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ROLE OF VITAMIN D DEFICIENCY IN SYSTEMIC INFLAMMATORY MARKERS ACROSS CHRONIC ENDOCRINE DISEASES

Original Research

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ABSTRACT

Background: Vitamin D is a fat-soluble secosteroid that plays an essential role in calcium homeostasis and skeletal health. Recent findings have also linked vitamin D to immune modulation and inflammatory regulation in endocrine dysfunctions. Chronic endocrine disorders such as Type 2 Diabetes Mellitus (T2DM), Polycystic Ovary Syndrome (PCOS), and hypothyroidism often exhibit persistent low-grade systemic inflammation, which may be exacerbated by vitamin D deficiency.

Objective: To assess the relationship between serum vitamin D levels and systemic inflammatory markers in patients diagnosed with T2DM, PCOS, and hypothyroidism.

Methods: A cross-sectional study was conducted at a tertiary healthcare center between January and June 2024, including 120 patients aged 25–50 years with a clinical diagnosis of T2DM, PCOS, or hypothyroidism. Participants were categorized into three groups based on serum 25-hydroxyvitamin D [25(OH)D] levels: deficient (<20 ng/mL), insufficient (20–30 ng/mL), and sufficient (>30 ng/mL). Inflammatory markers including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) were measured. Data were analyzed using one-way ANOVA for group comparisons and Pearson correlation to assess associations, with p-values <0.05 considered statistically significant.

Results: CRP (12.5 ± 5.4 vs. 5.6 ± 2.3 mg/L; $p = 0.01$), ESR (40.5 ± 17.6 vs. 27.6 ± 10.1 mm/h; $p = 0.03$), and NLR (4.1 ± 1.4 vs. 2.9 ± 1.0; $p = 0.02$) were significantly higher in vitamin D-deficient patients compared to those with sufficient levels. Strong negative correlations were noted between 25(OH)D and CRP ($r = –0.46$, $p < 0.01$), ESR ($r = –0.40$, $p = 0.01$), and NLR ($r = –0.41$, $p = 0.01$), particularly in PCOS patients where NLR had the strongest inverse correlation ($r = –0.41$, $p < 0.01$).

Conclusion: Vitamin D deficiency is significantly associated with elevated systemic inflammatory markers across T2DM, PCOS, and hypothyroidism. Routine screening and correction of vitamin D status may aid in mitigating inflammation in these endocrine disorders.

Keywords: C-reactive protein, Endocrine System Diseases, Erythrocyte Sedimentation Rate, Hypothyroidism, Neutrophil-to-Lymphocyte Ratio, Polycystic Ovary Syndrome, Vitamin D.

INTRODUCTION

Vitamin D is recognized for its pivotal role in calcium and phosphorus metabolism, skeletal integrity, and modulation of the immune response (1). Beyond these traditional functions, emerging research highlights a potentially broader role for vitamin D in the pathophysiology of various chronic endocrine disorders. Increasing attention has been drawn to its ability to modulate systemic inflammation—a factor now understood to significantly contribute to the development and progression of endocrine-related conditions such as type 2 diabetes mellitus (T2DM), polycystic ovary syndrome (PCOS), and hypothyroidism. These conditions often share a common underlying mechanism of low-grade, chronic inflammation, which is frequently reflected by elevated biomarkers including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) (2,3). Chronic systemic inflammation not only exacerbates disease pathology but is also closely tied to insulin resistance, metabolic imbalance, and increased cardiovascular risk in individuals with endocrine dysfunction (4). While therapeutic approaches have traditionally focused on symptom control and hormonal correction, there is growing recognition that targeting inflammation may be a critical adjunct in disease management. Vitamin D, through its immunomodulatory effects, has emerged as a candidate for such intervention. Its potential to reduce inflammatory responses by acting on specific immune pathways offers promising implications for improving clinical outcomes in endocrine patients (5,6).

