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Original Research Article

Impact of Vitamin D Deficiency on Clinical Severity and Recurrence Rate in Patients with Chronic Urticaria

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Abstract:

Background: Chronic urticaria (CU) is a debilitating dermatological condition characterized by recurrent wheals persisting for more than six weeks. Emerging evidence suggests that vitamin D, through its immunomodulatory properties, may influence the pathogenesis, clinical severity, and recurrence patterns of chronic urticaria. However, the precise relationship between vitamin D status and disease outcomes in chronic urticaria patients remains insufficiently characterized.

Methods: A prospective observational cohort study was conducted on 240 patients with chronic urticaria and 120 age- and sex-matched healthy controls. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured in all participants. Disease severity was assessed using the Urticaria Activity Score over 7 days (UAS7). Patients were categorized into vitamin D deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), and sufficient (≥30 ng/mL) groups. Recurrence was monitored over a 12-month follow-up period.

Results: The mean serum 25(OH)D level was significantly lower in CU patients compared to controls (16.4 ± 7.8 vs. 28.3 ± 9.2 ng/mL, $p < 0.001$). Among CU patients, 58.3% were vitamin D deficient, 26.7% were insufficient, and 15.0% were sufficient. Vitamin D deficient patients exhibited significantly higher UAS7 scores (32.6 ± 7.4) compared to insufficient (24.1 ± 6.8) and sufficient (17.3 ± 5.9) groups ($p < 0.001$). The 12-month recurrence rate was significantly elevated in the deficient group (71.4%) compared to the insufficient (46.9%) and sufficient (25.0%) groups ($p < 0.001$). Serum 25(OH)D levels demonstrated a significant negative correlation with UAS7 scores ($r = −0.614$, $p < 0.001$).

Conclusion: Vitamin D deficiency is highly prevalent among chronic urticaria patients and is significantly associated with greater disease severity and higher recurrence rates. Assessment and correction of vitamin D status may represent a valuable adjunctive strategy in the comprehensive management of chronic urticaria.

Keywords: Chronic Urticaria; Vitamin D Deficiency; 25-Hydroxyvitamin D; Urticaria Activity Score; Disease Severity; Recurrence; Immunomodulation.

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Introduction

Chronic urticaria is a prevalent and clinically significant dermatological disorder defined by the spontaneous or inducible occurrence of wheals, angioedema, or both for a duration exceeding six weeks [1]. The condition affects approximately 0.5–5% of the general population worldwide and imposes a substantial burden on patients' quality of life, psychological well-being, and healthcare resource utilization [2]. Despite advances in understanding its immunopathogenesis, chronic urticaria remains a therapeutic challenge, with a significant proportion of patients demonstrating inadequate response to conventional antihistamine therapy [3].

The pathophysiology of chronic urticaria involves complex interactions among mast cells, basophils, autoantibodies, complement activation, and various immunological mediators [4]. Mast cell degranulation and the subsequent release of histamine, leukotrienes, and pro-inflammatory cytokines constitute the central effector mechanisms driving wheal formation and associated symptomatology [5]. Within this immunological framework, numerous modulatory factors have been

investigated for their potential role in disease modification, among which vitamin D has garnered considerable scientific attention in recent years [6].

Vitamin D is a secosteroid hormone that exerts pleiotropic effects extending far beyond its classical role in calcium and phosphorus homeostasis. The discovery of vitamin D receptors (VDR) on virtually all immune cells, including T lymphocytes, B lymphocytes, macrophages, dendritic cells, and mast cells, has established vitamin D as a critical regulator of both innate and adaptive immunity [7]. Specifically, vitamin D has been shown to modulate mast cell activation and degranulation, suppress the synthesis of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), promote regulatory T cell differentiation, and enhance the production of antimicrobial peptides including cathelicidin [8].

Several cross-sectional studies have reported significantly lower serum 25-hydroxyvitamin D [25(OH)D] levels in patients with chronic urticaria compared to healthy individuals [9]. Furthermore, preliminary interventional studies have suggested that vitamin D supplementation may reduce disease severity and improve treatment response in selected CU patient populations [10]. A systematic review by Tuchinda and colleagues highlighted the potential therapeutic benefit of vitamin D supplementation as an adjunct to standard antihistamine therapy [11]. However, clinical data regarding the long-term impact of vitamin D status on recurrence patterns remain limited.

