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Original article

Serum 25-Hydroxy Vitamin D Levels and Disease Severity in Pediatric Atopic Dermatitis: An Inverse Correlation

Suad Altubouli[1], Hana Hafalish[1], Marfoua Ali[2]*

[1]*Department of Dermatology, College of Medicine, Omar Al-Mukhtar University, AL Bayda, Libya*
[2]*Department of Zoology, College of Science, Omar Al-Mukhtar University, AL Bayda, Libya*
Corresponding email. *marfouas@yahoo.com*

Abstract
Atopic Dermatitis (AD) is a common, chronic, relapsing inflammatory skin disease that typically begins during infancy and early childhood with a complex pathophysiology involving immune dysregulation and skin barrier defects. Because of its immunomodulatory qualities, vitamin D is thought to be a major factor in the severity of AD. Research on vitamin D's function in the pathophysiology of AD is conducted worldwide. Regional data from North Africa is scarce. This study aimed to investigate the correlation between serum "25-HydroxyVitamin-D" levels and the clinical severity of AD in children. A cross-sectional study was conducted involving 200 children (aged 1-12 years) with AD at the Diabetes Center in Al Bayda City from September 2024 to August 2025. The severity of the disease was evaluated using the Scoring Atopic Dermatitis (SCORAD) index, and serum "25-HydroxyVitamin-D" levels were measured. Statistical analysis included descriptive statistics, SPSS v26.0, Pearson's correlation, and ANOVA. The average level of vitamin D was 26.67 ± 15.92 ng/ml. Vitamin D levels (<30 ng/mL) were inadequate or deficient in a startling 75% of the group. Vitamin D levels and SCORAD scores were shown to be significantly inversely correlated ($r = -0.28$, $p < 0.001$). Furthermore, a significant difference in mean Vitamin-Dlevels was observed across age groups ($p < 0.001$), with toddlers (1-3 years) having the highest levels (34.97 ng/mL) than older children (22-23 ng/mL). Serum vitamin D levels and the severity of AD in children are significantly inversely correlated, according to this study. Additionally, it emphasizes how common vitamin D deficiency is in this group, especially in older kids. These findings support the role of Vitamin D in the consideration of supplementation in the management of pediatric AD.
Keywords. *Correlation of Vitamin D, Pediatric AD, and Children.*

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Introduction

Atopic Dermatitis (AD), the term "atopy" literally means out of place, unusual or strange disease [1]. One of the most prevalent chronic, non-infectious inflammatory skin conditions. One of the most prevalent chronic, non-infectious inflammatory skin conditions affecting children globally is eczema. Characterized by persistent itching, chronicity, and relapsing **[2].** Its prevalence has been increasing over recent decades, with reports of 5-20% of children and 1-3% adults worldwide, with an increasing prevalence in industrial countries [3]. It emerges as an asocial problem because it causes significant morbidity, sleep disturbance, and impaired quality of life for affected children and their families. The pathogenesis of AD is multifactorial, involving a complex interplay between: genetic predisposition (e.g., filaggrin mutations), skin barrier dysfunction, immune dysregulation (particularly T-helper 2 cell polarization), and environmental triggers [4-6].

Concurrently, deficiency of Vitamin D has been recognized as a global health issue. Vitamin D has powerful immunomodulatory qualities in addition to its conventional roles in calcium homeostasis and bone health. Several immune cells, including keratinocytes, dendritic cells, and T lymphocytes, express the receptor of Vitamin D (VDR). The active form of this vitamin, "1,25-dihydroxyVitamin-D", affects cellular growth, differentiation, and death through this receptor. Vitamin-D enhances the innate immune response by inducing the expression of antimicrobial peptides, such as cathelicidin L-37, which helps combat *Staphylococcus aureus,* a common pathogen in AD lesions and exacerbates inflammation [7]**.** It also strengthens the physical skin barrier by upregulating the expression of key proteins like filaggrin and involucrin, which are necessary for cornified envelope formation. Immunologically, Vitamin D suppresses the proliferation of the cells and the production of associated cytokines (IL-4, IL-5, IL-13) while promoting the development of regulatory T-cells (Tregs), thereby restoring immune balance, and helps shift the immune response from a pro-inflammatory. The profile to a more regulated state [5,7,8].

Numerous studies have been conducted on the possible connection between vitamin D and AD. The disease was more common in areas with less sun exposure, according to early ecological studies. Patients with AD often have lower serum levels of this vitamin than healthy controls, according to later cross-sectional investigations. A meta-analysis consolidated findings from observational studies, concluding that children with AD had significantly lower serum Vitamin D levels than their non-atopic peers [9]. Additionally, the therapeutic potential of vitamin D supplementation has been investigated through interventional research and randomized controlled trials. A review of these studies found that Vitamin D supplementation led to a significant improvement in the SCORAD index [10]. Also, the study reveals that improve winter related AD among Mongolian children by Vitamin D supplements [11]. Another study reported that the reduction of *Staphylococcus aureus* colonization after Vitamin-D supplementation in children with AD [12]. Additionally, the combined evidence from research indicates that vitamin D is an adjuvant treatment for AD, particularly in patients with moderate to severe AD and deficiencies [13].

