Archives of the Balkan Medical Union
vol. 60, no. 3, pp. 365-372
September 2025
Copyright © 2025 Balkan Medical Union

ORIGINAL PAPER

VITAMIN D DEFICIENCY AND ITS ASSOCIATION WITH CLINICAL AND HORMONAL PROFILES OF ALGERIAN WOMEN SUFFERING FROM POLYCYSTIC OVARY SYNDROME

Amira MESSAOUD[1,2], Anfal BELKACEM[1,2], Siheme OUALI[1,2], Soumia ZEGGAI[2,3], Noria HARIR[1,2,2], Meriem Rabia ZAHZEH[1,2], Amine El mokhtar DRICI[1], Chahinez HADJ SLIMENE[4], Feryel SENOUSSAOUI[5], Amina BENMESSAOUD[1,2], Wassila Nesrina ELMEZOUAR[1,2], Khalida ZEMRI[1]

[1] Biology Department, Faculty of Natural and Life Sciences, Djillali Liabes University of Sidi Bel Abbes, Algeria
[2] Laboratory of Molecular Microbiology, Proteomics and Health, Djillali Liabes University of Sidi Bel Abbes, Algeria
[3] Biology Department, Faculty of Sciences M'hamed Bougara, University of Boumerdes, Algeria
[4] EL ASSRAR Center, Oran, Algeria
[5] Medically Assisted Procreation, AL ANIS Clinic, Oran, Algeria

Received 15th July 2025, Accepted 25th Aug 2025
https://doi.org/10.31688/ABMU.2025.60.3.06

Abstract

Introduction. Vitamin D deficiency and polycystic ovary syndrome (PCOS) are becoming increasingly common.
The objective of the study was to investigate the impact of vitamin D deficiency on PCOS and to evaluate its association with clinical manifestations and hormonal profiles of Algerian women with PCOS.
Material and methods. Three hundred and three PCOS patients were divided into three groups: vitamin D deficiency (<20 ng/mL), insufficiency (21-29 ng/mL) and sufficiency (≥ 30 ng/mL). Demographic,

Résumé

La carence en vitamine D et son association avec les profils cliniques et hormonaux des femmes algériennes souffrant du syndrome des ovaires polykystiques

Introduction. La carence en vitamine D et le syndrome des ovaires polykystiques (SOPK) sont de plus en plus fréquents.
L'objectif de l'étude était d'étudier l'impact de la carence en vitamine D sur le SOPK et d'évaluer son association avec les manifestations cliniques et les

✉ Address for correspondence: Noria HARIR
Biology Department, Faculty of Natural and Life Sciences, Laboratory of Molecular Microbiology, Proteomics and Health, Djillali Liabes University of Sidi Bel Abbes, Algeria
Address: PO. Box N°89 Sidi-Bel-Abbes 22000, Algeria
E-mail: harirnoria16@gmail.com; Phone: +213 662 378 918

clinical and hormonal parameters were compared between the three groups.

Results. In 67.9% of the patients, vitamin D deficiency correlated statistically significant with a high body mass index, hirsutism, acne, alopecia/hair loss ($p<0.0001$) and secondary infertility ($p=0.017$). Higher concentrations of total testosterone, luteinizing hormone (LH) and anti-Müllerian hormone (AMH) were noted ($p<0.0001$) and lower concentrations of progesterone were detected ($p<0.0001$). 25(OH)D3 serum concentrations were negatively correlated with total testosterone, LH, AMH ($p<0.0001$). However, a positive correlation was reported with follicle-stimulating hormone (FSH) and progesterone levels ($p=0.005$) and ($p<0.0001$) respectively.

Conclusions. The results of the study showed that low levels of 25(OH)D3 were associated with higher androgen levels in women with PCOS. Vitamin D deficiency should be considered as an additional risk factor in the development of PCOS.

Keywords: polycystic ovarian syndrome, vitamin D deficiency, androgen hormones.

List of abbreviations:
25-OH D3 = 25-hydroxyvitamin D3
AMH = anti-Müllerian hormone
BMI = body mass index
FSH = follicle-stimulating hormone
LH = luteinizing hormone
PCOS = polycystic ovary syndrome

profils hormonaux des femmes algériennes atteintes de SOPK.

