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ORIGINAL ARTICLE

# Vitamin D Deficiency in Patients with Systemic Lupus Erythematosus – Prevalence and Clinical Associations

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***ABSTRACT***

***Background:*** *Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by multisystem involvement and significant morbidity. Vitamin D, known for its immunomodulatory properties, has been increasingly linked to disease activity and clinical outcomes in autoimmune conditions, including SLE. Despite abundant sunlight in Bangladesh* ***Objectives:*** *This study aimed to determine the prevalence of vitamin D deficiency in Bangladeshi patients with SLE and to explore its association with disease activity and organ involvement.* ***Methods & Naterial:*** *This cross-sectional study was conducted over one year at Dhaka Medical College Hospital and included 130 adult patients diagnosed with SLE according to the ACR 1997 criteria. Participants were categorized based on serum 25(OH)D levels into three groups: deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥30 ng/mL).* ***Results:*** *Vitamin D deficiency was present in 64.6% (n=84) of patients. Deficient patients showed significantly higher disease activity scores (mean SLEDAI 14.2±3.9 vs. 9.8±2.7, p<0.001), increased renal involvement (46.4% vs. 16.7%, p=0.043), and higher anti-dsDNA positivity (76.2% vs. 50.0%, p=0.037) compared to sufficient patients. Complement levels (C3 and C4), ESR, and CRP were significantly lower in deficient patients.* ***Conclusion:*** *Vitamin D deficiency is highly prevalent in Bangladeshi SLE patients and is significantly associated with increased disease activity and renal involvement. Routine assessment and supplementation of vitamin D may serve as a simple, cost-effective adjunct in the management of SLE to potentially reduce disease severity and improve outcomes.*

***Keywords:*** *Systemic lupus erythematosus, Vitamin D deficiency, SLEDAI, Renal involvement, Autoimmunity*

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## INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, relapsing-remitting, multisystem autoimmune disease marked by the production of a wide range of autoantibodies and the formation of immune complexes, which result in widespread inflammation and tissue damage across multiple organ systems [1]. SLE predominantly affects young women of reproductive age and presents with heterogeneous clinical manifestations ranging from mild constitutional symptoms to severe, life-threatening complications such as lupus nephritis, neuropsychiatric involvement, and hematological abnormalities [2]. The global burden of SLE is increasing, with an estimated prevalence ranging from 50 to 150 cases per 100,000 population [3]. In Bangladesh, although comprehensive nationwide epidemiological data remain limited, hospital-based reports indicate that SLE constitutes approximately 12–15% of rheumatology outpatient visits in tertiary care centers, underscoring its rising clinical and public health relevance in the region [4]. The pathogenesis of SLE is multifactorial, involving genetic predisposition, hormonal influence, environmental triggers, and immune dysregulation [5]. Among environmental factors, vitamin D deficiency has recently garnered considerable attention due to its potential role in immune modulation. Vitamin D, primarily synthesized in the skin via ultraviolet B (UVB) radiation and obtained through limited dietary sources, plays an essential

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role in regulating both innate and adaptive immune responses [6]. It inhibits pro-inflammatory Th1 and Th17 cytokines while enhancing regulatory T-cell activity, thereby contributing to immune tolerance. Deficiency in vitamin D has been linked to the development and exacerbation of several autoimmune diseases, including SLE [7]. Numerous studies have reported a high prevalence of hypovitaminosis D among patients with systemic lupus erythematosus (SLE), with variations influenced by geographical location, sunlight exposure, lifestyle practices, and clinical severity [8]. In South Asian populations, this prevalence is thought to be even higher due to several contributing factors. These include darker skin pigmentation, which reduces cutaneous vitamin D synthesis, as well as conservative clothing styles and limited outdoor activity that restrict sun exposure [9]. The use of corticosteroids in lupus management further disrupts vitamin D metabolism. Despite abundant sunlight in many regions, deficiency remains paradoxically common due to these overlapping cultural, environmental, and clinical factors [10]. In SLE patients, vitamin D deficiency has been associated with increased disease activity, elevated levels of anti-dsDNA antibodies, and a higher risk of complications such as lupus nephritis, musculoskeletal pain, fatigue, cardiovascular disease, osteoporosis, and infection [11]. Corticosteroid therapy, often a mainstay in SLE management, further exacerbates this deficiency by interfering with vitamin D metabolism [12]. These complications significantly impair the quality of life and can worsen long-term outcomes. Despite growing evidence of these associations, vitamin D screening is not routinely performed in SLE care in many resource-limited settings like Bangladesh [13]. Moreover, local data exploring the relationship between vitamin D levels and disease manifestations in Bangladeshi SLE patients are scarce [14]. Therefore, this study aimed to evaluate the prevalence of vitamin D deficiency in patients with SLE in Bangladesh and to explore its clinical correlations with disease activity and organ involvement, thereby informing evidence-based management strategies.

