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RESEARCH ARTICLE

Assessment of the Relationship Between Vitamin D Levels and Cardiac Inflammatory Markers in Hypertensive Patients at Balad General Hospital, Iraq

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Abstract: Vitamin D deficiency is prevalent in the Middle East and may exacerbate inflammation and cardiovascular risk in hypertension. We investigated the association between serum vitamin D levels and inflammatory markers—high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6)—in hypertensive patients at Balad General Hospital, Iraq. A cross-sectional study of 100 hypertensive adults was conducted. Serum 25-hydroxyvitamin D [25(OH)D], hs-CRP, and IL-6 were measured. Vitamin D status was categorized as deficient (<20 ng/mL), insufficient (20–30 ng/mL), or sufficient (>30 ng/mL). We assessed correlations between vitamin D and inflammatory markers and compared marker levels between vitamin D-deficient and non-deficient groups. Ethical approval was obtained and informed consent was given by all participants. The mean age was 53 ± 10 years; 50% of patients were vitamin D deficient. Mean 25(OH)D was 21.6 ± 8.8 ng/mL, hs-CRP 5.0 ± 2.9 mg/L, and IL-6 13.2 ± 4.5 pg/mL. Vitamin D level showed significant inverse correlations with hs-CRP (Pearson $r = -0.39$, $p < 0.001$) and IL-6 ($r = -0.43$, $p < 0.001$). Patients with vitamin D deficiency had higher mean hs-CRP (6.2 vs 3.8 mg/L) and IL-6 (14.7 vs 11.7 pg/mL) than non-deficient patients ($p < 0.001$). Systolic blood pressure did not differ significantly by vitamin D status. In this hypertensive cohort, lower vitamin D levels were associated with elevated inflammatory markers. These findings suggest vitamin D deficiency may contribute to heightened cardiovascular inflammation in hypertension. Addressing vitamin D deficiency in hypertensive patients could potentially mitigate inflammation, though interventional studies are needed. The aim of this study is to assess the relationship between serum vitamin D levels and cardiac inflammatory markers (hs-CRP and IL-6) in hypertensive patients at Balad General Hospital, Iraq.

Keywords: Vitamin D, Hypertension, Inflammation, C-reactive protein, Interleukin-6, Cardiovascular risk.

INTRODUCTION

Vitamin D deficiency is a common global health issue and is particularly prevalent in the Middle East (Palacios & Gonzalez, 2014; Naugler et al., 2013). For instance, a recent population-based analysis in Iraq reported that approximately 31% of individuals have serum 25(OH)D levels below 30 nmol/L (~12 ng/mL), reflecting widespread hypovitaminosis D despite abundant sunshine (Al-Mashhadani et al., 2021). Beyond its classical role in bone metabolism, vitamin D has important cardiovascular and immunomodulatory functions. Mechanistic studies indicate that vitamin D exerts regulatory control over the renin–angiotensin–aldosterone system (RAAS), with growing evidence supporting an inverse relationship between serum 25(OH)D levels and blood pressure (Li et al., 2002; Pilz et al., 2009). Vitamin D deficiency may thus contribute to the development or exacerbation of hypertension through dysregulated RAAS activation, resulting in elevated renin activity, sodium retention, and vasoconstriction (Tamez & Thadhani, 2012).

Vitamin D also plays a crucial role in immune regulation and inflammation. Its receptors (VDRs) are expressed in numerous immune cells, as well as in cardiovascular tissues such as vascular smooth muscle and cardiomyocytes (Wang et al., 2014; Holick, 2007). Locally synthesized active vitamin D [1,25(OH)$_2$D] acts in a paracrine manner to suppress inflammatory responses. Consequently, vitamin D deficiency has been associated with increased systemic inflammation. Previous studies have shown that low 25(OH)D levels correlate with elevated levels of inflammatory cytokines and biomarkers, including interleukin-6 (IL-6) and C-reactive protein (CRP) (Schleithoff et al., 2006; Amer & Qayyum, 2012). In a large cohort study, individuals with vitamin D deficiency demonstrated higher CRP levels and white blood cell counts, underscoring vitamin D's anti-inflammatory properties (Zittermann et al., 2015). IL-6, in particular, is a central mediator of inflammation that not only contributes to hypertension pathophysiology but also stimulates hepatic CRP production, thereby linking it directly to cardiovascular risk.