Matériel et méthodes. Trois cent trois patientes atteintes de SOPK ont été réparties en trois groupes : carence en vitamine D (< 20 ng/mL), insuffisance (21-29 ng/mL) et suffisance (≥ 30 ng/mL). Les paramètres démographiques, cliniques et hormonaux ont été comparés entre les trois groupes.

Résultats. Chez 67,9 % des patientes, la carence en vitamine D était corrélée de manière statistiquement significative à un indice de masse corporelle élevé, à l'hirsutisme, à l'acné, à l'alopécie/chute de cheveux ($p < 0,0001$) et à l'infertilité secondaire ($p = 0,017$). Des concentrations plus élevées de testostérone totale, d'hormone lutéinisante (LH) et d'hormone anti-Müllerienne (AMH) ont été observées ($p < 0,0001$) et des concentrations plus faibles de progestérone ont été détectées ($p < 0,0001$). Les concentrations sériques de 25(OH) D3 étaient corrélées négativement à la testostérone totale, à la LH et à l'AMH ($p < 0,0001$). Cependant, une corrélation positive a été rapportée avec les taux d'hormone folliculo-stimulante (FSH) et de progestérone ($p = 0,005$) et ($p < 0,0001$) respectivement.

Conclusions. Les résultats de l'étude ont montré que de faibles taux de 25(OH)D3 étaient associés à des taux d'androgènes plus élevés chez les femmes atteintes du SOPK. Une carence en vitamine D doit être considérée comme un facteur de risque supplémentaire dans le développement du SOPK.

Mots-clés: syndrome des ovaires polykystiques, carence en vitamine D, hormones androgènes.

Introduction

Polycystic ovary syndrome (PCOS) is an endocrine disease affecting women of reproductive age, characterized by chronic anovulation, hyperandrogenism and abnormal development of ovarian follicles[1]. The etiology of PCOS remains uncertain. Evidence indicates a multifactorial origin, with expression shown in women with a hereditary predisposition[2]. Numerous patients with PCOS present characteristics of metabolic syndrome, including visceral obesity, hyperinsulinemia, and insulin resistance[3]. This occurs in conjunction with weight gain and an increase in waist circumference and is strongly correlated with hyperandrogenemia and ovarian dysfunction[4]. These factors, in association with increased adipose tissue and hyperandrogenemia, increase the risk of cardiovascular disease (CVD), type 2 diabetes, inflammation-related conditions and gynecological cancer, especially endometrial carcinoma, in PCOS patients[5-7].

Vitamin D is crucial for bone growth and development and significantly influences calcium metabolism. Vitamin D deficiency is also implicated in the etiology of various chronic medical conditions, including obesity, cardiovascular disease, type 2 diabetes, malignancy, autoimmunity, infectious disease, and psychological disorders[8]. Furthermore, obesity is a known risk factor for vitamin D deficiency[9]. Previous findings have shown a negative correlation between body mass index (BMI) and serum 25(OH) D3 concentrations in women with PCOS[10]. The vitamin D receptor (VDR) is expressed in several tissues beyond skeletal muscle. Consequently, it is linked to glucose metabolism, as well as the established calcium-bone metabolism, the cardiovascular system, cancers, and female reproductive organs including the ovaries, placenta, endometrium, and fallopian tubes[11]. The association of metabolic risk factors in women suffering from PCOS is well-documented; nevertheless, the influence of vitamin D deficiency on PCOS and metabolic syndrome remains ambiguous[12].

