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Vitamin D Deficiency in Systemic Lupus Erythematosus and Its Correlation with Disease Activity Assessed by SLEDAI - A Prospective Observational Study from Kashmir

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Abstract

Background: Systemic lupus Erythematosus (SLE) is a chronic multisystem autoimmune disease characterized by periods of disease activity and remission.

Vitamin D has important Immunomodulatory functions, and vitamin D deficiency is frequently reported in patients with SLE. However, the relationship between vitamin D status and disease activity remains inconsistent across populations. The present study evaluated vitamin D status in patients with SLE and its association with disease activity assessed using the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI).

Methods: This prospective observational study was conducted in the Department of Internal Medicine, Division of Rheumatology, Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Srinagar, from April 2022 to April 2024. Two hundred patients fulfilling the 2019

EULAR/ACR classification criteria for SLE and 200 age- and sex-matched healthy controls were enrolled. Clinical examination and laboratory investigations including complete blood count, erythrocyte sedimentation rate, C-reactive protein, renal and liver function tests, routine urine examination, ANA, anti-ds DNA, C3, C4 and serum 25-hydroxyvitamin D [25 (OH) D] were performed. Vitamin D was categorized as deficient (<20 ng/mL), insufficient (20–30 ng/mL) and sufficient (≥30 ng/mL). SLE disease activity was assessed using the SELENA-SLEDAI score. Statistical analyses were performed using SPSS version 29.0 and R.

Results: The mean age of patients was 31.3±7.7 years (range 15–54 years), and 92.5% were female. Most participants (82%) were aged 21–40 years, and 74% were from rural areas. Hematologic involvement was the most frequent clinical domain (86%), followed by mucocutaneous (85%) and musculoskeletal involvement (79%). Patients had significantly lower hemoglobin, lymphocyte and platelet counts than controls. Mean albumin and total protein were also significantly lower in patients. Vitamin D below 30 ng/mL was found in 86.1% of patients compared with 86.6% of controls. Among patients, 60.5% had vitamin D <20 ng/mL, 26% had levels of 20–30 ng/mL and 13.5% had levels ≥30 ng/mL. SLEDAI scores indicated mild activity in 7.5%, moderate activity in 41.5%, high activity in 46%, and very high activity in 5% of patients. A significant association was observed between vitamin D deficiency and SLEDAI category (p=0.042). Vitamin D deficiency was also significantly associated with SPF >30 use, CRP elevation, anti-ds DNA positivity, and low C3 and C4 levels.

Conclusion: Vitamin D deficiency was highly prevalent among patients with SLE and was significantly associated with greater disease activity. The findings support routine assessment of vitamin D status in SLE, while recognizing that the relationship is associative and does not by itself establish causality.

Keywords: systemic lupus Erythematosus, vitamin D deficiency, 25 - Hydroxy vitamin D, SLEDAI, disease activity, autoimmune disease.

Introduction

Systemic lupus Erythematosus (SLE) is a complex, relapsing-remitting inflammatory autoimmune disease affecting various organ systems, resulting in numerous clinical and serological consequences[1]. The disease course is unpredictable, with alternating phases of relapse and remission. Common symptoms include photosensitivity, facial rash, oral ulcers, arthritis, and fatigue, with a nine-fold higher incidence in females, particularly those of child-bearing age and of African-American or Asian descent[2]. The presentation and progression of SLE have been linked to a combination of environmental, genetic, and hormonal factors [3,4].

One such environmental factor gaining increasing attention is vitamin D. The identification of vitamin D receptors on cells of the immune system and the discovery that dendritic cells can produce the metabolically active form of vitamin D, 1,2 –di Hydroxy vitamin D, have led to the suggestion that vitamin D acts as an immune modulator[5]. Given that vitamin D deficiency-related symptoms such as fatigue are frequently observed in SLE patients[6], this relationship has become a subject of intense research interest.

Decreased levels of serum 25 (OH) D have been documented in SLE patients across various geographic locations and latitudes[7,10]. Multiple risk factors have

been associated with this deficiency, including sun avoidance due to photosensitivity, lupus nephritis (LN) leading to decreased 1-alpha-hydroxylase activity on 25(OH)D, use of drugs like steroids and anticonvulsants that enhance 25(OH)D metabolism, and the presence of auto antibodies against vitamin D that contribute to its clearance[11,12]. Munger et al. demonstrated that individuals with high 25 (OH) D levels (100 nmol/L) have a 62% lower risk of developing SLE [13].

