THE PLANET

Original Article

Relationship of Serum Vitamin D Concentrations with Inflammatory Biomarkers in Adults

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ARTICLE INFO

Received: 12 Apr 2026
Accepted: 19 Apr 2026
Published Online: 5 May 2026

DOI: 10.5281/zenodo.20038797

Volume: 9, Number: 2, Page: 146-150

e-ISSN: 2789-5912
ISSN: 2617-0817

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ABSTRACT

Background: Vitamin D deficiency is prevalent in Bangladesh, and vitamin D is hypothesized to influence immune and inflammatory pathways in addition to its established role in bone health. This study aimed to examine the association between serum 25-hydroxyvitamin D and commonly measured inflammatory markers among adults. **Methods & Materials:** This analytical cross-sectional study (2023 to 2024) at NITOR and NIKDU, Dhaka, Bangladesh consecutively enrolled adults aged at least 18 years (n=184), excluding pregnancy, recent infection, major inflammatory or autoimmune disease, malignancy, advanced liver or kidney disease, high-dose vitamin D therapy, or immunosuppressant use. Serum 25(OH)D and routine inflammatory markers (hsCRP, ESR, albumin, uric acid, and CBC-derived NLR, PLR) were measured, with lifestyle and seasonality data recorded, and associations analyzed using correlation and adjusted linear regression in SPSS v26. **Results:** Among 184 adults (mean age 41.6 years; mean BMI 25.1 kg/m²), mean serum 25(OH)D was 19.4 ± 7.6 ng/mL, with 62% deficient, 25% insufficient, and 13% sufficient. Inflammatory burden was higher in vitamin D deficiency compared with sufficiency, including hsCRP, ESR, NLR, and PLR, while albumin was lower (all p≤0.041). Serum 25(OH)D correlated inversely with hsCRP, ESR, NLR, PLR, and positively with albumin (all p≤0.030). In adjusted models, each 10 ng/mL increase in 25(OH)D was associated with lower ln(hsCRP) (β = -0.18), lower ESR (β = -2.6 mm/hr), lower ln (NLR) (β = -0.12), and higher albumin (β = +0.07 g/dL). **Conclusion:** Vitamin D deficiency was common in this adult cohort, and lower serum 25(OH)D was independently associated with higher hsCRP, ESR, and NLR, and lower albumin.

Keywords: *Vitamin D deficiency, Serum 25(OH)D, Inflammatory Biomarkers, High-sensitivity C-reactive Protein*

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INTRODUCTION

Vitamin D is predominantly synthesized in the skin following ultraviolet-B exposure, with lesser contributions from dietary intake and supplementation. Beyond its established role in bone health, vitamin D exerts immunomodulatory effects, as evidenced by the presence of vitamin D receptors and activating enzymes in key immune cells, which enable modulation of inflammatory signaling [1,2]. Serum 25-hydroxyvitamin D (25(OH)D) is considered the standard marker of vitamin D status, as it most accurately reflects cumulative, long-term exposure. Vitamin D inadequacy is prevalent globally and demonstrates considerable variation by geography, ethnicity, and lifestyle factors. A global systematic review of population surveys identified substantial heterogeneity in 25(OH)D distributions across countries, with deficiency frequently observed even in regions with abundant sunlight [3]. Syntheses of nationally representative datasets further indicate that deficiency and inadequacy persist across world regions, influenced by factors such as latitude, urbanization, indoor occupational patterns, clothing practices, and limited dietary sources of vitamin D [4]. South Asia bears a particularly high burden; studies among South Asian adults consistently report high prevalence of deficiency across the region[5]. Bangladesh exemplifies this trend, with review-level evidence documenting widespread deficiency, common risk factors, and significant potential health consequences [6]. Empirical data from Bangladeshi adults further demonstrate that low vitamin D status is common even among apparently healthy individuals, underscoring concerns regarding subclinical deficiency in both community and hospital-based populations [7]. Systemic inflammation is a critical factor in the pathogenesis of cardiometabolic disease and other major chronic conditions. Evidence from cardiovascular outcomes trials demonstrates that reducing inflammation can decrease the incidence of major adverse events, independent of lipid-lowering interventions [8]. Due to the impracticality of cytokine assays in routine clinical care, particularly in resource-limited settings, commonly available markers such as high-sensitivity C-reactive protein (hsCRP), erythrocyte sedimentation rate (ESR), ferritin, albumin, and indices derived from complete blood counts are frequently employed to assess inflammatory burden. Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are increasingly utilized as composite markers of inflammation and immune balance in chronic disease contexts [9]. Mechanistic studies support multiple pathways through which vitamin D may modulate inflammatory activity. Recent reviews highlight vitamin D–mediated inhibition of nuclear factor kappa-B signaling, decreased production of pro-inflammatory cytokines, promotion of regulatory immune cell phenotypes, and enhancement of epithelial barrier and innate antimicrobial responses, collectively contributing to an anti-inflammatory environment [1,2]. However, evidence from human studies remains inconsistent, with challenges arising from confounding, reverse causality, and variability in baseline deficiency, adiposity, comorbidities, and assay methodologies across studies. Analyses integrating observational and genetic data have reported associations between vitamin D and C-reactive protein (CRP), while also illustrating the complexity of causal inference in population-based research [10]. Recent