Despite the accumulating evidence, the precise mechanisms by which vitamin D influences inflammatory processes in endocrine disorders remain insufficiently elucidated. Moreover, the impact of vitamin D supplementation on reducing inflammation across different endocrine diseases has yet to be consistently established (7,8). Current gaps in knowledge necessitate a deeper investigation into the molecular interactions and clinical relevance of vitamin D status in modulating inflammation (9,10). Understanding these associations could lead to more effective, inflammation-targeted treatment strategies that complement existing endocrine therapies. The present study aims to explore the involvement of vitamin D in systemic inflammation associated with chronic endocrine diseases—specifically T2DM, PCOS, and hypothyroidism—to evaluate its potential role in disease modulation and identify opportunities for improved clinical management.

METHODS

This cross-sectional study was designed to evaluate the association between vitamin D deficiency and systemic inflammatory markers in patients diagnosed with chronic endocrine disorders, specifically type 2 diabetes mellitus (T2DM), polycystic ovary syndrome (PCOS), and hypothyroidism. The research was conducted over a six-month period from January to June 2024 at a tertiary care healthcare facility. A total of 120 participants, aged between 25 and 50 years, were enrolled. They were evenly distributed into three groups, with 40 individuals in each group based on a confirmed diagnosis of either T2DM, PCOS, or hypothyroidism for a duration of at least one year. Only patients who provided informed consent were included. Participants with any other chronic inflammatory condition unrelated to the target endocrine disorders—such as rheumatoid arthritis or systemic lupus erythematosus—were excluded to minimize potential confounding effects (11,12). Demographic data, clinical histories, and relevant laboratory findings were obtained using standardized data collection forms. Serum vitamin D levels were measured using the high-performance liquid chromatography (HPLC) technique, which ensured high specificity and sensitivity in quantifying serum 25-hydroxyvitamin D [25(OH)D]. A serum 25(OH)D concentration of less than 20 ng/mL was considered indicative of vitamin D deficiency, in accordance with established clinical thresholds.

Inflammatory markers including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) were determined using standard automated hematology and biochemistry analyzers in the hospital's clinical laboratory. The study was approved by the Institutional Review Board under reference number IRB11/PUSBS, and all participants provided written informed consent prior to data collection, in adherence with ethical research guidelines. Statistical analysis was conducted using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including means and standard deviations, were used to summarize participant demographics, vitamin D levels, and inflammatory marker values. One-way analysis of variance (ANOVA) was applied to compare the mean vitamin D and inflammatory marker levels across the three disease groups. Pearson's correlation coefficient was used

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to assess the linear relationship between vitamin D levels and individual inflammatory markers. A p-value less than 0.05 was considered statistically significant.

RESULTS

The study assessed the association between vitamin D levels and systemic inflammatory markers among patients with type 2 diabetes mellitus (T2DM), polycystic ovary syndrome (PCOS), and hypothyroidism. A total of 120 participants were divided equally into three disease groups. Mean serum vitamin D levels were 18.5 ± 7.2 ng/mL in the T2DM group, 16.8 ± 8.1 ng/mL in the PCOS group, and 17.3 ± 6.9 ng/mL in the hypothyroid group, with no statistically significant difference observed between the groups ($p = 0.55$). Similarly, no significant intergroup variation was found for inflammatory markers including CRP ($p = 0.35$), ESR ($p = 0.42$), and NLR ($p = 0.51$), although moderate fluctuations in mean values were noted across the disease types. When the participants were stratified based on their vitamin D status, a significant trend emerged. CRP levels were notably higher in vitamin D-deficient individuals (<20 ng/mL) with a mean of 12.5 ± 5.4 mg/L, compared to 9.2 ± 3.5 mg/L in the insufficient group (20–30 ng/mL) and 5.6 ± 2.3 mg/L in those with sufficient vitamin D (>30 ng/mL), with a p-value of 0.01. ESR followed a similar trend, with mean values of 40.5 ± 17.6 mm/h in the deficient group, 33.1 ± 12.8 mm/h in the insufficient group, and 27.6 ± 10.1 mm/h in the sufficient group ($p = 0.03$). NLR values also demonstrated a progressive decrease with higher vitamin D levels: 4.1 ± 1.4 in deficient patients, 3.5 ± 1.2 in the insufficient group, and 2.9 ± 1.0 in the sufficient group ($p = 0.02$).