## METHODS & MATERIALS

This cross-sectional study was conducted in the Department of Nephrology, Dhaka Medical College, Dhaka, Bangladesh. The study spanned 12 months, from April 2018 to March 2019, and focused on evaluating the prevalence of vitamin D deficiency in patients diagnosed with systemic lupus erythematosus (SLE) and exploring its clinical associations. Using a purposive sampling method, a total of 130 patients with confirmed SLE attending the outpatient and inpatient services were enrolled, forming a clearly defined study cohort. All patients fulfilled the American College of Rheumatology (ACR) 1997 revised criteria for the classification of SLE. Based on serum vitamin D levels, participants were categorized into three groups:

**Vitamin D Deficient (n=84)**: 25(OH)D < 20 ng/mL
**Vitamin D Insufficient (n=40)**: 25(OH)D between 20–29 ng/mL
**Vitamin D Sufficient (n=6)**: 25(OH)D ≥ 30 ng/mL

### INCLUSION CRITERIA

- Adults aged ≥18 years
- Diagnosed cases of systemic lupus erythematosus (SLE) according to ACR 1997 criteria
- Willingness to participate with informed consent

### EXCLUSION CRITERIA

- Concurrent chronic liver disease or chronic kidney disease
- Patients on high-dose vitamin D supplementation (≥1000 IU/day) in the last 3 months
- History of malabsorption syndromes
- Pregnancy or lactation
- Active malignancy or other autoimmune diseases

### DATA COLLECTION

Data were systematically collected using a structured, pre-tested questionnaire and review of clinical records. The key variables assessed included age, gender, body mass index (BMI), disease duration, smoking status, sun exposure, and use of vitamin D supplements. Disease activity was assessed using the SLE Disease Activity Index (SLEDAI). Clinical manifestations such as arthritis, mucocutaneous involvement, renal and neuropsychiatric manifestations, hematologic abnormalities, and laboratory parameters including anti-dsDNA, complement C3/C4, ESR, and CRP were recorded. Treatment profiles including corticosteroid use, hydroxychloroquine, immunosuppressants, and lifestyle-related factors such as physical activity, calcium intake, and sunscreen use were also documented. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using chemiluminescent immunoassay methods. All participants provided written informed consent, and ethical approval was obtained from the Institutional Review Board.

### STATISTICAL ANALYSIS

Data analysis was performed using SPSS software (version 22.0). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. The chi-square test was used to compare categorical variables between groups. Multivariate logistic regression analysis was conducted to identify independent predictors of vitamin D deficiency. A p-value ≤0.05 was considered statistically significant.