bidirectional Mendelian randomization studies continue to investigate potential causal relationships between vitamin D deficiency and CRP, but have not definitively established whether vitamin D acts as a driver of inflammation, a marker of health status, or both, depending on context [11]. Interventional evidence also varies by population. Meta-analyses of randomized controlled trials indicate that vitamin D supplementation may reduce CRP and selected cytokines in certain metabolic-risk groups, although effect sizes are inconsistent and often context-specific [12,13]. Despite the high prevalence of vitamin D deficiency in Bangladesh, contemporary evidence evaluating serum 25(OH)D in relation to a comprehensive panel of routinely available inflammatory biomarkers among Bangladeshi adults remains limited. Existing studies often focus on single markers, specific diseases, or populations outside South Asia, thereby restricting the applicability of findings to urban Bangladeshi contexts where cardiometabolic comorbidities and low-grade inflammation are prevalent. This study therefore aimed to assess the association between serum 25-hydroxyvitamin D concentrations and systemic inflammatory biomarkers among adults attending NITOR in Dhaka, Bangladesh, while accounting for seasonality and behavioral determinants such as sun exposure and physical activity that influence both vitamin D status and inflammation.

METHODS & MATERIALS

This analytical cross-sectional study was conducted over a 12-month period (2023 to 2024) in the outpatient and inpatient departments of National Institute of Traumatology and Orthopaedic Rehabilitation & National Institute of Kidney Diseases and Urology, Dhaka, Bangladesh. Adults aged 18 years and above attending the hospital for routine care or minor medical complaints were screened consecutively and enrolled in the study. Exclusion criteria included pregnancy, acute febrile illness or clinically apparent infection within the preceding two weeks, known chronic inflammatory or autoimmune disease, active malignancy, chronic liver failure, advanced chronic kidney disease, current high-dose vitamin D therapy, and use of systemic corticosteroids or other immunosuppressive drugs, as these conditions may substantially alter inflammatory biomarkers. The target sample size was 184, calculated to detect a moderate correlation ($|r| = 0.25$) between serum 25(OH)D and inflammatory biomarkers with 90% power at a two-sided alpha of 0.05, incorporating approximately 10% non-evaluable laboratory or data loss. Data were collected using a structured case record form capturing socio-demographic variables (age, sex, education, occupation), lifestyle exposures (smoking, physical activity, habitual sun exposure, dietary pattern), and clinical history including comorbidities (diabetes, hypertension, dyslipidemia) and medication or supplement use. Anthropometry will be measured using standardized procedures, including weight and height for BMI calculation. Season of sampling was recorded, winter versus summer or monsoon, to address expected seasonal variation in vitamin D. Venous blood was collected under aseptic precautions. Serum 25(OH)D was measured using a validated immunoassay or chemiluminescent method in the hospital laboratory with internal quality control. Inflammatory biomarkers included hsCRP, ESR, total WBC count, differential counts to derive neutrophil–lymphocyte ratio and platelet–lymphocyte ratio, monocyte count or lymphocyte percentage, albumin, and uric acid. Units and reference ranges will be standardized before analysis. Vitamin D status was categorized as deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥30 ng/mL). Data was analyzed using SPSS (v. 26.0). Continuous variables were summarized as mean ± SD or median (IQR) based on distribution, categorical variables as frequency and percentage. Group comparisons across vitamin D categories were ANOVA or Kruskal–Wallis tests, with appropriate post-hoc testing. Associations between serum 25(OH)D and biomarkers were assessed using Pearson or Spearman correlation. Multivariable linear regression models were used to estimate adjusted associations between 25(OH)D and key biomarkers, with log transformation for skewed outcomes (for example, hsCRP, NLR), adjusting for age, sex, BMI, smoking, sun exposure, physical activity, season, comorbidities, and supplementation. Statistical significance was set at $p<0.05$. Ethical approval was obtained from the institutional review board, and all data were de-identified and stored securely.