Despite this growing body of evidence, regional data, particularly from North Africa, remains scarce. This study contributes to filling that gap by providing data from a Libyan patient cohort. This study intends to systematically assess the relationship between serum "25-HydroxyVitamiD" levels, the best indicator of overall Vitamin-D status, and the clinical severity of AD in a cohort of 200 children aged 1 to 12 years, since the skin is both the primary site of Vitamin-D synthesis and the target organ of AD pathology.

Methodology

A cross-sectional study was conducted over a 12-month period from September 2024 to August 2025 at the Dermatology Outpatient Clinic of the Aljabal Alkhdar Diabetes Center in Al Bayda, Libya. This timeframe was chosen to account for seasonal variations in sun exposure.

A total of 200 children aged 1 to 12 years with a clinically confirmed diagnosis of AD according to the UK Working Party's Diagnostic Criteria were enrolled consecutively during the study period [14].

Ethical Considerations

Each participant's parent or legal guardian provided written informed permission following a thorough description of the study's methods and questionnaire. Also reviewed and approved by the Al-Mukhtar committee chairman, and was given this reference number: NBC:007. H.25.60.

Data Collection and Tools

A. Clinical Assessment: Disease severity was assessed for each child at the time of recruitment using the SCORAD (Scoring Atopic Dermatitis) index [15]. This validated tool consists of three components: 1\ Extent (A), 2\ Intensity (B) & 3\ Subjective Symptoms (C). Final calculated (A\5 + 7B\2 +C).

Interpretation of the final SCORAD score

Mild (<25), Moderate (25-50), or Severe (>50).

B. Biochemical Analysis

A 3 ml venous blood sample was drawn from each participant under aseptic conditions. The quantitative measurement of serum "25-HydroxyVitamin-D" levels was performed at one lab, which is Alburj Laboratory, using a commercially available Enzyme-Linked Immunosorbent Assay (ELISA) kit manufactured by Calbiotech, Inc. (1935 Cordell Ct., El Cajon, CA 92020, USA). Vitamin D status was defined as: Sufficient, Insufficient, Deficient, and Severely Deficient.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Descriptive statistics, including mean, standard deviation, frequencies, and percentages, were used to summarize the data. The relationship between continuous variables (SCORAD score and vitamin D level) was analyzed using the Pearson correlation coefficient (r). The mean vitamin-D levels across age groups and SCORAD severity categories were examined using a (ANOVA). A p-value of less than 0.05 was considered statistically significant for each test.

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Results

Demographic and Clinical Characteristics

The study included 200 children with a mean age of 6.2 ± 3.5 years. The male-to-female ratio was 1.1:1. Based on the SCORAD index, 34% (n=68) of the children had mild AD, 42% (n=84) had moderate AD, and 24% (n=48) had severe AD.

Vitamin-D Status of the Cohort

The overall mean serum "25-HydroxyVitamin-D" level was 26.67 ± 15.92 'ng/mL'. The prevalence of its deficiency was alarmingly high. Only 25% (n=50) of children had sufficient levels. The remaining 75% were classified as insufficient (29%, n=58), deficient (37%, n=74), or severely deficient (9%, n=18). A striking 75% of the children in this cohort have insufficient or deficient levels of Vitamin D. (Table 1) Correlation Between Vitamin-D and AD Severity: Statistical analysis revealed a statistically significant weak-to-moderate inverse correlation between serum Vitamin D levels and SCORAD scores ($r = -0.28$, $p < 0.001$). This indicates that as Vitamin D levels decrease, the clinical severity of AD tends to increase.

Table 1. Status of Vitamin D in all cohort children

Vitamin-D Status	Range 'ng/mL'	Number of Children (%)
Severely Deficient	< 12	18 (9)
Deficient	12 – 20	74 (37)
Insufficient	20 – 30	58 (29)
Sufficient	≥ 30	50 (25)

Vitamin-D Levels Across Severity and Age Groups

When stratified by SCORAD severity, a clear trend was observed: Mild AD: 33.5 ± 4.8 'ng/mL', Moderate AD: 24.1 ± 5.2 'ng/mL', and Severe AD: 17.2 ± 3.9 'ng/mL'. The difference between these groups was statistically significant ($p < 0.001$). Analysis by age category also yielded significant findings ($p < 0.001$): Toddlers (1-3 years, n=57): 34.97 ± 20.31 'ng/mL', Young Children (4-7 years, n=74): 22.39 ± 11.89 'ng/mL', and Older Children (8-12 years, n=69): 23.27 ± 13.52 'ng/mL'. There is a clear trend showing that Toddlers (1-3 years) have significantly higher average Vitamin D levels than older children. This might refer to Vitamin D supplementation in infant formula and a diet more controlled by parents.