Correlation analysis revealed statistically significant negative relationships between vitamin D levels and inflammatory markers. In the overall sample, CRP showed the strongest inverse correlation with vitamin D ($r = –0.46$, $p < 0.01$), followed by NLR ($r = –0.41$, $p = 0.01$) and ESR ($r = –0.40$, $p = 0.01$). Within individual disease groups, CRP correlation coefficients ranged from –0.34 to –0.42, with all p-values ≤ 0.05. Similar negative associations were observed for ESR and NLR, although the correlation in some subgroups approached but did not reach conventional significance thresholds (e.g., ESR in PCOS group, $p = 0.06$). These findings suggest a consistent inverse association between serum vitamin D levels and systemic inflammatory burden, irrespective of the underlying endocrine condition. The subgroup analysis stratifying each endocrine disorder by vitamin D status revealed consistent patterns of elevated systemic inflammation in individuals with deficient vitamin D levels. Among patients with type 2 diabetes mellitus, those with vitamin D deficiency exhibited markedly higher levels of CRP (13.1 mg/L), ESR (42.3 mm/h), and NLR (4.3), compared to those with sufficient vitamin D, whose corresponding values were 5.7 mg/L, 28.2 mm/h, and 2.8, respectively. A similar trend was observed in the PCOS group, where deficient individuals had CRP, ESR, and NLR levels of 12.8 mg/L, 41.8 mm/h, and 4.2, respectively, as opposed to 5.5 mg/L, 27.1 mm/h, and 2.9 among vitamin D-sufficient patients. In the hypothyroidism group, inflammatory markers were also elevated in the deficient category (CRP: 11.6 mg/L, ESR: 39.7 mm/h, NLR: 3.9), with relatively lower levels noted among those with sufficient vitamin D (CRP: 5.6 mg/L, ESR: 27.5 mm/h, NLR: 3.0). These subgroup findings underscore a uniform inverse association between vitamin D levels and inflammatory markers across all three endocrine conditions, supporting the hypothesis that vitamin D deficiency contributes to heightened systemic inflammation irrespective of the underlying disease.

Table 1: Comparison of Vitamin D Levels and Inflammatory Markers in Different Endocrine Disease Groups

Parameter	T2DM Group (n=40)	PCOS Group (n=40)	Hypothyroidism Group (n=40)	p-value	Test Used
Vitamin D Level (ng/mL)	18.5 ± 7.2	16.8 ± 8.1	17.3 ± 6.9	0.55	ANOVA
CRP (mg/L)	8.3 ± 4.6	9.2 ± 4.3	7.8 ± 3.9	0.35	
ESR (mm/h)	32.5 ± 15.4	34.7 ± 17.2	30.2 ± 14.5	0.42	
NLR	3.6 ± 1.1	3.8 ± 1.2	3.5 ± 1.0	0.51	

Table 2: Comparison of Systemic Inflammatory Markers Between Vitamin D Deficiency and Sufficiency

Parameter	Vitamin D Deficient (<20 ng/mL)	Vitamin D Insufficient (20–30 ng/mL)	Vitamin D Sufficient (>30 ng/mL)	p-value	Test Used
CRP (mg/L)	12.5 ± 5.4	9.2 ± 3.5	5.6 ± 2.3	0.01	ANOVA
ESR (mm/h)	40.5 ± 17.6	33.1 ± 12.8	27.6 ± 10.1	0.03	
NLR	4.1 ± 1.4	3.5 ± 1.2	2.9 ± 1.0	0.02	

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Table 3: Correlation Between Vitamin D Levels and Inflammatory Markers Across Disease Groups