RESULTS

Table 1 shows that the 184 urban hospital-based adults were evenly split by sex (92 male, 92 female), with a mean age of 41.6 ± 12.8 years and a mean BMI of 25.1 ± 4.3 kg/m². Just over half had college-level education or higher (53.3%), 41.3% were employed, and 23.9% were current smokers. Low physical activity (58.7%) and limited sun exposure, defined as <30 minutes per day (60.9%), were common; 12.0% reported vitamin D supplementation. Comorbidities were frequent: diabetes 22.3%, hypertension 28.3%, dyslipidemia 29.9%, with most samples collected during summer or monsoon (66.3%) *Table I.*

Table I

Socio-demographic and clinical characteristics of participants (*n* = 184).

Variable	Category	n (%) or mean ± SD
Age (years)		41.6 ± 12.8
BMI (kg/m²)		25.1 ± 4.3
Sex	Male	92 (50)
	Female	92 (50)
Education	≤ Secondary	86 (46.7)
	College or higher	98 (53.3)
Employment	Employed	76 (41.3)
	Unemployed	108 (58.7)
Smoking status (Current smoker)		44 (23.9)
Physical activity (Low)		108 (58.7)
Sun exposure (<30 min/day)		112 (60.9)
Vitamin D supplementation		22 (12.0)
Comorbidity	Diabetes mellitus	41 (22.3)
	Hypertension	52 (28.3)
	Dyslipidemia	55 (29.9)
Season of sampling	Winter	62 (33.7)
	Summer or monsoon	122 (66.3)

Table II summarizes the biochemical profile: mean serum 25(OH)D was 19.4 ± 7.6 ng/mL (range 4.8–44.2), while inflammatory markers were modestly elevated overall, hsCRP median 3.1 mg/L (IQR 1.7–5.9) and ESR 18 mm/hr (IQR 10–30). Hematologic indices were within expected adult ranges, WBC 7.5 ± 1.9 ×10⁹/L, NLR 2.3 (1.7–3.2), PLR 134 (105–176), monocyte count 0.48 ± 0.16 ×10⁹/L, lymphocyte percentage 32.5 ± 7.8%. Albumin was 4.0 ± 0.4 g/dL, and uric acid 5.6 ± 1.4 mg/dL.

Table II
Distribution of serum 25-hydroxyvitamin D and inflammatory biomarkers (overall).