Despite the accumulating evidence, significant research gaps persist. The relationship between categorical vitamin D status (deficient, insufficient, sufficient) and graded clinical severity using validated scoring instruments has not been adequately characterized in prospective designs [12]. Moreover, longitudinal studies examining the predictive value of baseline vitamin D levels for recurrence risk are sparse. The heterogeneity in study populations, methodological approaches, and outcome assessment tools across existing investigations further underscores the need for well-designed prospective studies [13].

The aim of this study was to comprehensively evaluate the association between serum vitamin D levels and clinical severity, treatment response, and 12-month recurrence rate in patients with chronic urticaria, thereby elucidating the potential clinical significance of vitamin D assessment in the management of this condition.

Materials and Methods

Study Design and Setting: This prospective observational cohort study was conducted at the Department of Dermatology, Venereology, and Leprology of a tertiary care teaching hospital.

Study Population: A total of 240 consecutive patients diagnosed with chronic urticaria according to the international EAACI/GA²LEN/EDF/WAO guidelines were enrolled in the study. An additional 120 age- and sex-matched healthy volunteers without any history of urticaria, atopic conditions, or autoimmune diseases were recruited as a control group from the hospital's preventive health screening clinic.

Inclusion and Exclusion Criteria: Inclusion criteria encompassed adults aged 18–65 years with a confirmed diagnosis of chronic spontaneous urticaria (CSU) persisting for ≥6 weeks, willingness to participate in follow-up visits, and ability to provide informed consent. Exclusion criteria included inducible urticaria (dermographism, cold urticaria, solar urticaria), urticarial vasculitis confirmed by biopsy, concurrent use of vitamin D supplements or systemic corticosteroids within the preceding three months, pregnancy or lactation, chronic kidney disease, liver disease, malabsorption syndromes, hyperparathyroidism, known malignancy, and use of immunosuppressive agents.

Clinical Assessment: At baseline, a detailed clinical history was obtained including disease duration, frequency of episodes, and previous treatments. Disease severity was assessed using the Urticaria Activity Score over 7 days (UAS7), a validated composite score evaluating the number and intensity of wheals and pruritus severity on a daily basis, yielding a total score ranging from 0 to 42. UAS7 scores were categorized as well-controlled (0–6), mild (7–15), moderate (16–27), and severe (28–42). Quality of life was assessed using the Chronic Urticaria Quality of Life Questionnaire (CU-Q2oL). The Urticaria Control Test (UCT) was administered to evaluate disease control status.

Laboratory Investigations: Fasting venous blood samples were collected from all participants at enrollment. Serum 25(OH)D levels were measured using electrochemiluminescence immunoassay (ECLIA) on the Roche Cobas e601 platform. Vitamin D status was classified as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), or sufficient (≥30 ng/mL). Additional laboratory parameters included complete blood count with differential, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), total serum IgE, thyroid function tests, antinuclear antibody (ANA), and autologous serum skin test (ASST).

Treatment and Follow-up Protocol: All patients received standardized treatment according to current guidelines, consisting of second-generation H1-antihistamines at standard doses, with up-dosing (up to fourfold) for refractory cases. Treatment response was assessed at 4-week intervals. Patients were followed prospectively for 12 months with clinical assessments at months 1, 3, 6, 9, and 12. Recurrence was defined as the reappearance of urticarial

symptoms (UAS7 ≥ 7) after achieving remission (UAS7 ≤ 6) for at least four consecutive weeks. No vitamin D supplementation was administered during the study period to avoid confounding.

