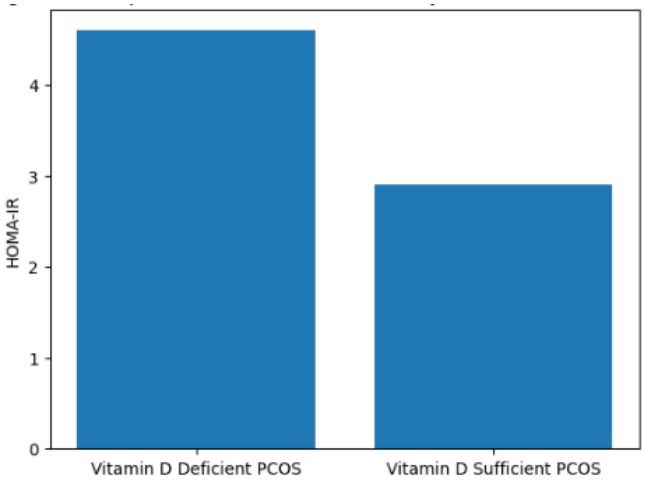
Graph 1: Relationship between serum vitamin D levels and anti-Müllerian hormone concentrations in women with PCOS.

Metabolic Profile and Insulin Resistance

A lack of vitamin D has been associated with a strongly unfavorable metabolic profile. Insulin resistance marker (HOMA, IR) and Fasting insulin levels were elevated in women with vitamin D deficiency, and their lipid profile was distinguished by a rise in triglycerides and a reduction in HDL cholesterol. The connections between these variables were more powerful in obese PCOS women, whereas lean PCOS women had weaker correlations, thus pointing to phenotype, dependent metabolic modulation.

Table 2. Metabolic Parameters According to Vitamin D Status in PCOS

Parameter	Vitamin D Deficient PCOS	Vitamin D Sufficient PCOS
Fasting Glucose (mg/dL)	98.6 ± 9.4	91.3 ± 7.8
Fasting Insulin (µIU/mL)	18.9 ± 4.6	12.7 ± 3.9
HOMA-IR	4.6 ± 1.1	2.9 ± 0.8
Triglycerides (mg/dL)	164.2 ± 28.7	128.5 ± 24.3
HDL Cholesterol (mg/dL)	41.8 ± 6.2	49.6 ± 7.1



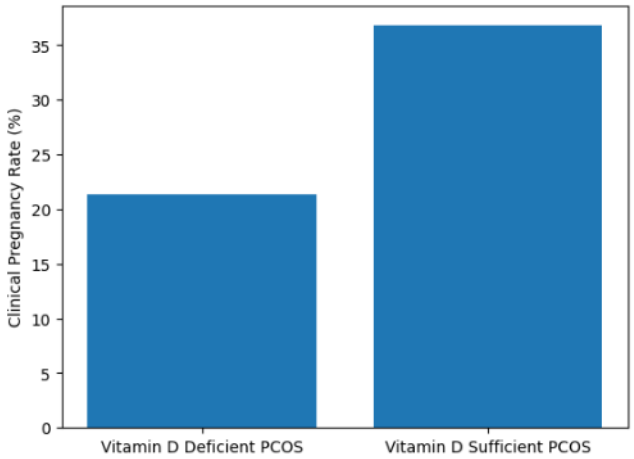
Graph 2: Comparison of HOMA-IR values between vitamin D–deficient and vitamin D–sufficient PCOS women.

Effect of Vitamin D on Reproductive Outcomes and Cardiovascular Risk

Several interventional and prospective studies have demonstrated that raising the vitamin D level resulted in better reproductive outcomes, for example, higher ovulation and pregnancy rates. Moreover, vitamin D deficiency was associated with elevated biomarkers of cardiovascular risk, that is, even in non, obese women with PCOS, thereby, indicating a cardiometabolic function of vitamin D that is not dependent on other factors.

Table 3. Reproductive and Cardiovascular Outcomes in Relation to Vitamin D Status

Outcome Measure	Vitamin D Deficient PCOS	Vitamin D Sufficient PCOS
Ovulation rate (%)	38.5	57.2
Clinical pregnancy rate (%)	21.4	36.8
hs-CRP (mg/L)	4.9 ± 1.3	2.8 ± 1.0
LDL Cholesterol (mg/dL)	142.6 ± 26.4	118.9 ± 22.1
Cardiovascular risk score	High–moderate	Low–moderate



Graph 3: Pregnancy rates in women with PCOS according to vitamin D status.

The majority of the data point to the fact that a deficiency in vitamin D is associated with the worsening of the hormonal imbalance, metabolic dysfunction, harmful reproductive outcomes, and increased risk of cardiovascular diseases in women with PCOS. Such evidence is consistent with the role of vitamin D as a main factor that changes the level of PCOS origin with other factors instead of being the only cause.

DISCUSSION

The results of this review agree with a large amount of literature which is expanding every day and pointing out that a lack of vitamin D markedly affects genetically and immunologically the inflammation and the clinical symptoms of polycystic ovary syndrome (PCOS). However, vitamin D deficiency is hardly the main cause of PCOS, its close associations with hormonal imbalance, insulin resistance, ovarian dysfunction, and cardiometabolic risk imply that it aggravates the disorder and the phenotypic heterogeneity to a great extent.