Parameter	Unit	Mean ± SD or Median (IQR)	Min-Max
Serum 25(OH)D	ng/mL	19.4 ± 7.6	4.8-44.2
CRP (hsCRP)	mg/L	3.1 (1.7–5.9)	0.2-28
ESR	mm/hr	18 (10–30)	2-90
Total WBC count	×10⁹/L	7.5 ± 1.9	3.2-13.6
Neutrophil–lymphocyte ratio (NLR)	ratio	2.3 (1.7–3.2)	0.8-8.5
Platelet–lymphocyte ratio (PLR)	ratio	134 (105–176)	55-340
Monocyte count	×10⁹/L	0.48 ± 0.16	0.12-1.15
Lymphocyte percentage	%	32.5 ± 7.8	12-52
Albumin	g/dL	4.0 ± 0.4	2.8-5
Uric acid	mg/dL	5.6 ± 1.4	2.4-10.2

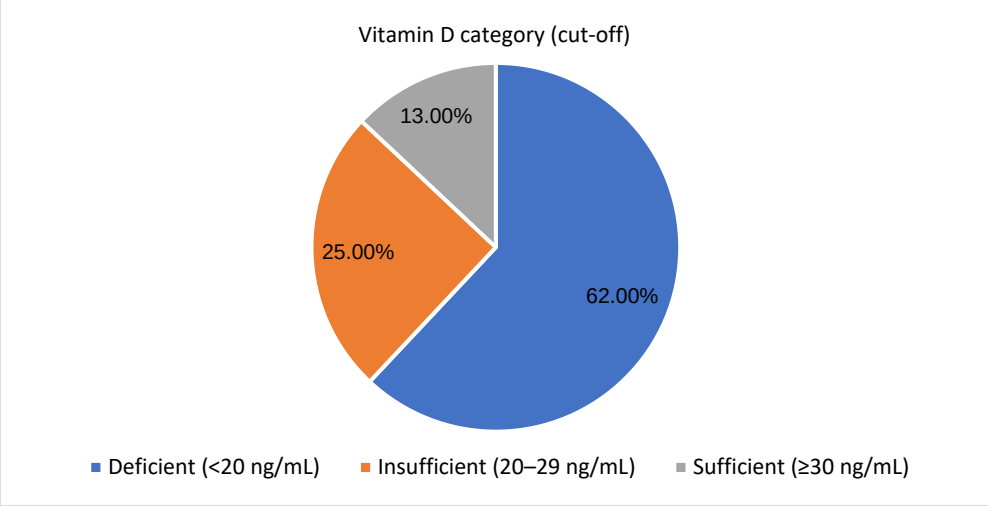

Figure 1 Vitamin D status categories among participants.

Figure 1 indicates a high burden of low vitamin D status, 62% were deficient (<20 ng/mL), 25% insufficient (20–29 ng/mL), and only 13% had sufficient levels (≥30 ng/mL), consistent with an overall mean below 20 ng/mL.

Table III demonstrates a graded pattern across vitamin D categories, participants with deficiency had higher inflammatory markers than those with sufficiency, hsCRP 3.7 (2.0–7.0) vs 2.0 (1.1–3.8) mg/L (p<0.001), ESR 20 (12–34) vs 12 (7–22) mm/hr (p=0.002), NLR 2.6 (1.9–3.6) vs 1.8 (1.3–2.5) (p<0.001), and PLR 145 (112–188) vs 118 (95–150) (p=0.004). Albumin showed the opposite direction, lower in deficiency, 3.96 ± 0.41 vs 4.14 ± 0.36 g/dL (p=0.012). WBC, monocyte count, lymphocyte percentage, and uric acid did not differ significantly by vitamin D status.

Table III
Inflammatory biomarkers by vitamin D status category.