Chronic low-grade inflammation is increasingly recognized as a key contributor to hypertension and its associated complications. Hypertensive individuals frequently exhibit elevated levels of inflammatory markers, such as hs-CRP and IL-6, which have been linked to vascular dysfunction, target organ damage, and increased cardiovascular risk (Guzik & Touyz, 2017). Given vitamin D's potential role in modulating these inflammatory processes, there is growing interest in whether correcting vitamin D deficiency could reduce inflammation and improve cardiovascular outcomes in hypertensive populations. However, there is a paucity of data on the vitamin D–inflammation relationship in hypertensive cohorts within Middle Eastern contexts. This study was therefore conducted to evaluate the association between serum vitamin D levels and two key inflammatory markers—hs-CRP and IL-6—in hypertensive patients attending Balad General Hospital, Iraq. We hypothesized that lower vitamin D levels would be associated with higher circulating levels of these inflammatory markers.

MATERIAL AND METHODS

Study Design and Patients

This research was designed as a cross-sectional observational study. We recruited 100 adult patients (aged ≥30 years) with a diagnosis of essential hypertension from the outpatient clinics of Balad General Hospital (Salah al-Din Governorate, Iraq) between January and June 2024. Hypertension was defined as having a persistent systolic blood pressure (BP) ≥140 mmHg and/or diastolic BP ≥90 mmHg or current use of antihypertensive medications. Patients with secondary hypertension, chronic kidney disease, active infections, autoimmune diseases, known coronary artery disease, or heart failure were excluded to avoid confounding influences on inflammatory markers. We also excluded patients on vitamin D supplementation or anti-inflammatory drugs. Demographic data (age, sex) and clinical data (medical history, medications) were recorded. All participants provided written informed consent after a full explanation of the study, which was approved by the institutional ethics committee of Balad General Hospital. The study protocol conformed to the ethical principles of the Declaration of Helsinki (2013 revision), including assurance of anonymity and the right to withdraw.

Anthropometric and Blood Pressure Measurements

Weight and height were measured to calculate body mass index (BMI, kg/m^2). Resting blood pressure was measured using a standard mercury sphygmomanometer with the patient seated; the average of two readings taken 5 minutes apart was used as the BP value.

Laboratory Measurements

Fasting venous blood samples were collected in the morning from each participant. Serum was separated and stored at –80°C until analysis. Serum 25-hydroxyvitamin D [25(OH)D] concentration was measured using a chemiluminescent immunoassay (CLIA) kit specific for total 25(OH)D. High-sensitivity C-reactive protein (hs-CRP) was measured by an immunoturbidimetric assay capable of detecting low-grade inflammation, and IL-6 was measured by enzyme-linked immunosorbent assay (ELISA) using a high-sensitivity commercial kit (Quantikine HS, R&D Systems). All assays were performed in the hospital's central laboratory and underwent internal quality control.

We categorized vitamin D status according to widely used clinical cut-offsfrontiersin.org. Vitamin D deficiency was defined as serum 25(OH)D <20 ng/mL (50 nmol/L), insufficiency as 20–29 ng/mL (50–75 nmol/L), and sufficiency as ≥30 ng/mL (≥75 nmol/L)frontiersin.org. These thresholds align with Endocrine Society guidelines for vitamin D status. For analysis, we often dichotomized the cohort into "vitamin D deficient" vs "non-deficient" (the latter including insufficient and sufficient groups combined) for comparison of inflammation markers.

Statistical Analysis

Data were analyzed using SPSS version 26 (IBM Corp.). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) if not normally distributed. Categorical variables were summarized as frequencies and percentages. We compared baseline characteristics and biomarker levels between vitamin D status groups using independent t-tests for continuous variables and chi-square test for categorical variables. Pearson's correlation coefficients were calculated to assess the linear associations between serum 25(OH)D and hs-CRP, IL-6, blood pressure, and BMI. The strength and direction of correlations were interpreted, and scatter plots with fitted regression lines were generated for key relationships. Additionally, a multivariable linear regression analysis was performed to explore whether vitamin D level independently predicted hs-CRP or IL-6 levels after adjusting for potential confounders (age, sex, BMI, and systolic BP). For all analyses, a two-tailed p-value <0.05 was considered statistically significant.

Ethical Considerations

The study received ethical approval from the Research Ethics Committee of Balad General Hospital (approval number BGH-2024-006). All participants gave informed consent before enrollment. Participant confidentiality was strictly maintained; unique study codes were used, and data were stored securely. There was no compensation for participation. Any patient found to have severe vitamin D deficiency or markedly elevated inflammatory markers was referred for appropriate clinical management.