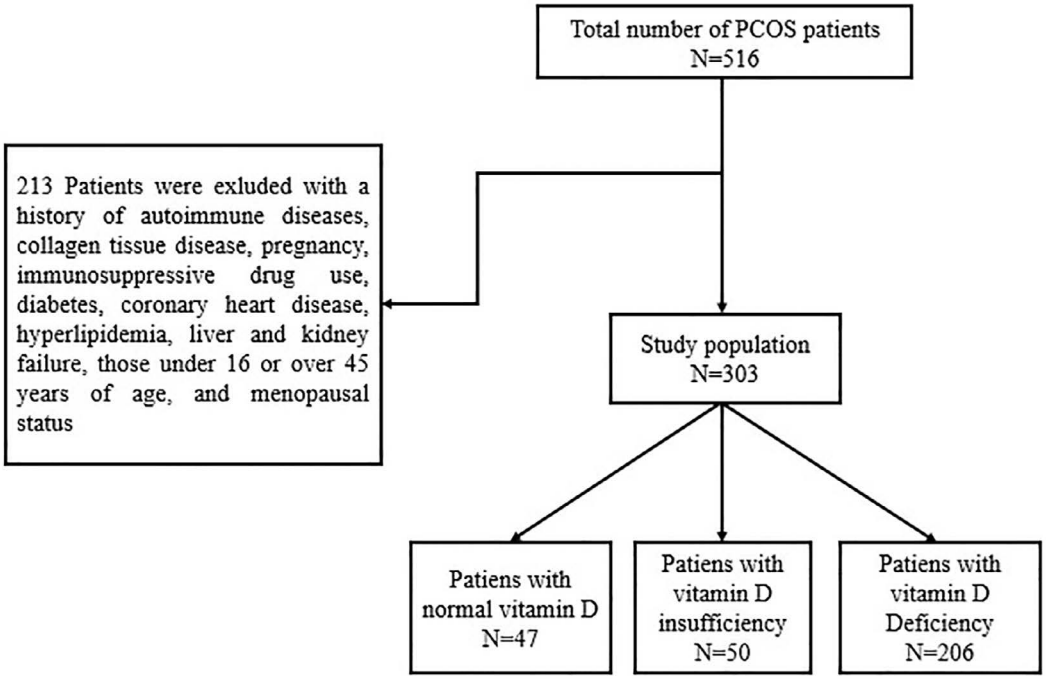


Figure 1. Flow chart of study population

The correlation between PCOS and vitamin D has been examined in several prior research. A deficit in vitamin D may increase the risk of PCOS[13]. Further research indicated that vitamin D supplementation improves blood pressure profiles and diminishes insulin resistance, as well as total testosterone and androstenedione levels in individuals with PCOS[14]. Obese women with PCOS have lower vitamin D levels than their non-obese counterparts with the condition[15]. Impaired glucose tolerance and heightened risk of type 2 diabetes development are associated with escalating insulin resistance in PCOS, and vitamin D insufficiency has been linked to an increased risk of type 2 diabetes[16].

The objective of the study was to investigate both the prevalence of vitamin D deficiency and its possible correlations with hormonal profile in women with PCOS from Western Algeria.

Material and methods

The study comprised 303 women of reproductive age, between 16 to 45 years, diagnosed with PCOS in the gynecological clinics EL-ASSRAR Center and Al-ANIS Clinic, Oran, Algeria, from April 2023 to October2024.

The patients were diagnosed according to European Society of Human Reproduction and Embryology/ American Society for Reproductive Medicine (Rotterdam) criteria 2004[17]. These included the presence of at least two ovulatory abnormalities, such as oligo-ovulation or anovulation, clinical manifestations of hyperandrogenism and/or biochemical hyperandrogenism, and polycystic ovarian morphology. All participants provided written informed consent and the study protocols were approved by the Ethics Committee of the University (N°16/D/FSNV/2022 dated 14/02/2023).

We studied patients demographic and clinical characteristics such as age, anthropometric measures (height, weight, BMI), hirsutism, alopecia/hair loss and acne; biochemical and hormonal profiles were recorded after overnight fasting and blood specimens were collected to measure of serum 25(OH)D levels (cut-off level 30-50 ng/mL), follicle stimulating hormone (FSH) (cut-off level 3.5-12 mlU/mL), luteinizing hormone (LH) (cut-off level 0.5-5.8 mlU/mL), progesterone (cut-off level 02-1.4 µg/mL), testosterone (cut-off level 0.15-0.6 ng/mL) and anti-Müllerien hormone (AMH) (cut-off level 1.2-5 ng/mL).

Vitamin D status was evaluated by 25(OH)D levels, which were divided into three groups: vitamin D sufficiency (≥ 30 ng/mL), insufficiency (21-29 ng/mL) or deficiency (<20 ng/mL).

The exclusion criteria were a history of autoimmune diseases, collagen tissue disease, pregnancy, immunosuppressive drug use, diabetes, coronary heart disease, hyperlipidemia, liver and kidney failure, patients under 16 or over 45 years of age, and menopausal status (Figure 1).