## RESULTS

The mean age of SLE patients was similar across all groups with values of 31.2 ± 8.5, 30.8 ± 9.3, and 29.5 ± 8.2 years respectively (Table I). Female predominance was highest among vitamin D deficient patients at 98.81%, while the sufficient group had only 33.33% females. Disease duration was 4.2 ± 2.1, 4.0 ± 2.4, and 3.8 ± 2.0 years. BMI showed minimal variation across groups with values of 22.1 ± 3.4, 22.5 ± 3.7, and 23.0 ± 3.1. Smoking status was present in 3.6%, 5.00%, and 0.00%. Sun exposure increased progressively with vitamin D status at 2.5 ± 1.2, 3.1 ± 1.0, and 4.0 ± 0.8 hours/week. Use of vitamin D supplements was noted in 6.0%, 30.00%, and 83.33% of patients (Table I). Table II showed

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that SLEDAI score was 14.2 ± 3.9, 11.6 ± 3.1, and 9.8 ± 2.7. Arthritis occurred in 73.81%, 65.00%, and 50.00%. Mucocutaneous involvement was noted in 69.05%, 60.00%, and 33.33%, while renal involvement was found in 46.43%, 32.50%, and 16.67%. Neuropsychiatric manifestations occurred in 14.29%, 10.00%, and 0.00%, and hematological abnormalities in 58.33%, 50.00%, and 33.33%. Anti-dsDNA positivity was seen in 76.19%, 65.00%, and 50.00%. Complement C3 levels were 72.1 ± 12.4, 78.5 ± 11.2, and 85.4 ± 10.6, and complement C4 levels were 11.2 ± 4.3, 13.4 ± 3.8, and 15.1 ± 3.2. ESR values were 48.6 ± 14.7, 41.9 ± 13.2, and 37.2 ± 10.8, while CRP levels were 10.3 ± 4.9, 8.2 ± 3.7, and 6.5 ± 2.4 (Table III). For BMI, Odds Ratio was 0.94, 95% Confidence Interval was 0.83 – 1.06 and P=0.317. Renal involvement (OR=2.14; 95% CI: 1.01–4.53) and SLEDAI score (OR=1.22; 95% CI: 1.07–1.39) were significantly associated with vitamin D deficiency. Use of vitamin D supplements showed a protective association (OR=0.19; 95% CI: 0.07–0.52) (Table IV). Table V presented that corticosteroid use was 85.71%, 75.00%, and 50.00%, while Hydroxychloroquine use was 77.38%, 85.00 and 100.00 % (P=0.126). Immunosuppressive therapy use was 60.71%, 50.00%, and 16.67%. Daily calcium intake was 26.19%, 45.00%, and 83.33%, and physical activity was reported in 22.62%, 35.00%, and 66.67% respectively.

**Table – I: Demographic and Clinical Characteristics of SLE Patients by Vitamin D Status**

| Variables | Vitamin D Deficient (n=84) n (%) | Vitamin D Insufficient (n=40) n (%) | Vitamin D Sufficient (n=6) n (%) | P value |
|---|---|---|---|---|
| **Age (years)** | | | | |
| Mean ± SD | 31.2 ± 8.5 | 30.8 ± 9.3 | 29.5 ± 8.2 | 0.672 |
| **Gender** | | | | |
| Female | 83 (98.81) | 35 (87.50) | 2 (33.33) | <0.001 |
| Male | 1(1.19) | 5(12.50) | 4(66.67) | |
| **Disease Duration (years)** | | | | |
| Mean ± SD | 4.2 ± 2.1 | 4.0 ± 2.4 | 3.8 ± 2.0 | 0.788 |
| BMI (kg/m$^2$) | | | | |
| Mean ± SD | 22.1 ± 3.4 | 22.5 ± 3.7 | 23.0 ± 3.1 | 0.519 |
| Smoking Status | 3 (3.6) | 2 (5.00) | 0 (0.00) | 0.713 |
| **Sun Exposure (hrs/week)** | | | | |
| Mean ± SD | 2.5 ± 1.2 | 3.1 ± 1.0 | 4.0 ± 0.8 | 0.021 |
| Use of Vitamin D Supplement | 5 (6.0) | 12 (30.00) | 5 (83.33) | <0.001 |