Statistical Analysis: Continuous variables were expressed as mean ± standard deviation and compared using independent samples t-test or one-way analysis of variance (ANOVA) with post-hoc Tukey's test. Categorical variables were presented as frequencies and percentages and compared using the chi-square test. Pearson's correlation coefficient was used to assess the relationship between serum vitamin D levels and UAS7 scores. Kaplan-Meier survival analysis was employed to estimate recurrence-free survival, with log-rank test for group comparisons. Multivariate logistic regression was performed to identify independent predictors of recurrence. A p-value < 0.05 was considered statistically significant. All analyses were conducted using SPSS version 27.0 and GraphPad Prism version 9.5.

Results

Baseline Characteristics and Vitamin D Status: The demographic and baseline clinical characteristics of the study participants are presented in Table 1. The mean age of CU patients was 36.2 ± 10.4 years, with a female predominance (62.5%). The mean disease duration was 14.8 ± 9.6 months. Serum 25(OH)D levels were significantly lower in CU patients compared to healthy controls (16.4 ± 7.8 vs. 28.3 ± 9.2 ng/mL, p < 0.001). Among CU patients, 140 (58.3%) were vitamin D deficient, 64 (26.7%) were insufficient, and 36 (15.0%) were sufficient. In contrast, among controls, 18 (15.0%) were deficient, 34 (28.3%) were insufficient, and 68 (56.7%) were sufficient (p < 0.001).

Table 1: Baseline Demographic and Clinical Characteristics

Variable	CU Patients (n = 240)	Healthy Controls (n = 120)	p-value
Age (years), mean ± SD	36.2 ± 10.4	35.8 ± 9.7	0.726
Female sex, n (%)	150 (62.5%)	74 (61.7%)	0.872
BMI (kg/m²), mean ± SD	25.9 ± 4.3	25.4 ± 3.8	0.287
Disease duration (months), mean ± SD	14.8 ± 9.6	—	—
Serum 25(OH)D (ng/mL), mean ± SD	16.4 ± 7.8	28.3 ± 9.2	<0.001
Vitamin D deficient (<20 ng/mL), n (%)	140 (58.3%)	18 (15.0%)	<0.001
Vitamin D insufficient (20–29.9 ng/mL), n (%)	64 (26.7%)	34 (28.3%)	0.738
Vitamin D sufficient (≥30 ng/mL), n (%)	36 (15.0%)	68 (56.7%)	<0.001
Total IgE (IU/mL), mean ± SD	312.5 ± 186.4	87.6 ± 42.3	<0.001
CRP (mg/L), mean ± SD	6.8 ± 4.2	2.1 ± 1.4	<0.001
ESR (mm/hr), mean ± SD	22.4 ± 12.6	10.2 ± 5.8	<0.001
Positive ASST, n (%)	86 (35.8%)	—	—

Association Between Vitamin D Status and Disease Severity: Significant differences in disease severity parameters were observed across the three vitamin D status groups among CU patients. Vitamin D deficient patients exhibited markedly higher mean UAS7 scores (32.6 ± 7.4) compared to insufficient (24.1 ± 6.8) and sufficient (17.3 ± 5.9) groups (p < 0.001). Similarly, CU-Q2oL scores were significantly worse and UCT scores significantly lower in the deficient group. A significant negative correlation was observed between serum 25(OH)D levels and UAS7 scores (r = −0.614, p < 0.001). Inflammatory markers including CRP and total IgE levels were also significantly elevated in the vitamin D deficient group (Table 2).

Table 2: Clinical Severity and Laboratory Parameters by Vitamin D Status Among CU Patients

Parameter	Deficient (n = 140)	Insufficient (n = 64)	Sufficient (n = 36)	p-value
UAS7 score, mean ± SD	32.6 ± 7.4	24.1 ± 6.8	17.3 ± 5.9	<0.001
Severe CU (UAS7 28–42), n (%)	98 (70.0%)	18 (28.1%)	4 (11.1%)	<0.001
Moderate CU (UAS7 16–27), n (%)	34 (24.3%)	32 (50.0%)	14 (38.9%)	0.001
Mild CU (UAS7 7–15), n (%)	8 (5.7%)	14 (21.9%)	18 (50.0%)	<0.001
CU-Q2oL score, mean ± SD	62.8 ± 14.6	44.3 ± 12.8	30.5 ± 10.2	<0.001
UCT score, mean ± SD	5.2 ± 2.8	8.6 ± 3.1	12.4 ± 2.6	<0.001
Total IgE (IU/mL), mean ± SD	386.2 ± 198.7	248.6 ± 142.3	178.4 ± 96.8	<0.001
CRP (mg/L), mean ± SD	8.6 ± 4.8	5.2 ± 3.1	3.4 ± 2.0	<0.001
Positive ASST, n (%)	62 (44.3%)	16 (25.0%)	8 (22.2%)	0.006
Antihistamine up-dosing required, n (%)	108 (77.1%)	30 (46.9%)	10 (27.8%)	<0.001