Table 1. Demographic and clinical data based on vitamin D status in PCOS patients

Characteristics N (%)	PCOS patients n=303			p-value
	Sufficiency n=47	Insufficiency n=50	Deficiency n=206	
Age	28.894±6.673	27.900±5.768	31.180±6.679	**0.002**
Residence				
Rural	15 (31.9%)	13 (26%)	67 (32.5%)	0.669
Urban	32 (68.1%)	37 (74%)	139 (67.5%)	
BMI	22.898±3.100	25.0.13±4.325	26.958±5.937	**<0.0001**
Normal	33 (70.2%)	21 (42%)	51 (24.8%)	
Underweight	00 (00%)	5 (10%)	55 (26.7%)	**<0.0001**
Overweight	11 (23.4%)	24 (48%)	84 (40.8%)	
Obese	3 (6.4%)	00 (00%)	16 (7.8%)	
Cycle regularity				
Regular	16 (34%)	00 (00%)	22 (11.7%)	
Absent	00 (00%)	00 (00%)	5 (2.4%)	**<0.0001**
Irregular	31 (66%)	50 (100%)	177 (85.9%)	
Primary infertility	24 (51.1%)	33 (66%)	110 (53.4%)	0.229
Secondary infertility	11 (23.4%)	6 (12%)	65 (31.6%)	**0.017**
Amenorrhea	00 (00%)	00 (00%)	6 (2.9%)	0.237
Oligo-amenorrhea	45 (95.7%)	50 (100%)	199 (96.6%)	0.381
Hirsutism	6 (12.8%)	35 (70%)	201 (97.6%)	**<0.0001**
Alopecia/hair loss				
Slight	8 (17%)	15 (30%)	58 (28.2%)	
Moderate	00 (00%)	9 (18%)	44 (21.4%)	**<0.0001**
Androgenic Alopecia	00 (00%)	00 (00%)	5 (2.4%)	
Severe	00 (00%)	4 (8%)	55 (26.7)%	
Acne				
Cystic acne	00 (00%)	00 (00%)	1 (2%)	
Slight	00 (00%)	13 (26%)	45 (21.8%)	**<0.0001**
Moderate	9 (19.1%)	11 (22%)	33 (16%)	
Severe	00 (00%)	13 (26%)	65 (31.6%)	

Table 2. Hormonal parameters data based on vitamin D status in PCOS patients.

Characteristics n (%)	PCOS patients, n=303			p-value
	Sufficiency n=47	Insufficiency n=50	Deficiency n=206	
FSH	5.577±2.084	5.229±1,553	4.868±2.293	0,102
Normal	42 (89.4%)	40 (80%)	157 (76.2%)	
Low	4 (8.5%)	9 (18%)	46 (22.3%)	0.307
High	1 (2.1%)	1 (2%)	3 (1.5%)	
LH	6.547±3.487	11.094±3.297	11.453±5.183	**<0.0001**
Normal	26 (55.3%)	4 (8%)	24 (11.7%)	
Low	2 (4.3%)	00 (00%)	2 (1%)	**<0.0001**
High	19 (40.4%)	46 (92%)	180 (87.4%)	
LH/FSH	1.226±0.609	2.164±0.452	2.458±0.584	**<0.0001**
Progesterone	0.863±0.614	0.533±0.458	0.331±0.368	**<0.0001**
Low	00 (00%)	21 (42%)	114 (55.3%)	**<0.0001**
Total testosterone	0.360±0.109	0.740±0.126	1.363±0.363	**<0.0001**
Interpretation of hyperandrogenism				
< 0.6	47 (100%)	1 (2%)	00 (00%)	**<0.0001**
≥ 0.6	00 (00%)	49 (98%)	206 (100%)	
Hypertestosteronemia	00 (00%)	40 (80%)	206 (100%)	**<0.0001**
AMH	3.655±0.813	8.878±2.308	12.674±2.553	**<0.0001**
High	00 (00%)	46 (92%)	206 (100%)	**<0.0001**

Statistical analysis

To compare qualitative values, we used the chi-square test, while the quantitative values were expressed as mean±standard deviation. Pearson's correlation and the one-way ANOVA test were applied for the comparative study.

The study data were analyzed with SPSS version 22.0 software (IBM Corp). p-values <0.05 were regarded as statistically significant.

Results

Demographic and clinical characteristics

A total of 303 patients aged between 16 and 45 years were included in our study. The patients' demographic and clinical characteristics are presented in Table 1.

A low serum level of 25-hydroxyvitamin D (deficiency) was mainly noted in 206 (67.9%) cases and 50 (16.5%) cases had an insufficient level, while only 47 (15.5%) cases had normal vitamin D levels.