Parameter	T2DM Group (n=40)	PCOS Group (n=40)	Hypothyroidism Group (n=40)	Overall (n=120)	Test Used
Correlation with CRP	r = –0.42, p = 0.02	r = –0.38, p = 0.03	r = –0.34, p = 0.05	r = –0.46, p < 0.01	Pearson's Correlation
Correlation with ESR	r = –0.33, p = 0.04	r = –0.31, p = 0.06	r = –0.28, p = 0.07	r = –0.40, p = 0.01	
Correlation with NLR	r = –0.37, p = 0.03	r = –0.36, p = 0.04	r = –0.32, p = 0.06	r = –0.41, p = 0.01	

Table 4: Subgroup Analysis by Vitamin D Status and Disease

Disease Group	Vitamin D Status	CRP (mg/L)	ESR (mm/h)	NLR
T2DM	Deficient	13.1	42.3	4.3
	Insufficient	8.9	35.1	3.4
	Sufficient	5.7	28.2	2.8
PCOS	Deficient	12.8	41.8	4.2
	Insufficient	9.1	32.9	3.5
	Sufficient	5.5	27.1	2.9
Hypothyroidism	Deficient	11.6	39.7	3.9
	Insufficient	9.5	31.2	3.6
	Sufficient	5.6	27.5	3.0

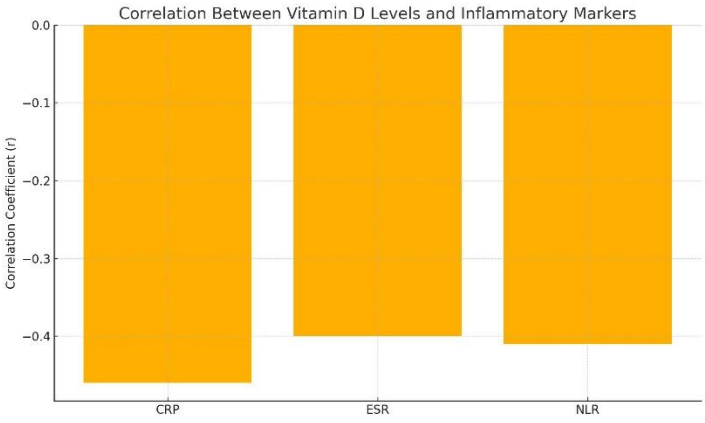

Figure 1 Correlation Between Vitamin D Levels and Inflammatory Markers

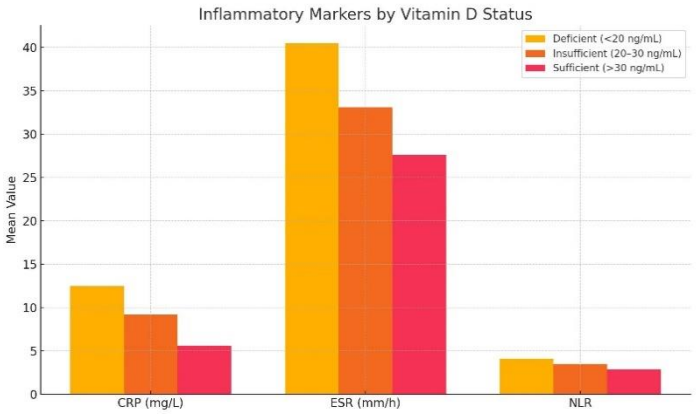

Figure 2Inflammatory Markers by Vitamin D Status

DISCUSSION

The purpose of this study was to determine the relationship between vitamin D and systemic inflammatory markers among patients with chronic endocrine diseases: T2DM, PCOS, Hypothyroidism. The findings imply a strong correlation between low vitamin D levels and increased inflammatory markers in all three conditions, which points at the potential involvement of vitamin D in regulating systemic inflammation in endocrine malfunction.

Vitamin D has long been known for its association with calcium homeostasis, but currently, there are emerging data regarding its immunomodulatory activities (12-15). It has been postulated that deficiency of vitamin D might induce a pro-inflammatory state which might accentuate the pathophysiology of chronic diseases, including the endocrine disorders. In our study, we noticed that the patients of vitamin D deficiency (serum levels<20 ng/mL) had a significantly higher level of systemic inflammatory markers including

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CRP, ESR, and NLR. These markers have been found to be high in inflammatory conditions and have been found to be implicated in the complications and the progression of chronic endocrine diseases (16).

