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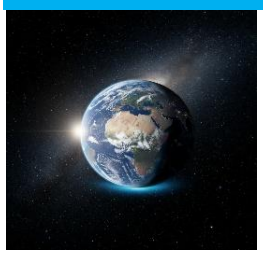

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Vitamin D Management in Cutaneous Lupus Erythematosus: Balancing Photoprotection and Immunomodulation

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Abstract

Background: Cutaneous lupus erythematosus (CLE) is an autoimmune inflammatory disorder which demands strict photoprotections, leading to vitamin D deficiency. This narrative review evaluates the interplay between vitamin D status, disease activity, and modern management challenges.

Methods: A comprehensive literature search was conducted across PubMed, EMBASE, and Scopus databases for studies published up to January 2026, focusing on CLE, vitamin D metabolism, and clinical outcomes.

Results: Vitamin D insufficiency is almost universal for all CLE patients. The etiology of this insufficiency is multifactorial, including therapy involving antimalarial agents, sun-avoidance, or sunscreen use. It correlates with higher disease activity, while supplementation significantly reduces Cutaneous Lupus Erythematosus Disease Area and Severity Index- Activity (CLASI-A) scores and shortens the duration of active skin lesions. Current 2026 vitamin D supplementation protocols for the general population may not be enough for CLE patients.
Conclusions: Vitamin D should become an integral component of CLE management. Regular monitoring and individualized supplementation are crucial to overcome pharmacological and behavioral barriers. The chronological gap in existing literature suggests that there is a notable need for new randomized clinical trials that align with 2026 therapeutic standards. It would facilitate the development of supplementation protocols tailored to CLE patients.

Key words: Systemic lupus erythematosus; Dermatoses; Photodermatoses, Vitamin D; Vitamin D deficiency, Photoprotection, Immunomodulation

1. INTRODUCTION

Cutaneous lupus erythematosus (CLE) is an autoimmune inflammatory disorder with a broad spectrum of clinical manifestations. It may occur as a component of systemic lupus erythematosus (SLE), frequently it manifests as a skin-limited entity. [1] CLE is categorized into three subtypes according to Gilliam classification: acute cutaneous lupus erythematosus (ACLE), subacute cutaneous lupus erythematosus (SCLE) and chronic cutaneous lupus erythematosus (CCLE). [2]

The pathogenesis of CLE is caused by interplay between genetic predisposition, immune system dysregulation and environmental triggers. Ultraviolet (UV) radiation is recognized as the pivotal external factor that induces or exacerbates skin lesions. [1] UV exposure drives local inflammatory response by triggering apoptosis of keratinocytes that leads to release of nuclear antigens. [3] Stringent photoprotection and avoiding sun are indispensable as non-

pharmacological management for CLE. [4];[1] However, it leads to significant clinical challenges - the patients become deprived of endogenous vitamin D synthesis, which can potentially lead to chronic deficiency associated with demineralization and immunomodulatory consequences. [4] [5]

2. RESEARCH AND METHODS

To identify relevant studies investigating relation of vitamin D levels, photoprotection, and pharmacological treatment in patients with Cutaneous Lupus Erythematosus (CLE). A comprehensive literature search was conducted within PubMed and Scopus in December 2025. The search used the terms ‘Cutaneous Lupus Erythematosus’ and ‘CLE’, ‘vitamin D levels’, ‘photoprotection’, ‘25-hydroxyvitamin D’, ‘sunscreen’, ‚antimalarials’, combined into the following query: ‘Cutaneous Lupus Erythematous* AND [(‘vitamin D levels’ OR ‘photoprotection*’ OR ‘25-hydroxyvitamin D*’ OR ‘sunscreen’ OR ‚antimalarials’). This strategy yielded a wide range of publications. First, we read the abstracts and excluded articles that did not present relations between vitamin d levels, sunscreen usage, antimalarials and CLE. Secondly, the vast majority of articles concerned themselves with SLE (systemic lupus erythematosus) and not CLE, so we decided to conclude some of them into the review. This resulted in 20 articles in total, which are included in the review.