Considering the mean age, the deficient group was the oldest (31.180±6.679 years), followed by the insufficient group (27.900±5.768 years), and the normal group (28.894±6.673 years); the p-value of 0.002 indicates a statistically significant difference in age among the three groups (Table 1).

Most of the group 3 (vitamin D deficiency) cases were from urban areas (67.5%); the distribution across the three groups was almost similar, so the findings were not statically significant (Table 1).

We noted a highly significant correlation between vitamin D deficiency and BMI, especially overweight, irregular cycle, hirsutism, slight alopecia/hair loss, severe acne and (p=<0.0001). We also noted a significant correlation between vitamin D status and secondary infertility (p=0.017).

Biochemical and hormonal profiles

Hormonal assessment showed that abnormal levels were mostly found in the deficient vitamin D group than in those with normal vitamin D levels.

High total testosterone, LH, AMH, LH/FSH ratio and low progesterone levels were statistically significantly higher in group 3 (vitamin D deficiency) (p<0.0001). FSH levels were not statistically significant (p>0.05), as seen in Table 2.

We observed a negative correlation between 25(OH)VD3 serum concentrations and LH (p<0.0001) (Figure 2a), total testosterone (p<0.0001) (Figure 2B), and AMH (p<0.0001) (Figure 2c). We also noticed a positive correlation between serum 25(OH)VD3 concentrations and FSH (p=0.005) (Figure 2d) and progesterone (p<0.0001) (Figure 2e).

Discussion

Our study revealed a predominance of hypovitaminosis D in Algerian PCOS patients. However, the mechanism that causes PCOS has not yet been elucidated[18]. Our cross-sectional study aimed to evaluate the relationship between vitamin D levels, demographic data, clinical characteristics, and hormonal profiles in Algerian women with PCOS.

Thys-Jacobs et al. demonstrated that vitamin D deficiency was correlated with a mean age of 31 years in women diagnosed with PCOS[19]. Bhattarai et al. stated that vitamin D synthesis is also influenced by age[20], which is consistent with the results of our study.

Obesity is recognized as a risk factor for vitamin D deficiency. Our results, along with other international reports, highlight that a low vitamin D concentration is associated with increased BMI in women with PCOS[10,15,21]. Li et al. reported that 25-hydroxyvitamin D concentrations were negatively correlated with BMI[14]. Our results are also consistent to those published by Artur et al., who noted a statistically significant correlation between serum levels of vitamin D and BMI[22].

Previous studies have revealed that the changes in intracellular calcium concentrations caused by vitamin D deficiency may lead to ovulation and reproductive abnormalities in PCOS[10]; other studies results demonstrated that low serum vitamin D is correlated with menstrual irregularity[23] and infertility[24,25].

The present study is consistent with the Hanif et al. study, which revealed that vitamin D deficiency is associated with clinical features of hyperandrogenism[26], including hirsutism, alopecia, infertility, acne, and central obesity[27].

In the literature, statistically significantly higher values of LH, total testosterone[28-30], LH/FSH rapport[31], and AMH levels[23] were noted, while progesterone concentrations were significantly lower in women with PCOS (p<0.0001). FSH level did not show any statistically significant association[31]. Similarly, significant positive correlations were observed between serum 25(OH)D levels, and FSH (p=0.005)[32] and progesterone levels (p<0.0001)[33]. Conversely, serum 25(OH)D levels in PCOS patients were negatively correlated (p < 0.0001) with LH, FSH, testosterone and AMH levels[32]. These results suggest that vitamin D deficiency is one of the alarming factors in the prevalence of PCOS and its clinical and hormonal complications[34].

This study is subject to several methodological constraints and limitations. First, the restricted accessibility to the clinical center limited the sample size and hence the statistical power to detect subtle