Biomarker	Deficient (n = 114)	Insufficient (n = 46)	Sufficient (n = 24)	p-value
CRP (hsCRP) (mg/L)	3.7 (2.0–7.0)	2.8 (1.5–5.3)	2.0 (1.1–3.8)	<0.001
ESR (mm/hr)	20 (12–34)	16 (9–28)	12 (7–22)	0.002
Total WBC count (×10⁹/L)	7.6 ± 1.9	7.4 ± 1.8	7.2 ± 1.7	0.29
NLR (ratio)	2.6 (1.9–3.6)	2.2 (1.6–3.0)	1.8 (1.3–2.5)	<0.001
PLR (ratio)	145 (112–188)	134 (105–170)	118 (95–150)	0.004
Monocyte count (×10⁹/L)	0.50 ± 0.16	0.47 ± 0.15	0.44 ± 0.14	0.08
Lymphocyte percentage (%)	31.6 ± 7.7	32.9 ± 7.6	34.4 ± 7.5	0.1
Albumin (g/dL)	3.96 ± 0.41	4.05 ± 0.38	4.14 ± 0.36	0.012
Uric acid (mg/dL)	5.7 ± 1.4	5.6 ± 1.3	5.3 ± 1.2	0.18

Table IV supports these patterns with correlation analysis: serum 25(OH)D showed statistically significant inverse correlations with hsCRP (ρ = -0.26), ESR (ρ = -0.22), NLR (ρ = -0.28), PLR (ρ = -0.20), and a positive correlation with albumin (r = 0.19), all with p≤0.030. Correlations with WBC (r = -0.08) and uric acid (r = -0.12) were weak and not statistically significant.

Table IV
Correlation between serum 25(OH)D concentration and inflammatory biomarkers.

Biomarker	Correlation (r or ρ)	95% CI	p-value
hsCRP	ρ = -0.26	-0.39 to -0.12	<0.001
ESR	ρ = -0.22	-0.35 to -0.08	0.003
NLR	ρ = -0.28	-0.41 to -0.14	<0.001
PLR	ρ = -0.20	-0.33 to -0.06	0.006
Albumin	r = 0.19	0.05 to 0.33	0.01
WBC	r = -0.08	-0.22 to 0.07	0.28
Uric acid	r = -0.12	-0.26 to 0.03	0.10

Table V confirms that the associations persist after adjustment: for every 10 ng/mL increase in 25(OH)D, ln(hsCRP) decreased by 0.18 (95% CI -0.28 to -0.08, p<0.001), ESR decreased by 2.6 mm/hr (95% CI -4.8 to -0.4, p=0.020), and ln(NLR) decreased by 0.12 (95% CI -0.19 to -0.05, p=0.001), while albumin increased by 0.07 g/dL (95% CI 0.02 to 0.12, p=0.006), indicating an independent inverse relationship between vitamin D status and systemic inflammatory burden in this adult cohort.

Table V
Adjusted association, multivariable regression models (key predictors only).

Outcome	Vitamin D term	β (unstd.)	95% CI	p-value
ln(hsCRP)	25(OH)D per 10 ng/mL	-0.18	-0.28 to -0.08	<0.001
ESR	25(OH)D per 10 ng/mL	-2.6	-4.8 to -0.4	0.020
ln(NLR)	25(OH)D per 10 ng/mL	-0.12	-0.19 to -0.05	0.001
Albumin	25(OH)D per 10 ng/mL	0.07	0.02 to 0.12	0.006