3. RESEARCH RESULTS

3.1. THE DUAL ROLE OF VITAMIN D IN LUPUS: FROM CALCIUM HOMEOSTASIS TO IMMUNOMODULATION

The primary role of vitamin D3 is in systemic calcium and phosphate homeostasis, which is essential for maintaining proper bone mineral density. [4] In patients with cutaneous lupus erythematosus (CLE), this function is impaired due to chronic glucocorticoid intake and sun-avoidance. [5]

However, vitamin D3 exerts more widespread effects. Given that nearly all of the immune cells express the vitamin D receptor (VDR), it presents immunomodulatory functions as well. [6] Recent evidence confirms that vitamin D acts as a epigenetic modulator, influencing the expression of over 200 genes involved in immune tolerance. [7]

There are several paths in the autoimmune response in CLE, in which vitamin D can act as a "molecular brake", inhibiting the inflammatory response. [6] Calcitriol suppresses the maturation of myeloid dendritic cells (mDCs), which leads to a reduced capacity to present self-antigens. [6] Vitamin D promotes the differentiation of regulatory T cells (Tregs) while simultaneously impairing the development of T helper 1 (Th1) and T helper 17 (Th17) cells. This lowers the level of interferon-gamma (IFN-γ) and interleukin-17 (IL-17) - pro-inflammatory cytokines crucial in the CLE pathogenesis. [6]

Moreover it can inhibit plasma cells formation from B cells, thereby potentially reducing the production of autoantibodies. [5] In addition, calcitriol works also within the skin, it limits the release of pro-inflammatory chemokines by keratinocytes induced by ultraviolet (UV) exposure, resulting in reduced recruitment of inflammatory cells to the skin. [8] Furthermore, emerging evidence suggests that VDR gene polymorphisms may have influence on developing systemic lupus erythematosus and modulate the clinical response to vitamin D supplementation. [9]

3.2. THE GENETIC ARCHITECTURE: VITAMIN D RECEPTOR (VDR) POLYMORPHISMS IN SLE AND CLE

Some CLE patients respond strongly to supplementation, while others remain resistant. It shows the importance of genetic predisposition. [10] The VDR gene polymorphisms, such as FokI, BsmI, and TaqI, have been identified as potential modulators of systemic lupus erythematosus (SLE) susceptibility and treatment response. [10] [11] Patients carrying certain alleles may exhibit a "functional resistance" to calcitriol, meaning that even with adequate serum 25(OH)D levels, immunological signaling remains suboptimal. This genetic heterogeneity suggests that the therapeutic window for vitamin D is dictated by the binding affinity of the VDR. [12] It leads to the conclusion that standardized approaches often fail to address the specific needs of patients with high-risk genotypes in both SLE and CLE patients. Future clinical management may incorporate genetic screening to tailor supplementation regimens, ensuring that those with low-sensitivity receptors receive higher doses to achieve the desired immunomodulatory threshold.

3.3. PREVALENCE OF VITAMIN D DEFICIENCY IN CLE PATIENTS

The prevalence of vitamin D inadequacy in patients with Cutaneous Lupus Erythematosus (CLE) is substantially high with no significant association with latitude.

In Mediterranean regions with high solar irradiance, the rates of deficiency are paradoxically high. In a cohort from Spain, approximately 95% of CLE patients were found to have serum 25(OH)D levels below 30 ng/ml. [6] Patients with CLE had a significantly higher likelihood of vitamin D insufficiency, with an odds ratio of 4.2 compared to healthy controls. [6]

In Central Europe, seasonal fluctuations play a crucial role in vitamin D levels in the general population. While healthy individuals present an increase in 25(OH)D levels during summer months, CLE patients remain deficient. Their deficiency levels reach 85,7% in summer and 97,1% in winter. [5]

Similar outcomes were noted in the Northern Hemisphere, where suboptimal 25(OH)D levels were recorded in 65,4% of patients in Ireland during the summer. [4]

Moreover, in equatorial regions characterized by high UV indexes, such as Singapore, 51% of Asian CLE patients demonstrated inadequate vitamin D levels. This leads to the conclusion that disease-related behaviors and skin phototypes may play a more significant role than environmental UV availability. [13]

3.4. FACTORS CONTRIBUTING TO HYPOVITAMINOSIS D IN CLE

The etiology of low vitamin D status in this population is multifactorial, involving behavioral, pharmacological, and constitutional factors.