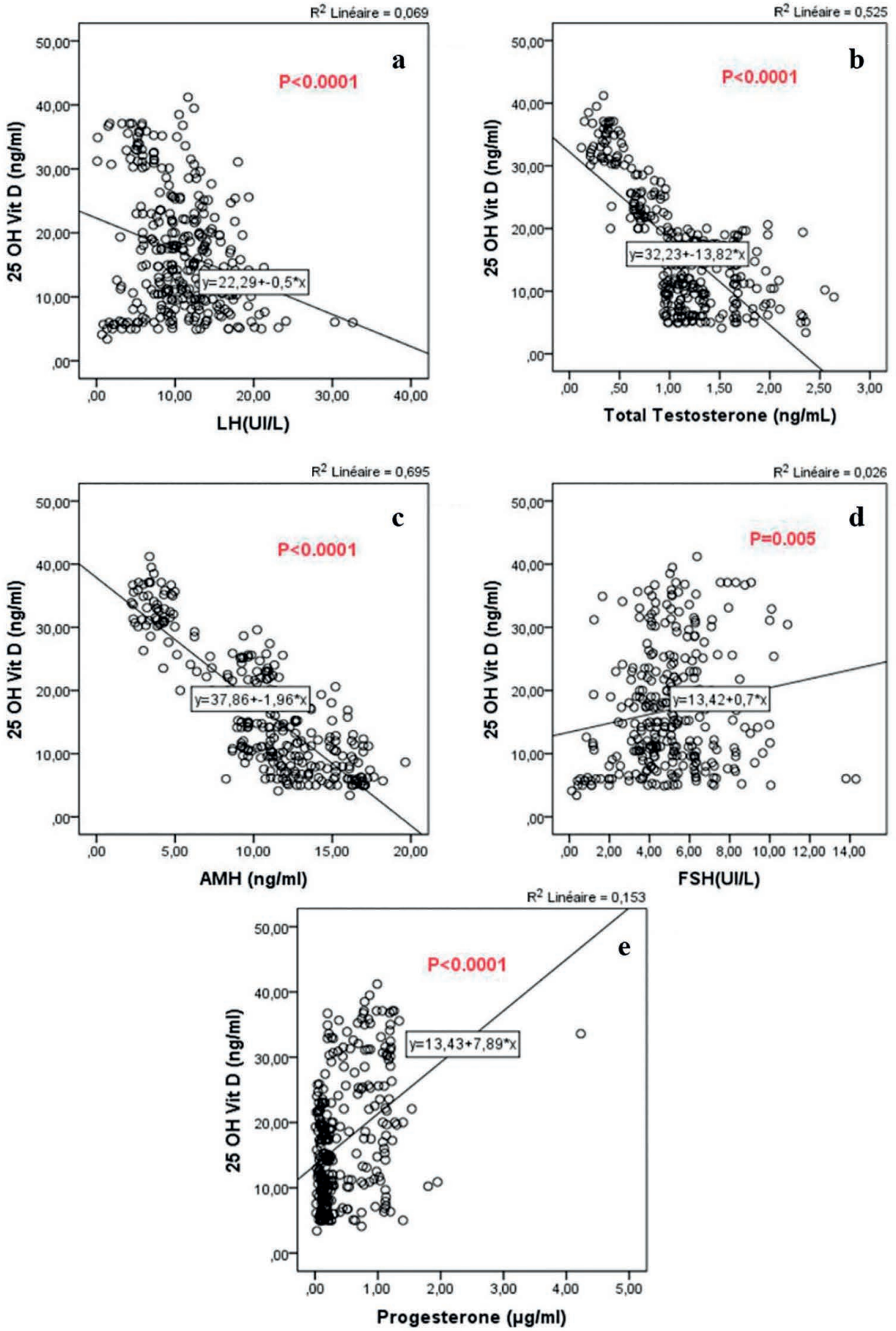


Figure 2. Correlation of 25(OH)D levels and hormonal parameters with PCOS.

associations. Moreover, there was an insufficiency of comprehensive demographic and biochemical data, which undermines the capacity to control for potential confounders and effect modifiers. The absence of longitudinal follow-up precludes inference on causality and temporal dynamics of vitamin D influence on PCOS pathophysiology. Additionally, the study did not incorporate advanced molecular diagnostics, such as extensive genetic and epigenetic profiling, which could elucidate underlying mechanistic pathways and interindividual variability. Future investigations would benefit from integrating multi-omics approaches and conducting randomized controlled trials to enhance the evidentiary framework regarding vitamin D's modulatory role in PCOS.

Conclusions

Overall, the data of the current study suggest that vitamin D deficiency is highly prevalent among women with PCOS and is associated with clinical manifestations and altered hormonal profiles in PCOS patients. However, the current observations cannot be generalized to all Algerian women and further studies with large sample sizes and in different locations are warranted to validate these findings and improve diagnostic and treatment strategies for women with PCOS.