DISCUSSION

Within this urban, hospital-based adult cohort in Dhaka, vitamin D deficiency was highly prevalent, and lower serum 25(OH)D concentrations were consistently associated with increased systemic inflammatory burden. Individuals with deficiency exhibited significantly higher hsCRP, ESR, NLR, and PLR, as well as lower albumin. These associations remained statistically significant after adjustment for age, sex, BMI, smoking, sun exposure, physical activity, season, comorbidities, and supplementation. The observed graded differences across vitamin D status categories, along with modest but significant inverse correlations and the persistence of associations in multivariable models, collectively indicate an independent, dose-related relationship between vitamin D status and low-grade inflammation in adults. These findings align with population-level evidence indicating an inverse association between 25(OH)D and inflammatory activity, particularly CRP. For example, an NHANES analysis by Amer and Qayyum demonstrated an inverse relationship between serum 25(OH)D and CRP, with non-linear patterns and a more pronounced gradient at lower vitamin D levels. This is consistent with the present cohort, where mean 25(OH)D was below 20 ng/mL and deficiency were predominant [14]. Disease-stratified NHANES analyses have also reported inverse associations between CRP and 25(OH)D across multiple conditions, supporting the notion that the vitamin D–inflammation relationship is not limited to a single diagnostic group [15]. In postmenopausal women, higher 25(OH)D concentrations have been associated with lower CRP, reinforcing cross-cohort consistency and suggesting that the inverse association may be observed across sex and age strata, although effect sizes may vary [16]. Collectively, these studies support the observation that lower vitamin D status is associated with higher hsCRP and ESR, which are commonly used markers in clinical practice. A clinically relevant aspect of this study is the concordant relationship between 25(OH)D and accessible hematologic indices, particularly NLR and PLR. These ratios are increasingly utilized as cost-effective proxies for systemic inflammation and immune balance. The data demonstrated higher NLR and PLR in individuals with deficiency, inverse correlations with 25(OH)D, and an independent inverse association for ln (NLR) in adjusted models. These findings are consistent with those of Akbas et al., who reported significant inverse associations between vitamin D levels and both NLR and PLR, highlighting the utility of these ratios as inflammation surrogates in the context of vitamin D insufficiency or deficiency [17]. Evidence of similar relationships in younger populations further supports biological plausibility; a large pediatric analysis reported inverse associations between 25(OH)D and hsCRP as well as other inflammatory biomarkers, suggesting that vitamin D status may reflect systemic inflammatory tone throughout the life course, although baseline immune-metabolic contexts may differ [18]. The albumin findings further support the presence of a low-grade inflammatory phenotype in vitamin D deficiency. Albumin, a negative acute-phase reactant, exhibited an opposite pattern, which is consistent with lower inflammatory tone and potentially improved nutritional-inflammatory balance at higher vitamin D concentrations, although albumin levels are also influenced by hydration status and chronic disease burden. Interpretation of these findings should also account for the mixed evidence from interventional studies. A meta-analysis of randomized trials found that vitamin D supplementation can reduce circulating hsCRP in certain contexts, but substantial heterogeneity exists related to baseline deficiency, dosage, and follow-up duration. This suggests that benefits may be most apparent in deficient individuals or specific inflammatory states [19]. A more recent meta-analysis focusing on postmenopausal women also reported variable effects on inflammatory and cardiometabolic measures, further emphasizing heterogeneity and the importance of baseline risk and study design [13]. In addition to clinical trials, bidirectional Mendelian randomization analyses have supported a potentially causal role of vitamin D deficiency in elevating CRP, while also indicating possible reverse pathways in which inflammation or illness may reduce vitamin D levels through behavioral and metabolic mechanisms [11]. This is particularly relevant to the cross-sectional design of the present study, where temporality cannot be established and residual confounding may persist despite adjustment.

LIMITATIONS

Because this was a single-center, cross-sectional study, temporality and causality between vitamin D status and inflammatory biomarkers cannot be

established, and residual confounding may persist despite multivariable adjustment. The hospital-based, predominantly urban sample limits generalizability to the wider community, and single-time-point measurements, plus potential assay and unmeasured dietary or subclinical infection effects, may have introduced misclassification of vitamin D status and inflammatory activity.

CONCLUSION

Vitamin D deficiency was common among adults attending NITOR, Dhaka, and lower serum 25(OH)D concentrations were independently associated with higher systemic inflammatory biomarkers, including hsCRP, ESR, and NLR, with lower albumin. These findings suggest that poor vitamin D status may contribute to a low-grade inflammatory state in adults, and support considering vitamin D assessment, alongside lifestyle risk evaluation, in populations with a high burden of deficiency.

RECOMMENDATIONS

Screen and treat vitamin D deficiency in high-risk adults, alongside advice on safe sun exposure, diet, and physical activity. Conduct multicenter longitudinal studies and trials in Bangladesh to test whether raising 25(OH)D reduces hsCRP, ESR, and NLR.

FUNDING

No funding sources

CONFLICT OF INTEREST

None declared

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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