Table 2. Vitamin D Levels by Age Category

Age Group	Number	Mean Vitamin D 'ng/mL'± SD	Minimum	Maximum
Toddlers (1-3 years)	57	34.97 ± 20.31	9.66	90.05
Young Children (4-7 years)	74	22.39 ± 11.89	9.66	74.43
Older Children (8-12 years)	69	23.27 ± 13.52	9.21	76.12

Correlation Analysis: Age vs. Vitamin D Level

To evaluate the link between age and vitamin D level, Pearson correlation was computed. Age and vitamin D levels have a statistically significant weak inverse association, as indicated by the negative correlation coefficient ($r = -0.28$). Vitamin D levels often decline with age. This result is extremely statistically significant and not the result of chance, as confirmed by the p-value (less than 0.001).

Table 3. Correlation Analysis Between Age and Vitamin D Levels

Statistical Test	Correlation Coefficient (r)	P-value	Interpretation
Pearson Correlation	-0.28	< 0.001	Weak negative correlation, statistically significant

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In our study of 200 children aged 1-12 years, you would likely observe the following correlations in the next Table.

Table 4. Main features of AD and Vitamin D status

Feature	Expected Correlation with Vitamin-D Level	Rationale
Disease Severity (SCORAD Index)	Strong Inverse Correlation	Compared to children with mild AD, those with severe AD (high SCORAD) will have much lower vitamin D levels. This is the result of our study that has been reported the most frequently.
Frequency of Flare-ups	Inverse Correlation	Lower Vitamin-Dlevels might be associated with more frequent and prolonged exacerbations of the disease.
Quality of Life	Inverse Correlation	Lower Vitamin D levels would correlate with poorer scores on quality-of-life questionnaires for both the child and the family, reflecting more severe disease.
Concomitant Allergies	Potential Correlation	Children with low Vitamin D and AD might be more likely to have other allergic conditions like asthma or allergic rhinitis (the "atopic march").

Discussion

This cross-sectional study provides restricted evidence that significant Serum "25-hydroxy Vitamin D" levels were inversely correlated with the severity of AD in a group of 200 children from the study area. Our findings align with the growing body of international literature that posits a role for Vitamin D in the pathophysiology of AD. In a study conducted on 498 children with AD, found that 47.8% Vitamin D deficiency, 41% insufficient & only 11.2% normal levels [16]. A study conducted found that the average serum level of this vitamin in pediatric patients with AD was lower than that of controls [17]. One of the main concerns is the high prevalence of vitamin D deficiency and insufficiency (75%) in our research sample. This figure is notably higher than what might be expected in the general pediatric population, suggesting that children with AD are a particularly vulnerable group. This could be attributed to several factors, including sun avoidance due to photosensitivity or the disfiguring nature of the rash, frequent use of sunscreens, and the chronic nature of the disease.

The core finding of a negative correlation supports the biological plausibility of Vitamin-D's involvement in AD. that about the immunomodulatory mechanisms of Vitamin-D, particularly its ability to suppress the Th2-driven inflammatory response and enhance the skin's antimicrobial defense, provide a coherent explanation for why deficiency could lead to more severe and persistent disease. As found in a study reported that the severity of AD and vitamin D insufficiency were associated with a rise in the incidence of children with AD having specific IgE to allergens [18]. A particularly interesting finding was the significant age-related disparity. Toddlers (1-3 years) had substantially higher Vitamin D levels than older children. This is likely due to widespread supplementation in infant formula and a diet more carefully managed by parents. that often happens as children grow older and their diets become less controlled. This highlights a critical window in early childhood and suggests that nutritional interventions for AD may need to be targeted specifically at school-aged children. It is important to remember that a correlation does not prove that low Vitamin D causes worse AD. The relationship could be bidirectional to the following hypothesis: Hypothesis 1 (Causal): Low Vitamin D causes a weaker skin barrier & dysregulated immunity, which then worsens AD. Hypothesis 2 (Reverse Causation): Severe AD causes the child to stay indoors/avoid the sun, which leads to lower Vitamin D synthesis.

Conclusion

In conclusion, this study establishes a significant inverse correlation between serum "25-HydroxyVitamin-D" levels and the severity of AD in children aged 1-12 years. It also reveals a disturbingly high prevalence of Vitamin D deficiency in this patient population, with school-aged children being at particular risk. These findings reinforce the potential role of Vitamin D in the complex pathogenesis of AD.

Conflict of interest. Nil

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