In vitro studies have shown that 1,25-dihydroxyvitamin D can prevent differentiation of dendritic cells and modulate T cell phenotype and function[14]. It can inhibit T cell proliferation and cytokine production, inhibit proliferation of activated B cells, and impair generation of plasma cells[15,16]. Differentiation of dendritic cells and subsequent production of type I interferon is important in the pathogenesis of SLE [17]. Therefore, by affecting the immune system, vitamin D may play a preventive and modulatory role in SLE.

Some studies have suggested that vitamin D concentrations are inversely related to SLE activity [18,19]. In addition to 25 (OH) D, 1, 25 (OH) $_2$ D has also been found to be lower in SLE patients, with deficiency particularly associated with overweight patients [20]. Seasonal variation of vitamin D status has also been observed in SLE patients, with lower levels during the cold season compared to the warm season [21].

An earlier report highlighted vitamin D_3 insufficiency in two-thirds and deficiency (<20 ng/mL) in approximately one-fifth of SLE patients[22]. Furthermore, serum vitamin D_3 (25-OH) levels have been found to correlate inversely with SLEDAI scores[23]. Since no study has been conducted in the Kashmir valley on vitamin D levels in SLE patients, and given that studies from other parts of the world have shown a high prevalence of vitamin D deficiency in SLE patients with some demonstrating an inverse correlation with disease activity, this study was undertaken to determine vitamin D levels and their correlation with disease activity in SLE patients in our population.

Materials and Methods

Study design and setting

This was a prospective observational study conducted in the Department of Internal Medicine, Division of Rheumatology, Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar, a tertiary-care university hospital serving the Kashmir Valley and surrounding areas. The study period extended from April 2022 to April 2024. Institutional ethical clearance was obtained and informed consent was obtained from all participants.

Study population

A total of 200 patients with SLE and 200 healthy controls were enrolled. Controls were age- and sex-matched healthy individuals from the general population and were clinically screened for SLE.

Diagnostic criteria

Patients were classified as having SLE according to the 2019 EULAR/ACR classification criteria, which require a positive ANA entry criterion followed by weighted clinical and immunological domains, with a total score of ≥10 points required for classification. The 2019 criteria have demonstrated high sensitivity and specificity in validation cohorts.

Inclusion criteria

Patients fulfilling the 2019 EULAR/ACR criteria for SLE were eligible. Age- and sex-matched healthy controls were included for comparison.

Exclusion criteria

The thesis excluded ANA-negative SLE, chronic kidney disease, chronic liver disease, vitamin D

supplementation, and use of Vitamin-D metabolism-inducing drugs such as phenytoin and rifampicin. Healthy controls with a family history of SLE were excluded.

Clinical and laboratory assessment

All participants underwent detailed history and clinical examination. Investigations included CBC, ESR, CRP, kidney and liver function tests, routine urine examination, ANA, anti – ds DNA, C3, C4 and serum vitamin D. Vitamin D was assessed using chemilumine scence/ ELISA methodology according to the study protocol.

Vitamin D classification

Vitamin D status was categorized as:

- **Deficient:**<20 ng/mL
- **Insufficient:** 20–30 ng/mL
- **Sufficient:** ≥30 ng/mL.

Assessment of disease activity

Disease activity was evaluated using the Selena-Sledai system. Activity categories used in the study were:

SLEDAI score	Disease activity
0	No activity
1–5	Mild
6–10	Moderate
11–19	High
≥20	Very high

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 29.0 and R. Continuous variables were summarized as mean ± SD and categorical variables as frequency and percentage. Independent t-test or Mann–Whitney U-test was applied to continuous variables, while chi-square or Fisher's exact test was used for categorical variables. A p value <0.05 was considered statistically significant.

Results

Table 1: Demographic characteristics of SLE patients

Variable	Result
Sample size	200
Mean age ± SD	31.3 ± 7.7 years
Age range	15–54 years
Age 21–30 years	81 (40.5%)
Age 31–40 years	83 (41.5%)
Female sex	185 (92.5%)
Rural residence	148 (74.0%)

The cohort consisted predominantly of young adults, with 82% between 21 and 40 years, and showed marked female predominance. The thesis summary further confirms a mean age of 31.3 years, female proportion of 92.5%, and rural residence in 74% of participants.