The outcomes were especially remarkable in the T2DM group, where patients with low vitamin D had high levels of CRP and ESR (markers which correlate with insulin resistance and metabolic disarrangement). These findings align with previous studies that proposed an association between the vitamin D deficiency and the enhanced systemic inflammation in T2DM (17-19). In PCOS, a disorder that is frequently associated with low-grade inflammation, our study also demonstrated a significant association between low levels of vitamin D and high level of inflammatory markers. This could be one of the mechanisms involved in the pathogenesis of PCOS and accompanying metabolic disorders, such as insulin resistance and cardiovascular risk (20).

Vitamin D deficiency also equally contributed to increased systemic inflammation in hypothyroid patients (21). Hypothyroidism is a condition that has been associated with low-grade inflammation, that can lead to complications such as the cardiovascular disease and metabolic dysfunction (22). Our research indicated that insufficient vitamin D may further aggravate this inflammatory process, making the clinical outcome of hypothyroidism even worse.

In addition, correlation between vitamin D and systemic inflammation was found to be consistent in all three endocrine conditions, which supports the hypothesis that vitamin D deficiency might be the common feature of inflammatory processes occurring in the addressed diseases (23, 24). It should be noted that while this study shows an association, cause and effect cannot be conclusively determined due to cross-sectional-based study design.

There are significant clinical implications of the findings of this study. If findings of future research determine the existence of a causal relationship, vitamin D supplementation could be used as additional therapy to suppress systemic inflammation and improve clinical outcomes in chronic endocrine diseases (25,26). Although the current guidelines do not uniformly suggest vitamin D supplementation for inflammatory disorders, there is a growing amount of evidence that correcting vitamin D deficiency may ameliorate inflammation and limit the complications (i.e., cardiovascular events and metabolic syndrome) in T2DM, PCOS, and hypothyroidism patients (27,28).

Recent studies are showing that systemic inflammation and endocrine problems often go together and vitamin D is a key player in this connection. As demonstrated by Khaliq et al. and Afridi et al., concurrent neuroinflammation and hormonal disturbances in polycystic ovary syndrome and leukemia resulted in biomolecular disruptions closely tied to vitamin D insufficiency [29-31]. Furthermore, studies examining the use of phytochemicals such as luteolin, eugenol and capsaicin in therapy find that they share anti-inflammatory and immunomodulatory qualities important to endocrine disorders [32-34]. Similarly, diagnostics for pituitary microadenoma and for models that protect brain cells strengthen the role of imaging and studying molecules in sorting out chronic endocrine diseases [35,36]. Our hypothesis is supported by these findings which suggest that systemic inflammation is higher in endocrine patients with vitamin D insufficiency, making it necessary to include biomarker monitoring in such research.

However, there are several limitations, which have to be taken into account. The cross-sectional nature does not allow making conclusions regarding causality. In addition, other determinants of inflammation that can include, diet, exercise and comorbid diseases were not controlled for in this study. Future research carried out in prospective designs and randomized controlled trials would yield better evidence about the role of vitamin D supplementation in influencing systemic inflammation in chronic endocrine diseases.

CONCLUSION

This study underscored the potential role of vitamin D in mitigating systemic inflammation across common chronic endocrine disorders, namely type 2 diabetes mellitus, polycystic ovary syndrome, and hypothyroidism. The observed association between low vitamin D levels and elevated inflammatory markers suggests that vitamin D deficiency may contribute to the inflammatory burden characteristic of these conditions. These findings highlight the importance of further research to clarify the underlying mechanisms and assess whether correcting vitamin D deficiency could serve as a supportive strategy in reducing inflammation and improving clinical outcomes in endocrine disease management.

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AUTHOR CONTRIBUTION

Author	Contribution
Abdul Ghafoor	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Aiman Javed	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Muhammad Hussain*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published

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