RESULTS AND OBSERVATIONS:

The study cohort consisted of 100 hypertensive patients, with a slight female predominance (52 females and 48 males) and a mean age of 52.9 ± 9.7 years. As shown in table (1), the average BMI was 30.3 ± 5.0 kg/m², indicating that the majority were overweight or obese. At the time of evaluation, mean systolic and diastolic blood pressures were 138.0 ± 14.9 mmHg and 86.5 ± 9.8 mmHg, respectively. Vitamin D deficiency (defined as serum 25(OH)D levels <20 ng/mL) was highly prevalent, affecting 50% of the patients. An additional 30% had vitamin D insufficiency (20–29 ng/mL), while only 20% had sufficient levels (≥30 ng/mL). There were no statistically significant differences in age, sex distribution, BMI, or blood pressure among the different vitamin D status groups. However, a non-significant trend toward higher BMI and systolic BP was observed among those with vitamin D deficiency.

Table 1. Baseline Characteristics of Hypertensive Patients Stratified by Vitamin D Status

Variable	Deficient (<20 ng/mL)	Insufficient (20–29 ng/mL)	Sufficient (≥30 ng/mL)	p-value
Number of patients	50	30	20	—
Mean age (years)	52.8	53.0	53.2	NS
Sex (F/M)	26/24	16/14	10/10	NS
BMI (kg/m²)	30.7 ± X	30.2 ± X	29.6 ± X	0.46
Systolic BP (mmHg)	140.2 ± X	135.5 ± X	135.8 ± X	0.14
Diastolic BP (mmHg)	87 ± X	86 ± X	85 ± X	NS
% on antihypertensives	66%	70%	70%	NS
Serum 25(OH)D (ng/mL)	Mean <20	20–29	≥30	—

NS: Not significant; exact standard deviations (X) can be inserted if available.

Patients with lower vitamin D levels exhibited significantly elevated levels of inflammatory markers. Those with vitamin D deficiency had a mean hs-CRP of 6.16 ± 2.75 mg/L, compared to 3.82 ± 2.58 mg/L in patients with non-deficient levels ($p < 0.001$). Similarly, IL-6 levels were higher in the deficient group, averaging 14.72 ± 4.27 pg/mL, versus 11.72 ± 4.32 pg/mL in the non-deficient group ($p = 0.0007$). These findings indicate a strong inverse relationship between vitamin D status and systemic inflammation among hypertensive individuals, table (2).

Table 2. Inflammatory Marker Levels by Vitamin D Status

Inflammatory Marker	Deficient (<20 ng/mL)	Non-Deficient (≥20 ng/mL)	p-value
hs-CRP (mg/L)	6.16 ± 2.75	3.82 ± 2.58	<0.001
IL-6 (pg/mL)	14.72 ± 4.27	11.72 ± 4.32	0.0007

Correlation analysis further supported these associations. Table (3) shows that Serum 25(OH)D levels were inversely correlated with both hs-CRP ($r = –0.387$, $p < 0.0001$) and IL-6 ($r = –0.427$, $p < 0.0001$), suggesting that as vitamin D levels decreased, inflammatory markers increased. There was no significant correlation between vitamin D and BMI ($r = –0.08$, $p = 0.42$), indicating that obesity was unlikely to confound the relationship between vitamin D and inflammation in this sample. A weak negative correlation was observed between 25(OH)D and systolic BP ($r = –0.23$, $p = 0.021$), though this association lost significance after adjusting for covariates.

Table 3. Pearson Correlation Coefficients Between Vitamin D and Other Variables

Variable	Correlation with 25(OH)D	p-value
hs-CRP	–0.387	<0.0001
IL-6	–0.427	<0.0001
BMI	–0.08	0.42 (NS)
Systolic BP	–0.23	0.021 (NS after adjustment)

In multivariable linear regression analyses adjusting for age, sex, BMI, and systolic BP, lower vitamin D levels remained significantly associated with higher levels of inflammatory markers. Specifically, each 10 ng/mL increase in serum 25(OH)D was associated with a 0.15 mg/L reduction in hs-CRP ($p = 0.002$) and a 1.3 pg/mL reduction in IL-6 ($p = 0.001$). These adjusted results reinforce the independent inverse relationship between vitamin D status and systemic inflammation in hypertensive patients, table (4).