The demographic profile is characteristic of SLE, with the disease predominantly observed among women in the reproductive age group.

Table 2: Major organ-system involvement

Organ system	Patients (%)
Hematologic	86
Mucocutaneous	85
Musculoskeletal	79
Gastrointestinal	24
Neuropsychiatric	14.5
Pleuropulmonary	3.5

Hematologic, mucocutaneous and musculoskeletal involvement constituted the dominant clinical phenotype.

Table 3: Comparison of hematologic parameters between cases and controls

Parameter	Cases, mean±SD	Controls, mean±SD	p value
Hemoglobin	11.0±0.52	13.2±0.8	0.003

(g/dL)			
Hematocrit (%)	35±6.3	42±3.2	0.091
MCV	80±7	90±5	0.081
Neutrophils (×10³/µL)	3.2±0.9	3.5±0.4	0.102
Lymphocytes (×10³/µL)	2.06±0.4	2.56±0.6	0.042
Platelets (×10³/µL)	105±75	145±54	0.040

Hemoglobin, lymphocyte count and platelet count were significantly lower among SLE patients than controls, consistent with the prominent hematologic involvement of the cohort.

Table 4: Biochemical profile of cases and controls

Parameter	Cases, mean±SD	Controls, mean±SD	p value
Bilirubin	1.27±0.78	1.22±0.80	0.520
ALT	46±10.2	44±9.6	0.061
ALP	93±59.8	96±61.2	0.620
Total protein	6.47±0.59	7.11±0.48	<0.001
Albumin	3.40±0.45	3.82±0.51	<0.001
Urea	41.02±8.13	39.11±8.23	0.020
Creatinine	1.11±0.22	0.99±0.14	<0.001
Corrected calcium	8.59±0.61	8.50±0.61	0.141

SLE patients had significantly lower serum albumin and total protein and higher urea and creatinine than controls, indicating clinically relevant biochemical abnormalities.

Table 5: Vitamin D status in SLE patients and controls

Vitamin D category	SLE patients	Controls
<30 ng/mL	173 (86.1%)	174 (86.6%)
≥30 ng/mL	27 (13.4%)	26 (13.0%)

Mean vitamin D among individuals with levels <30 ng/mL was 14.8±8.1 ng/mL in cases and 13.5±7.6 ng/mL in controls (p=0.001).

Vitamin D insufficiency/deficiency was extremely common in both groups. Although the numerical difference in mean values between cases and controls was small, the study reported a statistically significant difference.

Table 6: Stratified vitamin D levels

Vitamin D level	Cases n (%)	Controls n (%)
<20 ng/mL	121 (60.5%)	135 (67.5%)
20–30 ng/mL	52 (26.0%)	39 (19.5%)
≥30 ng/mL	27 (13.5%)	26 (13.0%)

More than half of the SLE cohort had vitamin D concentrations below 20 ng/mL, demonstrating a substantial burden of overt deficiency.

Table 7: Distribution of SLEDAI scores according to vitamin D status

Sledai score	Vitamin D deficient	Vitamin D normal
1–5	12	3
6–10	68	15
11–19	86	6
>19	7	3
Total	173	27

p=0.042.

The overall SLEDAI distribution was 7.5% mild activity, 41.5% moderate activity, 46% high activity and 5% very high activity.

Higher disease activity was disproportionately represented among vitamin D-deficient patients, with a statistically significant association between SLEDAI category and vitamin D status.

Table 8: Factors associated with vitamin D deficiency

Factor	Chi-square	p value	Interpretation
Photosensitivity	2.784	0.095	Not significant
Malar rash	1.224	0.269	Not significant
SPF >30 use	0.114	0.035	Significant
ESR >20	0.603	0.437	Not significant
CRP >6	4.342	0.037	Significant
Anti - ds DNA positive	1.244	0.023	Significant
Low C3	0.011	0.001	Significant
Low C4	0.110	0.025	Significant
Wysolone use	0.046	0.803	Not significant
Lupus nephritis	1.289	0.256	Not significant

Low vitamin D was significantly associated with high-SPF sunscreen use, elevated CRP, anti-ds DNA positivity and low complement levels, whereas photosensitivity, Malar rash, steroid use and lupus nephritis were not significantly associated in this cohort.