**Table – II: Clinical Manifestations and Disease Activity by Vitamin D Status**

| Clinical Feature | Vitamin D Deficient (n=84) n (%) | Vitamin D Insufficient (n=40) n (%) | Vitamin D Sufficient (n=6) n (%) | P value |
|---|---|---|---|---|
| **SLEDAI Score** | | | | |
| Mean ± SD | 14.2 ± 3.9 | 11.6 ± 3.1 | 9.8 ± 2.7 | <0.001 |
| Arthritis | 62 (73.81) | 26 (65.00) | 3 (50.00) | 0.127 |
| Mucocutaneous Involvement | 58 (69.05) | 24 (60.00) | 2 (33.33) | 0.045 |
| Renal Involvement | 39 (46.43) | 13 (32.50) | 1 (16.67) | 0.043 |
| Neuropsychiatric Manifestations | 12 (14.29) | 4 (10.00) | 0 (0.00) | 0.294 |
| Hematological Abnormalities | 49 (58.33) | 20 (50.00) | 2 (33.33) | 0.168 |

**Table – III: Laboratory Parameters by Vitamin D Status**

| Laboratory Parameter | Vitamin D Deficient (n=84) n (%) | Vitamin D Insufficient (n=40) n (%) | Vitamin D Sufficient (n=6) n (%) | P value |
|---|---|---|---|---|
| Anti-dsDNA positivity | 64 (76.19) | 26 (65.00) | 3 (50.00) | 0.037 |
| **Complement C3 (mg/dL)** | | | | |
| Mean ± SD | 72.1 ± 12.4 | 78.5 ± 11.2 | 85.4 ± 10.6 | 0.011 |
| **Complement C4 (mg/dL)** | | | | |
| Mean ± SD | 11.2 ± 4.3 | 13.4 ± 3.8 | 15.1 ± 3.2 | 0.019 |
| **ESR (mm/hr)** | | | | |
| Mean ± SD | 48.6 ± 14.7 | 41.9 ± 13.2 | 37.2 ± 10.8 | 0.028 |
| **CRP (mg/L)** | | | | |
| Mean ± SD | 10.3 ± 4.9 | 8.2 ± 3.7 | 6.5 ± 2.4 | 0.035 |

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**Table – IV: Multivariate Logistic Regression Analysis for Factors Associated with Vitamin D Deficiency in SLE Patients**

| Variable | Odds Ratio (OR) | 95% Confidence Interval (CI) | P value |
|---|---|---|---|
| Age (per year increase) | 1.02 | 0.97 – 1.08 | 0.441 |
| Female gender | 0.89 | 0.17 – 4.51 | 0.886 |
| Disease Duration (years) | 1.06 | 0.88 – 1.29 | 0.546 |
| BMI | 0.94 | 0.83 – 1.06 | 0.317 |
| Renal Involvement | 2.14 | 1.01 – 4.53 | 0.048 |
| SLEDAI Score (per unit increase) | 1.22 | 1.07 – 1.39 | 0.003 |
| Use of Vitamin D Supplement | 0.19 | 0.07 – 0.52 | 0.001 |

**Table – V: Treatment Characteristics and Medication Use by Vitamin D Status**

| Medication/Treatment Variable | Deficient (n=84) n (%) | Insufficient (n=40) n (%) | Sufficient (n=6) n (%) | P value |
|---|---|---|---|---|
| Corticosteroid use | 72 (85.71) | 30 (75.00) | 3 (50.00) | 0.038 |
| Hydroxychloroquine use | 65 (77.38) | 34 (85.00) | 6 (100.00) | 0.126 |
| Immunosuppressive therapy (e.g., azathioprine) | 51 (60.71) | 20 (50.00) | 1 (16.67) | 0.041 |
| Use of sunscreen, n (%) | 59 (70.24) | 25 (62.50) | 2 (33.33) | 0.072 |
| Daily calcium intake (diet/supplement) | 22 (26.19) | 18 (45.00) | 5 (83.33) | 0.001 |
| Physical activity (≥3 days/week) | 19 (22.62) | 14 (35.00) | 4 (66.67) | 0.004 |