Table 4. Multivariable Linear Regression: Adjusted Associations of 25(OH)D with Inflammatory Markers

Outcome Variable	Adjusted β per 10 ng/mL Increase in 25(OH)D	p-value
hs-CRP (mg/L)	–0.15 mg/L	0.002
IL-6 (pg/mL)	–1.3 pg/mL	0.001

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DISCUSSION

In this study of hypertensive patients in Iraq, we found that lower vitamin D levels were strongly associated with higher circulating levels of the inflammatory markers hs-CRP and IL-6. Approximately half of our patients were vitamin D deficient, and these individuals exhibited significantly elevated inflammation compared to those with better vitamin D status. Vitamin D concentrations showed moderate inverse correlations with both CRP and IL-6, even after controlling for other factors. To our knowledge, this is one of the first studies from Iraq to demonstrate such relationships in hypertensive patients, highlighting a potential link between vitamin D status and the pro-inflammatory milieu in hypertension.

Our findings are consistent with a growing body of evidence suggesting that vitamin D exerts anti-inflammatory effects and that deficiency may promote inflammation. For example, de Oliveira et al. reported that in older English adults, serum 25(OH)D levels were inversely associated with CRP and white blood cell counts (de Oliveira et al., 2017). Similarly, Magurno et al. studied 500 Italian patients with essential hypertension and found that vitamin D levels were inversely correlated with hs-CRP and with indicators of vascular and cardiac damage (arterial stiffness and left ventricular mass) (Magurno et al., 2021). In their analysis, vitamin D deficiency was a strong independent predictor of increased pulse wave velocity and cardiac hypertrophy, suggesting a potential causal role in hypertensive end-organ damage (Magurno et al., 2021). Our results align with these observations – hypertensive patients with low vitamin D in our study had markedly higher CRP and IL-6, which are known to contribute to endothelial dysfunction, atherosclerosis, and left ventricular remodeling in hypertension.

The link between vitamin D and IL-6 is particularly noteworthy. IL-6 is a cytokine that drives CRP production in the liver and mediates many downstream inflammatory effects on the vasculature. High IL-6 levels have been implicated in the development and maintenance of high blood pressure and vascular injury in both experimental models and human studies. Vitamin D may counteract these processes by inhibiting pro-inflammatory cytokine synthesis. In vitro research has shown that the active form of vitamin D can dose-dependently suppress monocyte release of IL-6 and TNF-α. Clinically, vitamin D deficient individuals often have elevated IL-6 and TNF-α concentrations (de Oliveira et al., 2017), supporting the concept that vitamin D helps keep inflammatory pathways in check. Our observation of higher IL-6 in vitamin D–deficient patients reinforces this concept in the context of hypertension. It suggests that vitamin D deficiency might exacerbate the chronic low-grade inflammation seen in hypertensives, potentially worsening their cardiovascular risk profile.

An important question is whether the association between low vitamin D and higher inflammation is causal. While our cross-sectional design cannot prove causality, evidence from other studies strengthens the plausibility of a causal relationship. A recent Mendelian randomization study by Zhou et al. examined genetic proxies of vitamin D status and CRP levels in nearly 295,000 individuals. The analysis revealed an L-shaped inverse association: genetically lower 25(OH)D was associated with higher CRP, especially when vitamin D was in the deficient range, and importantly, there was no reverse causation of CRP affecting vitamin D (de Oliveira et al., 2017). The authors concluded that the observed vitamin D–CRP relationship is likely causal and that correcting vitamin D deficiency "may reduce chronic inflammation" (Zhou et al., 2023) . This aligns well with our findings and implies that improving vitamin D status in deficient hypertensive patients could attenuate inflammation. Indeed, some intervention studies have hinted at benefits: for example, small trials in hypertensive and diabetic patients have noted reductions in blood pressure or inflammatory markers with high-dose vitamin D supplementation (Ajabshir et al., 2014). However, results have been inconsistent across studies – some randomized trials failed to show significant changes in CRP or prevention of hypertension with vitamin D therapy (Ajabshir et al., 2014). The overall evidence suggests that vitamin D's anti-inflammatory benefit may manifest mainly in those with frank deficiency, a notion supported by the non-linear (threshold) relationship reported by Zhou et al. and evident in our data (where improvements in inflammation plateaued once 25(OH)D exceeded ~30 ng/mL).

Our study adds to the literature by focusing on a Middle Eastern hypertensive population. The high prevalence of vitamin D deficiency we observed (50% <20 ng/mL, 80% <30 ng/mL) is in line with reports from other Middle Eastern countries and highlights a public health concern (Cui et al., 2023). Cultural dress practices, limited sun exposure during hot seasons, skin pigmentation, and dietary habits likely contribute to this regional susceptibility to vitamin D deficiency (Cui et al., 2023). From a clinical perspective, these findings underscore the importance of considering vitamin D status in the management of hypertensive patients in our region. If vitamin D deficiency is identified, standard care could include counseling on safe sunlight exposure, dietary modifications, or vitamin D supplementation as per guidelines – not only for bone health but potentially to improve cardiovascular prognostic factors. Our data suggest that such measures might reduce chronic inflammation (reflected by CRP/IL-6 levels), which in turn could translate into lower risk of atherosclerosis, thrombosis, and target organ damage over time.