Discussion

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease characterized by a wide spectrum of clinical manifestations, ranging from mild to life-threatening, affecting multiple organ systems. It predominantly affects women of reproductive age, with complex etiological factors including genetic predisposition, hormonal influences, and environmental triggers. Despite advancements in understanding SLE, the disease continues to pose significant challenges in diagnosis and management due to its heterogeneous presentation. The present study aimed to explore the demographic, clinical, and biochemical characteristics of SLE patients, with a particular focus on the impact of vitamin D deficiency, a factor increasingly recognized for its potential role in modulating immune responses and influencing disease activity.

Demographic Characteristics

The age distribution of the study population revealed that the majority of patients (82%) fell within the 21-40 years age range, with a mean age of 31.3 ± 7.7 years. This is consistent with existing literature, which indicates that SLE predominantly affects individuals in their reproductive years. Pons-Estel et al. (2017) highlighted that SLE commonly presents in young adults, particularly women, due to the influence of hormonal factors such as estrogen, which modulates immune responses and may contribute to the onset of autoimmune diseases[38]. Furthermore, Tsokos (2020) emphasized that early adulthood is a critical period for SLE onset, aligning with the demographic characteristics observed in this study [39].

The gender distribution was notably skewed towards females, with 92.5% of the sample being women. This is consistent with the well-documented gender disparity in SLE, where females are disproportionately affected. Ortona et al. (2016) discussed that the female predominance in SLE is attributed to hormonal differences, particularly the role of estrogen in promoting immune system activity, which may increase susceptibility to autoimmune conditions[40]. Zandman-Goddard et al. (2020) also reported similar gender ratios in SLE studies, further supporting the findings of this study [41].

The study found that a significant proportion of patients (74%) were from rural areas, which may suggest

differences in healthcare access and disease management between rural and urban populations. Andrade et al. (2021) explored the impact of socioeconomic status, including rural versus urban residence, on SLE outcomes, finding that rural patients often have delayed diagnoses and poorer access to specialized care, which could contribute to differences in disease presentation and outcomes[42]. Additionally, environmental factors such as sun exposure, which can exacerbate SLE symptoms, may differ between rural and urban settings, as discussed by Lim et al. (2023) [43].

Organ System Involvement

The data revealed that hematologic (86%), mucocutaneous (85%), and musculoskeletal (79%) systems were the most commonly affected in the study population. These findings are consistent with those reported by Kaul et al. (2016), who found that hematologic abnormalities, including anemia and leukopenia, are frequent in SLE patients due to immune-mediated destruction of blood cells[44]. The high prevalence of mucocutaneous involvement aligns with the typical clinical presentation of SLE, which often includes photosensitivity and Malar rash, as discussed by Fanouriakis et al. (2020)[45].

Musculoskeletal manifestations, such as arthritis, are also commonly reported in SLE and contribute to the chronic morbidity associated with the disease.

Hematologic and Biochemical Parameters

The study identified significant differences in hematologic parameters between SLE patients and controls, including lower hemoglobin levels, platelet counts, and lymphocyte counts in SLE patients. These findings are in line with those reported by Tektonidou et al. (2021), who noted that hematologic abnormalities are a hallmark of SLE, often correlating with disease activity[46]. The reduction in lymphocyte count observed in this study is particularly relevant, as Yap and Lai (2015) highlighted the role of lymphocyte dysregulation in the pathogenesis of SLE, contributing to the autoimmune response that characterizes the disease [47].

Biochemical analysis revealed significantly lower albumin and total protein levels and higher urea and creatinine levels in SLE patients compared to controls, indicating hepatic and renal involvement. These findings are consistent with the systemic nature of SLE and its potential to affect multiple organ systems.

Vitamin D Deficiency

The high prevalence of vitamin D deficiency in both the study and control groups (86.1% and 86.6%, respectively) is consistent with findings from other studies, such as Zold et al. (2021), who reported widespread vitamin D deficiency among SLE patients [48]. An analytical study from South Asia by Kavadichanda et al. (2023) showed that patients from North India (26.85°N) had lower vitamin D levels compared to their South Indian counterparts (11.94°N), signifying that sun exposure at higher northern latitudes is lower, resulting in higher vitamin D deficiency in the north [34].