The primary drivers of deficiency are strict sun avoidance and the daily application of sunscreen. [4] Sunscreens with Sun Protection Factor (SPF) as low as 8 can significantly impair cutaneous vitamin D production up to 95%. [14]

Antimalarials remain the gold standard for the treatment of cutaneous lupus erythematosus (CLE). They have been significantly associated with lower vitamin D levels. [13] Evidence suggests that antimalarials may inhibit 1α-hydroxylation of 25(OH)D, further complicating the metabolic status of CLE patients. [13] This clinical association is no longer viewed only as a statistical correlation, but as a likely interference in vitamin D metabolism. [15];[16]. This

mechanism creates a “biochemical resistance” that often cannot be overcome by standard vitamin D doses. [1]; [15]

The longer the disease duration (particularly over 5 years) the higher the odds of vitamin D deficiency. [6]; [13] Similarly, patients with Fitzpatrick skin phototypes IV–VI are at increased risk due to constitutively reduced cutaneous photosynthesis. [13]

3.5. CLINICAL OUTCOMES OF VITAMIN D REPLACEMENT

Therapeutic potential of vitamin D sufficiency in patients with Cutaneous Lupus Erythematosus extends far beyond bone health alone, as it correlates with this disease activity. [1] Evidence from longitudinal observations suggests that the management of vitamin D deficiency plays a crucial role in stabilizing disease activity over time. [1]

The benefits of vitamin D supplementation are most accurately represented in changes in Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI) activity scores. [1] A prospective longitudinal analysis revealed that oral vitamin D3 supplementation (initial dose of 1400 IU/day, followed by 400 IU maintenance) led to a marked reduction in CLASI-A scores. Over the one-year follow-up, mean scores declined significantly from 2.7±2.9 at baseline to 0.9±1.4 ($p=0.003$). [1] By comparison, CLASI activity scores remained unchanged in the control cohort, highlighting the specific impact of vitamin D3 replacement. [1]

Vitamin D restoration alters the clinical course of CLE by stabilizing the exacerbations. A significant shortening of the duration of active lesions was observed in the group supplementing vitamin D, with the mean period dropping by more than half from 118±153 days to 56±114 days ($p=0.009$), but not in the control group. [1] While the reduction in the absolute numbers of exacerbations per year showed a downward trend in treated patients, it did not reach independent statistical significance ($p=0.17$). [1] It suggests that vitamin D mitigates the severity and persistence of skin lesions rather than the initial triggering of a flare. [1]

The clinical improvement observed in objective scales is further supported by subjective assessments, which are crucial in chronic dermatological conditions.
Approximately 59% of participants in the treatment group reported that their disease status was "better" than the previous year, compared to only 24% in the untreated group and none of the

treated patients vs. 16% of the untreated ones assessed it as worse than before (p=0,01). [1] Clinical evaluations consistently matched patient reports, noting a reduction in erythema and scaling in supplemented individuals. [1]

The long-term tolerability and safety of vitamin D therapy are critical determinants of its clinical utility. Replacement regimens using cholecalciferol (vitamin D3), often combined with calcium, were found to be exceptionally well tolerated. In the longitudinal analysis, no serious adverse events, such as hypercalcemia or nephrolithiasis, were reported during the follow-up period. [1] This supports the implementation of vitamin D screening and replacement as a safe, low-cost intervention that can be easily integrated into standard CLE care.