Author Contributions:

Conceptualization, A.M., K.Z., N.H., and S.O.; methodology, A.M., A.BEL., and A.B.; software, A.M., S.O., and A.BEL.; validation, A.M., N.H., K.Z., and A.E.D.; formal analysis, S.O., and M.R.Z.; investigation, A.M., A.BEL., C.H., and F.S.; resources, A.M., C.H., and F.S.; data curation, A.M., W.N.E. and S.O.; writing–original draft preparation, A.M., and Z.K.; writing–review and editing, A.M., N.H., K.Z., O.S., and S.Z.; visualization, A.M., N.H., and K.Z.; supervision, N.H., K.Z., and A.E.D.; project administration, N.H., and K.Z. All the authors have read and agreed with the final version of the article.

Compliance with Ethics Requirements:

"The authors declare no conflict of interest regarding this article".

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2000(5), as well as the national law. Informed consent was obtained from all the patients included in the study".

"No funding for this study".

Acknowledgements:

We would like to thank the patients for their participation and staff at EL ASSRAR Center and AL ANIS Clinic, for their invaluable support, guidance, and educational insight.

References

1. Franks S. Polycystic ovary syndrome. *The New England Journal of Medicine*. 1995;333(13):853-861.
2. Teede HJ, Hutchison S, Zoungas S, Meyer C. Insulin resistance, the metabolic syndrome, diabetes, and cardiovascular disease risk in women with PCOS. *Endocrine*. 2006;30(1):45-53.
3. Ali AT. Polycystic ovary syndrome and metabolic syndrome. *Ceska Gynekol*. 2015;80(4):279-89.
4. Szczuko M, Zapałowska-Chwyć M, Drozd A, Maciejewska D, Starczewski A, Stachowska E. Effect of IGF-I and TNF-α on intensification of steroid pathways in women with PCOS phenotypes are not identical. Enhancement of progesterone pathway in women with PCOS increases the concentration of TNF-α. *Gynecol Endocrinol*. 2016;32(9):714-717.
5. Harris HR, Terry KL. Polycystic ovary syndrome and risk of endometrial, ovarian, and breast cancer: a systematic review. *Fertility Research and Practice*. 2016;2, 14.
6. Han J, Guo X, Yu X, et al. 25-Hydroxyvitamin D and total cancer incidence and mortality: a meta-analysis of prospective cohort studies. *Nutrients*. 2019;11(10):2295.
7. Yang J, Ou-Yang J, Huang J. Low serum vitamin D levels increase the mortality of cardiovascular disease in older adults: A dose-response meta-analysis of prospective studies. *Medicine (Baltimore)*. 2019;98(34):6733.
8. Pludowski P, Holick M, Pilz S, et al. Vitamin D effects on musculoskeletal health, immunity, autoimmunity, cardiovascular disease, cancer, fertility, pregnancy, dementia and mortality-a review of recent evidence. *Autoimmunity Reviews*. 2013;12(10):976-89.
9. Rafiq S, Jeppesen PB. Body mass index, vitamin D, and type 2 diabetes: a systematic review and meta-analysis. *Nutrients*. 2018;10(9):1182.
10. Yildizhan R, Kurdoglu M, Adali E, et al. Serum 25-hydroxyvitamin D concentrations in obese and non-obese women with polycystic ovary syndrome. *Archives of Gynecology and Obstetrics*. 2009;280(4):559-563.
11. Parikh G, Varadinova M, Suwandhi P, et al. Vitamin D regulates steroidogenesis and insulin-like growth factor binding protein-1 (IGFBP-1) production in human ovarian cells. *Hormone and Metabolic Research*. 2010;42(10):754-757.
12. Norman RJ, Dewailly D, Legro RS, et al. Polycystic ovary syndrome. *Lancet*. 2007;370:685-97
13. Krul-Poel YH, Snackey C, Louwers Y, et al. The role of vitamin D in metabolic disturbances in polycystic ovary syndrome: A systematic review. *European Journal of Endocrinology*. 2013;169(6):853-65.
14. Li HW, Brereton RE, Anderson, RA, Wallace AM, Ho CK. Vitamin D deficiency is common and associated with metabolic risk factors in patients with polycystic ovary syndrome. *Metabolism: Clinical and Experimental*. 2011;60(10):1475-81.
15. Hahn S, Haselhorst U, Tan S, et al. Low serum 25-hydroxyvitamin D concentrations are associated with insulin resistance and obesity in women with polycystic ovary syndrome. *Experimental and Clinical Endocrinology and Diabetes*. 2006;114(10):577-583.