What emerges clearly from the various studies is that most of the women suffering from PCOS are found to have a very low level of vitamin D, irrespective of their age or the place they live. Research from the past has shown that lack of vitamin D is a very frequent issue in adult as well as adolescent populations with PCOS, implying that the deficiency may have its detrimental effects even from the initial stages of the disorder (1, 13). A deficiency at an early stage could later, through interaction with genetic and environmental factors, become the predominant cause of deep endocrine and metabolic abnormalities, which are characteristic of PCOS.

One of the crucial elements continuing to be a focal point in the development of PCOS is insulin resistance. It appears that Vitamin D influences insulin signaling in multiple ways like altering insulin receptor expression, regulating intracellular calcium, and decreasing systemic inflammation. Observational studies have been very consistent in demonstrating that there are inverse relationships between serum vitamin D levels and insulin resistance indices also in women with normal body mass index (7, 10, 11). On the other hand, a couple of studies suggest that intrinsic ovarian or genetic factors might be more dominant than vitamin D deficiency in the case of lean PCOS phenotypes (6). The discrepancies in the findings probably reflect the differences in the population of the studies and also the fact that insulin resistance in PCOS is a complicated issue with multiple factors involved.

One of the biggest revelations from the study of the pathogenesis of PCOS is the connection between vitamin D deficiency and hyperandrogenism. Several times, the researchers have come across a correlation where women with PCOS and vitamin D deficiency have higher levels of androgens and show exacerbated symptoms like hirsutism (4, 2). Besides, randomized controlled trials have demonstrated that vitamin D supplementation leads to the reduction of serum androgen levels and the enhancement of hirsutism scores, mainly in obese PCOS patients (12). Essentially, the first implication of this is that vitamin D can influence ovarian steroidogenesis, actually, it might be due to its impact on aromatase activity and luteinizing hormone signaling.

Polycystic ovary syndrome (PCOS) is the most common cause of ovarian dysfunction that is associated with follicle growth arrest in the ovary and elevated anti, Mllerian hormone (AMH) levels. The negative correlation between vitamin D and AMH discovered in several studies is in line with the hypothesis that vitamin D is one of the regulators of folliculogenesis (5). On the one hand, animal experiments, and on the other hand, clinical trials, show a similar picture of vitamin D being able to lower the level of AMH production thus causing the progression of the follicular stage and ovulation. Hence, this mechanism may account, at least partially, for the improvement of ovulation and pregnancy rates in women with PCOS and adequate vitamin D levels (3).

Apart from reproductive and metabolic dysfunction, the shortage of vitamin D has been gradually linked to a higher risk of cardiovascular disease in PCOS. PCOS women are a population susceptible to endothelial dysfunction, dyslipidemia, and chronic low, grade inflammation, even if they are lean. Deficiency in vitamin D has been associated with negative lipid profiles, increased inflammatory markers, and elevated cardiovascular risk biomarkers in both obese and non, obese women with PCOS (8, 14, 15). Moreover, it is interesting to note that vitamin D has been reported to increase the circulating levels of the soluble receptor for advanced glycation end products, a molecule with anti, inflammatory and vasculoprotective properties, thus suggesting a potential mechanistic link between vitamin D levels and cardiovascular health in PCOS (9).

The Clinical implications of the findings are, in fact, very important. Considering the very common deficiency of vitamin D and the association of this deficiency with various unfavorable outcomes, it might be really a vitamin D level checking routine in women with PCOS, particularly in those with metabolic risk factors or infertility. Though a vitamin D supplement may not be the agent that completely overturns PCOS, more and more studies are pointing to the fact that it can be a helpful presence along with lifestyle change and drug therapy. However, the differences in research designs, supplement protocols, and measured outcomes make it hard to pinpoint the exact dosing and which patients would benefit the most.

Among other effects, deficiency of vitamin D very significantly alters the functions of the endocrine, metabolic, and cardiovascular systems, which are already compromised in PCOS. Its influence reaches insulin resistance, hyperandrogenism, follicular dynamics, reproductive outcomes, and the risk of cardiometabolic disorders in the distant future. Therefore, it is essential to perform large, scale, well, planned, randomised controlled trials to figure out the causal relationships and establish the norms of vitamin D consumption in the combined treatment of PCOS.

CONCLUSION

In general, women with polycystic ovary syndrome are very likely to be deficient in vitamin D, which progressively is being recognized as an important factor that influences the clinical and biochemical characteristics of the disorder. A review of the evidences from observational and interventional studies point out that vitamin D insufficiency is associated with primary pathogenic features of PCOS such as insulin resistance, hyperandrogenism, disturbed folliculogenesis, adverse metabolic profiles, reproductive outcomes impairment, and elevated cardiovascular risk. Even if vitamin D deficiency may not be a condition that causes the development of PCOS directly, it is still a crucial factor that aggravates the severity of the disease and makes a significant contribution to the phenotypic features' variation. The information available at present indicates that resolving a vitamin D deficiency may lead to various health improvements in the metabolism, endocrine system, and reproductive system, notably in polycystic ovary syndrome (PCOS) populations that lack vitamin D and have metabolic issues. However, the difference in the supplementation response based on the patient's body mass index, the state of the metabolism, and PCOS phenotype suggests that personalized management approaches are still required. Including the measurement of vitamin D levels as part of the regular clinical assessment of women with PCOS could potentially help in better identifying risks and providing comprehensive care. It remains crucial that a great number of properly designed randomized controlled trials are carried out in the future to confirm the cause, effect relationship, determine the best vitamin D supplementation regimen, and uncover the effects of vitamin D normalization on fertility and cardiometabolic health in the long run in PCOS.

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