4. DISCUSSION

The findings of this narrative review highlight a profound clinical paradox in the management of cutaneous lupus erythematosus. While ultraviolet (UV) radiation is established as the primary external factor that triggers skin lesions in CLE [17] [5], the resulting behavioral and pharmacological interventions directly contribute to severe vitamin D deficiency, potentially undermining the patient's immune tolerance. [6]

Photoprotection remains the cornerstone of CLE management, yet its impact on health is often underestimated. As demonstrated by [4] even low- level sunscreen use (SPF 8) significantly impairs the cutaneous synthesis of vitamin D3. The fact that patients in sun-rich regions remain deficient [6], proves that environmental UV availability cannot offset disease-mandated sun avoidance. This "behavioral winter" experienced by CLE patients requires a proactive approach from clinicians, moving toward individualized supplementation protocols beyond recommendations for the general population.

A noteworthy finding is the interplay between antimalarial therapy and vitamin D status. Hydroxychloroquine (HCQ) is essential for controlling CLE, yet its association with lower 1,25(OH)D levels [13] may suggest it can disrupt vitamin D metabolic pathways. [18] Patients on these medications may require higher doses of cholecalciferol or even treatment with active vitamin D analogues to achieve the desired immunomodulatory effects. This association between standard pharmacotherapy and vitamin D status may explain why some patients remain resistant to standard pharmacotherapy.

The significant reduction in CLASI activity scores and the shortening of active lesion duration [1] provide strong evidence that vitamin D is more an immunomodulatory agent than a dietary supplement. By shifting the cytokine profile from Th1/Th17 responses to Th2, vitamin D acts to modulate the underlying immunological dysregulation in CLE. [6] [19] A key clinical insight is that vitamin D status primarily modulates the persistence of active lesions rather than the periodicity of flares. [1] It suggests that it may not prevent the initial UV-induced damage, it is vital for inhibiting the subsequent inflammatory cascade and promoting skin healing.

1,25(OH)D suppresses production of interferon-gamma and interferon-alfa, which is crucial for patients with active disease because they frequently exhibit an overexpression of interferon-responsive genes. [20] Furthermore, vitamin D3 significantly induces apoptosis in activated B cells and inhibits class-switch recombination. [6] This process is essential for preventing the maturation of high-affinity self-reactive antibodies that contribute to tissue damage. It could potentially lower the titers of nuclear antibodies. [5] In addition, it has been suggested that vitamin D is able to increases cathelicidin expression. [21] CLE, where the skin barrier is chronically compromised due to inflammatory erosions and secondary atrophy, this dual capacity to suppress autoinflammation while enhancing innate immunity represents a fundamental therapeutic synergy. It creates a protective environment that lowers the risk of secondary infections while simultaneously silencing the inflammatory signals induced by UV exposure. [1] Vitamin D works as a sophisticated immunostatic agent deprived of broad-spectrum, non-specific immune suppression and leads to physiological homeostasis.

Despite the promising data, several limitations must be acknowledged. Most of the analyzed studies involve relatively small cohorts. There is a lack of large-scale randomized controlled trials (RCTs) investigating the long-term effects of high-dose vitamin D supplementation specifically in CLE. Furthermore, the role of Vitamin D Receptor (VDR) polymorphisms in determining individual response to supplementation remains an area that requires further exploration. The association between VDR and SLE is well-documented, still there is a profound lack of studies specifically correlating VDR polymorphism with the therapeutic outcome in CLE patients. Future research should focus on establishing standardized supplementation guidelines tailored to CLE patients, considering their unique photoprotective needs and pharmacological profiles.