16. Al-Hazmi AS. Association of vitamin D deficiency and vitamin D receptor gene polymorphisms with type 2 diabetes mellitus Saudi patients. *African Health Science*. 2019;19(4):2812-2818.
17. Geisthövel F, Rabe T. The ESHRE/ASRM consensus on polycystic ovary syndrome (PCOS): an extended critical analysis. *Reproductive BioMedicine Online*. 2007;14(4):522-535.
18. He C, Lin Z, Robb SW, Ezeamama AE. Serum vitamin D levels and polycystic ovary syndrome: a systematic review and meta-analysis. *Nutrients*. 2015;7(6):4555-4577.
19. Thys-Jacobs S, Donova D, Papadopoulos A, et al. Vitamin D and calcium dysregulation in the polycystic ovarian syndrome. *Steroids*, 1999;64:430-435.
20. Bhattarai HK, Shrestha S, Rokka K, Shakya R. Vitamin D, calcium, parathyroid hormone, and sex steroids in bone health and effects of aging. *Journal of Osteoporos*. 2020;2020:1-10.
21. Wehr E, Pilz S, Schweighofer N, et al. Association of hypovitaminosis D with metabolic disturbances in polycystic ovary syndrome. *European Journal of Endocrinology*. 2009;161:575-82.
22. Artur R, Mariusz W, Kamil B, et al. The correlation of vitamin D level with body mass index in women with polycystic ovary syndrome. *Ginekologia Polska*. 2023;94(11):883-888.
23. Sanchari C, Tapan KN, Barnali RB. Vitamin D deficiency, insulin resistance, and antimüllérien hormone level: A tale of trio in the expression of polycystic ovary syndrome. *F and S Science*. 2024;5(3):252-264.
24. Lerchbaum E, Obermayer-Pietsch B. Mechanisms in endocrinology: vitamin D and fertility: a systematic review, *European Journal of Endocrinology*. 2012;166(5):765–778.
25. Madjid MA, Hassann WF, Ridha AF, Prevalence of 25-hydroxyvitamin D (vitamin D) deficiency in a group of infertile women from Baghdad City. *Biochemistry Research International*. 2023;2023(1):1-6.
26. Hanif F, Ilyas N, Qamar T, Association of vitamin D deficiency with hyperandrogenism in females with polycystic ovarian syndrome. *International Journal of Clinical and Biomedical Research*. 2018;4:31.
27. Harish N, Kannan L, Ganesh Balaji R, et al. A study to find out the prevalence and associated risk factors of Hirsutism in polycystic ovarian syndrome and non-polycystic ovarian syndrome individuals. *Indian Journal of Applied Research*. 2017;7:65-68.
28. Bachelot A. Polycystic ovarian syndrome: clinical and biological diagnosis. *Annales de Biologie Clinique*. 2016;74:661–7.
29. McCartney CR, Marshall JC. Clinical practice. Polycystic ovary syndrome. *The New England Journal of Medicine*. 2016;375(14):54–64.
30. Macut D, Bjekić-Macut J, Rahelić D, Doknić M. Insulin and the polycystic ovary syndrome. *Diabetes Research and Clinical Practice*. 2017;130:163–70.
31. Zerrouki D, Rami I, Assarrar I, Bouichrat N, Rouf S, Latrech H. Is there any association between vitamin D status and PCOS disease? *Gynecological Endocrinology*, 2024;40(1):1-7.
32. Kensara OA. Prevalence of hypovitaminosis D and its association with hypoadiponectinemia and hyperfollistatinemia, in Saudi women with naïve polycystic ovary syndrome. *Journal of Clinical and Translational Endocrinology*. 2018;12:20–25.
33. Kolcsár M, Berecki B, Gáll Z. Relationship between serum 25-hydroxyvitamin D levels and hormonal status in infertile women: a retrospective study. *Diagnostics (Basel)*. 2023;13(19):3024.
34. Gokosmanoglu F, Onmez A, Ergenç H. The relationship between vitamin D deficiency and polycystic ovary syndrome. *African Health Sciences*. 2020;20(4):1880-6.