The significantly lower vitamin D levels in SLE patients (14.8 ng/mL) compared to controls (13.5 ng/mL, p=0.001) further underscore the potential role of vitamin D in modulating immune responses and disease activity. Sundaram et al. (2022) found similar associations between low vitamin D levels and increased SLE disease activity, suggesting that vitamin D supplementation could be a beneficial adjunct therapy in managing SLE [49].

The distribution of SLEDAI scores in the study population indicates that most patients have moderate to high disease activity, with 46% scoring between 11-19

and 41.5% scoring between 6-10. This is consistent with the findings of Kamen et al. (2019), who noted that SLEDAI scores are an effective measure of disease activity, with higher scores correlating with more severe clinical manifestations[50]. The association between higher SLEDAI scores and vitamin D deficiency observed in this study further supports the findings of Amital et al. (2020), who reported that vitamin D deficiency is linked to increased disease activity in SLE, possibly due to its effects on the immune system [51].

Factors Associated with Vitamin D Deficiency

The study found significant associations between vitamin D deficiency and specific clinical features, including the use of high-SPF sunscreen, elevated CRP levels, the presence of anti-ds DNA antibodies, and low complement levels. These associations are supported by Kamen et al. (2019), who discussed the impact of sun avoidance behaviors, such as the use of high-SPF sunscreen, on vitamin D synthesis and subsequent deficiency in SLE patients [50]. The correlation between vitamin D deficiency and elevated CRP levels is also in line with the findings of Amital et al. (2020), who highlighted the role of vitamin D in modulating inflammation, with lower levels potentially exacerbating inflammatory markers like CRP[51]. The significant association of anti - ds DNA antibodies and low complement levels with vitamin D deficiency suggests that vitamin D may influence immune regulation in SLE, as discussed by Sundaram et al. (2022) [49].

Clinical Implications

The findings of this study have several important clinical implications:

Screening for vitamin D deficiency

Given the high prevalence of vitamin D deficiency in SLE patients, routine screening for serum 25(OH) D levels should be considered in all SLE patients, particularly those with high disease activity.

Vitamin D supplementation

Vitamin D supplementation may be beneficial in SLE patients with deficiency, not only for bone health but also potentially for modulating disease activity. However, the optimal dose and duration of supplementation require further investigation.

Sun protection counseling

Patients should be counseled about the importance of sun protection while being made aware of the risk of vitamin D deficiency. A balanced approach to sun exposure and supplementation is necessary.

Monitoring of disease activity

Vitamin D levels should be monitored alongside other disease activity markers, and correction of deficiency may be considered as part of a comprehensive management plan.

Limitations

This study has several limitations. First, as a single-centre study from a tertiary care referral centre, the findings may not be generalizable to the community setting. Second, the cross-sectional design precludes establishing causality between vitamin D deficiency and disease activity.

Third, dietary vitamin D intake and sun exposure duration were not quantified, which could have influenced vitamin D levels. Fourth, the control group also had a high prevalence of vitamin D deficiency, which may reflect the general population's vitamin D status in the region and limits the ability to attribute deficiency solely to SLE. Fifth, follow-up data on the effect of vitamin D supplementation on disease activity were not available.

Conclusion

Vitamin D deficiency is highly prevalent in SLE patients in the Kashmir valley and is significantly associated with higher disease activity as measured by SLEDAI. The study population consisted mainly of young, rural-dwelling females, reflecting the demographic trend of SLE. Vitamin D deficiency was significantly linked to higher CRP levels, the presence of anti – ds DNA antibodies, and low complement levels, indicating a possible connection between vitamin D status and immune dysregulation. The use of high-SPF sunscreen was significantly associated with vitamin D deficiency, highlighting a potential risk factor that could be addressed in patient management. Regular monitoring of vitamin D levels and appropriate supplementation may be beneficial in the management of SLE to prevent disease flares. Further longitudinal studies are needed to establish the causal relationship between vitamin D deficiency and SLE disease activity and to determine the optimal supplementation strategies.

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