A significant observation in this review is the chronological gap in the available clinical evidence. Most studies regarding vitamin D in CLE were conducted over a decade ago [4]; [5] ; [6]; [1]; [13]). Since then, our understanding of vitamin D metabolism and its role in autoimmune homeostasis has evolved. Current global guidelines for 2026 recognize daily doses of 1000–2000 IU as standard maintenance for the general population [15] , whereas historical CLE studies often utilized doses (e.g. 1400 IU) that were then considered high, but are now viewed as baseline. [1]

There is an urgent need for contemporary, large-scale randomized controlled trials (RCTs) that utilize these updated dosage standards. Such studies should evaluate whether higher, individualized doses (reaching up to the 2000-4000 IU range) could provide even more significant clinical benefits in terms of CLASI activity reduction and systemic immunomodulation, especially in patients with "biochemical resistance" induced by antimalarial therapy. [13]

4.1. CLINICAL MANAGEMENT AND SUPPLEMENTATION STRATEGIES

The established link between hypovitaminosis D and increased disease activity in CLE highlights the importance of developing tailored vitamin D dosing protocols. Current evidence suggests that standard, general-population supplementation guidelines are insufficient for this patient group due to physiological and pharmacological limitations.

It is paramount to monitor 25(OH)D concentrations, as CLE patients lack the endogenous seasonal compensation even during periods of high UV intensity. [5] It is recommended to assess that level at least twice a year. The primary goal of supplementation in CLE is to achieve serum 25(OH)D levels that allow for optimal immunomodulatory activity. Current global guidelines for 2026 suggest that standard maintenance dose for the general adult population range from 1000 IU to 2000 IU daily, depending on body mass index (BMI) and lifestyle. [15] However, for CLE patients, these standard doses may still be insufficient to reach disease stabilization. While historical studies such as [1] proposed 1400 IU daily (with significant success in reducing CLASI score), now it should be viewed as the minimum baseline for CLE patients. Due to strict photoprotection and antimalarias, many patients may require doses at the upper limit of the current standard (2000 IU) or higher individualized regimens to maintain serum levels above 30 ng/ml consistently throughout the year. [1]

A critical in determining the required dose is the use of hydroxychloroquine (HCQ). As patients on antimalarias have significantly higher risk of persistent deficiency [13], they may develop a form of "biochemical resistance" to standard supplementation.

Despite the shift toward higher daily intakes in 2026, the safety profile of vitamin D3 remains excellent, especially when using cholecalciferol. While historical studies in CLE patients, such as those by [1], focused on a daily dose of 1400 IU, they reported a complete absence of serious adverse events, such as hypercalcemia or hypercalciuria, over a one-year period. This provides a strong safety foundation for the CLE population.

5. CONCLUSIONS

The management of Cutaneous Lupus Erythematosus (CLE) remains a clinical challenge that requires a balance between photoprotection and adequate vitamin D status. This narrative review underscores that hypovitaminosis D is a widespread phenomenon among CLE patients across all geographical latitudes, including regions with high solar radiation. A significant barrier to optimal disease control is the stringent prevention of UV-induced exacerbations.

Based on the analyzed evidence, the following conclusions can be drawn:

Biannual monitoring of serum 25(OH)D concentrations should be mandatory due to CLE patients' lack of natural seasonal increase in vitamin D levels even in high-irradiance regions. Considering 2026 standards, where 1000–2000 IU is recognized as a maintenance dose for the general population, CLE patients require individualized regimens at the upper end of this range (or higher) to achieve therapeutic efficacy. This is particularly crucial for patients on antimalarial therapy, w

hich may impair vitamin D metabolism. Vitamin D should no longer be viewed only as a dietary supplement but as safe, complementary intervention. Correcting its deficiency leads to a measurable reduction in disease activity (CLASI scores) and shortens the duration of inflammatory skin lesions. There is an urgent need for new, large-scale randomized controlled trials (RCTs) as the majority of key literature in this field dates back more than ten years. Future research must evaluate the efficacy of current supplementation dose standards in the context of modern CLE pharmacotherapy.

DISCLOSURE

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While preparing this manuscript, the authors used the Gemini tool to enhance language quality and readability. After using this tool, the authors thoroughly reviewed and edited the text as necessary and accept full responsibility for the scientific content of the publication.

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