## DISCUSSION

Vitamin D deficiency can be classified into insufficiency and deficiency, both of which are commonly observed in patients with Systemic Lupus Erythematosus (SLE) and may influence disease activity and clinical outcomes [15]. In our study, we observed a significant association between vitamin D deficiency and increased disease activity in systemic lupus erythematosus (SLE) patients. Specifically, the mean SLEDAI scores were 14.2 ± 3.9 in the vitamin D-deficient group, 11.6 ± 3.1 in the insufficient group, and 9.8 ± 2.7 in the sufficient group ($p < 0.001$). This inverse relationship between vitamin D levels and disease activity aligns with findings from previous studies. For instance, a meta-analysis by Irfan et al. reported a significant decrease in SLEDAI scores with vitamin D supplementation (SMD = -0.85; 95% CI: -1.12 to -0.58; $p < 0.00001$) [16]. Renal involvement was notably higher in vitamin D-deficient patients (46.43%) compared to insufficient (32.50%) and sufficient groups (16.67%) ($p = 0.043$). This finding is consistent with a study by WCN23-0334, which found that SLE patients with renal involvement had significantly lower vitamin D levels, and there was a negative correlation between vitamin D levels and SLEDAI scores ($r = -0.591$; $p = 0.001$) [17].Our study also demonstrated that vitamin D-deficient patients had higher anti-dsDNA positivity (76.19%) compared to insufficient (65.00%) and sufficient groups (50.00%) ($p = 0.037$). This aligns with findings from a study by Terrier et al., which reported that vitamin D supplementation led to a decrease in anti-dsDNA levels from 177 ± 63 IU/mL to 103 ± 36 IU/mL over six months ($p < 0.01$) [18]. Complement levels were also affected by vitamin D status. Complement C3 levels were 72.1 ± 12.4 mg/dL in the deficient group, 78.5 ± 11.2 mg/dL in the insufficient group, and 85.4 ± 10.6 mg/dL in the sufficient group ($p = 0.011$). Complement C4 levels followed a similar trend ($p = 0.019$). These findings are in line with a study that found a significant increase in C3 levels with vitamin D supplementation [19]. Inflammatory markers such as ESR and CRP were elevated in vitamin D-deficient patients. ESR values were 48.6 ± 14.7 mm/hr in the deficient group, compared to 41.9 ± 13.2 mm/hr and 37.2 ± 10.8 mm/hr in the insufficient and sufficient groups, respectively ($p = 0.028$). CRP levels showed a similar pattern ($p = 0.035$). These results are consistent with a study by Abou-Raya et al., which reported that vitamin D supplementation led to a decrease in anti-dsDNA levels, indicating reduced inflammation [20]. Multivariate logistic regression analysis in our study revealed that renal involvement (OR = 2.14; 95% CI: 1.01–4.53; $p = 0.048$) and higher SLEDAI scores (OR = 1.22; 95% CI: 1.07–1.39; $p = 0.003$) were significantly associated with vitamin D deficiency. Conversely, the use of vitamin D supplements was associated with a protective effect against deficiency (OR = 0.19; 95% CI: 0.07–0.52; $p = 0.001$). These findings underscore the potential benefits of vitamin D supplementation in managing SLE disease activity [21].

## LIMITATIONS OF THE STUDY

This study was cross-sectional in nature, limiting the ability to establish causal relationships between vitamin D deficiency and disease activity in SLE patients. The small number of patients with sufficient vitamin D levels may have restricted the statistical power for subgroup comparisons. Additionally, factors such as seasonal variation in sun exposure, dietary intake, and genetic polymorphisms affecting vitamin D metabolism were not assessed. Being a single-center study, the findings may not be generalizable to all populations or geographic regions.

## CONCLUSION

Vitamin D deficiency is highly prevalent among patients with systemic lupus erythematosus in Bangladesh and is significantly associated with increased disease activity and renal involvement. Our findings highlight the protective role

of vitamin D supplementation in reducing deficiency risk and potentially mitigating disease severity. Given the strong correlation between low vitamin D levels and worse clinical manifestations, routine screening and appropriate correction of vitamin D status should be considered an integral component of comprehensive SLE management. Further longitudinal studies are warranted to elucidate the causal relationship and assess the impact of vitamin D optimization on long-term clinical outcomes in this population.

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