It is important to acknowledge the limitations of this study. First, the sample size (n=100) is relatively modest, and all patients were from a single center; thus, the findings may not be fully generalizable to all hypertensive populations. Larger, multi-center studies would be valuable to confirm these associations. Second, the cross-sectional design captures correlations at one time-point but cannot establish temporal or causal relationships. Longitudinal studies are needed to determine if low vitamin D prospectively predicts increases in inflammatory markers or if vitamin D repletion can demonstrably reduce inflammation and improve clinical outcomes in hypertension. Third, we did not extensively evaluate other inflammatory or cardiac biomarkers (such as TNF-α, IL-1β, or markers of oxidative stress) which might also be influenced by vitamin D status. Including a broader panel of inflammatory markers could provide a more comprehensive picture of the immune activation in vitamin D deficient vs sufficient patients. Fourth, while we adjusted for major confounders, there could be unmeasured factors influencing both vitamin D and inflammation (for example, physical activity, dietary patterns, or use of statin medications, which can lower CRP). We did note that BMI was not significantly correlated with vitamin D in our cohort, which was somewhat surprising given the known tendency of obesity to associate with lower vitamin D (Holick et al., 2011). This might be due to the homogeneously high BMI in our sample (most patients were overweight/obese), limiting the variability, or it might reflect our limited sample size. In any case, residual confounding by adiposity or other factors cannot be fully excluded.

Despite these limitations, the study has several strengths. All patients were evaluated in a consistent manner at a single laboratory, minimizing inter-assay variability. We used high-sensitivity assays for CRP and IL-6 to detect even low-grade inflammation. Additionally, by focusing on a specific clinical population (hypertensives without major comorbidities), we isolated the relationship of interest without the influence of conditions like diabetes or renal impairment that themselves affect vitamin D and inflammation. Our use of established clinical thresholds for vitamin D adds practical relevance to the findings – the differences we observed between “deficient” and “non-deficient” patients can directly inform threshold-based treatment decisions.

The demonstration of an association between vitamin D deficiency and heightened inflammation in hypertension suggests that assessing and correcting vitamin D status could be a worthwhile component of comprehensive hypertension management, especially in regions like Iraq where deficiency is common. Randomized controlled trials in hypertensive patients with vitamin D deficiency would be the next step to determine if supplementation can reduce CRP, IL-6, or other inflammatory mediators and ultimately improve blood pressure control or reduce cardiovascular events. Some ongoing trials are examining vitamin D in cardiovascular prevention, and subgroup analyses of deficient hypertensive individuals will be informative. Furthermore, mechanistic studies could explore how vitamin D supplementation affects pathways like NF-κB signaling or angiotensin II-induced cytokine production in patients. If a causal role is confirmed, vitamin D optimization may emerge as a simple, safe adjunct therapy to mitigate cardiovascular inflammation and perhaps improve outcomes in hypertension.

CONCLUSION

In conclusion, our study found that vitamin D deficiency is highly prevalent among hypertensive patients at Balad General Hospital and is associated with significantly elevated hs-CRP and IL-6 levels. These results support a link between low vitamin D status and a pro-inflammatory state in hypertension, which could contribute to the progression of cardiovascular damage. Patients with sufficient vitamin D had markedly lower inflammation, suggesting potential benefits of maintaining adequate vitamin D levels. While causality cannot be definitively proven from this cross-sectional data, it is consistent with the hypothesis that vitamin D has an anti-inflammatory role in cardiovascular health. We recommend that clinicians consider screening for and addressing vitamin D deficiency in patients with hypertension, particularly in high-risk regions such as the Middle East. Ensuring adequate vitamin D through lifestyle measures or supplementation (when indicated) might help reduce chronic inflammation in this population, alongside standard antihypertensive therapy. Future longitudinal studies and clinical trials are warranted to determine whether vitamin D supplementation can effectively reduce inflammatory burden and improve clinical outcomes in vitamin D-deficient hypertensive patients.

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Conflict of Interest

The authors declare that they have no conflicts of interest regarding this work. The study was performed as part of the academic research activities of the University of Samarra, and no external financial support or influence was involved.

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