Recurrence Analysis: Over the 12-month follow-up period, the overall recurrence rate among CU patients was 57.5% (138/240). Recurrence was significantly higher in the vitamin D deficient group (71.4%) compared to the insufficient (46.9%) and sufficient (25.0%) groups ($p < 0.001$). Kaplan-Meier analysis demonstrated significantly shorter recurrence-free survival in vitamin D deficient patients (median 5.2 months) compared to insufficient (median 8.4 months) and sufficient (median not reached) groups (log-rank $p < 0.001$). Multivariate logistic regression analysis identified vitamin D deficiency (OR 4.82, 95% CI: 2.14–10.86, $p < 0.001$), positive ASST (OR 2.36, 95% CI: 1.38–4.04, $p = 0.002$), baseline UAS7 ≥ 28 (OR 3.18, 95% CI: 1.76–5.74, $p < 0.001$), and elevated CRP (OR 1.94, 95% CI: 1.12–3.36, $p = 0.018$) as independent predictors of recurrence (Table 3).

Table 3: Multivariate Logistic Regression Analysis of Independent Predictors of 12-Month Recurrence

Predictor Variable	Odds Ratio	95% CI	p-value
Vitamin D deficiency (<20 ng/mL)	4.82	2.14–10.86	<0.001
Vitamin D insufficiency (20–29.9 ng/mL)	2.16	0.88–5.30	0.092
Positive ASST	2.36	1.38–4.04	0.002
Baseline UAS7 ≥ 28	3.18	1.76–5.74	<0.001
Elevated CRP (>5 mg/L)	1.94	1.12–3.36	0.018
Total IgE > 300 IU/mL	1.62	0.94–2.79	0.081
Disease duration > 12 months	1.78	1.06–2.99	0.029
Female sex	1.24	0.74–2.08	0.412
Age > 40 years	0.92	0.54–1.57	0.764
BMI > 30 kg/m²	1.38	0.76–2.51	0.294

Discussion

The findings of this prospective cohort study provide robust evidence that vitamin D deficiency is significantly more prevalent among patients with chronic urticaria compared to healthy controls and is independently associated with greater disease severity and substantially higher recurrence rates over a 12-month follow-up period. These observations carry important implications for the clinical management of chronic urticaria and support the growing body of evidence implicating vitamin D in the immunopathogenesis of allergic and inflammatory dermatological conditions.

The markedly lower mean serum 25(OH)D levels observed in our CU cohort (16.4 ± 7.8 ng/mL) compared to healthy controls (28.3 ± 9.2 ng/mL) are consistent with findings from several previous investigations. Thorp and colleagues were among the first to demonstrate significantly reduced vitamin D levels in chronic urticaria patients [14]. Subsequently, Grzanka and coworkers reported that vitamin D deficiency was present in approximately 50–70% of CU patients, corroborating our finding of 58.3% prevalence [15]. A meta-analysis by Tsai and Lin further consolidated these observations, confirming a statistically significant difference in serum 25(OH)D levels between CU patients and healthy individuals [16].

The strong negative correlation between serum 25(OH)D levels and UAS7 scores ($r = −0.614$, $p < 0.001$) observed in our study reinforces the hypothesis that vitamin D status directly influences disease activity. This finding aligns with the work of Boonpiyathad and colleagues, who reported a similar inverse correlation between vitamin D levels and urticaria severity indices [17]. The mechanistic basis for this relationship likely involves the immunomodulatory actions of vitamin D on mast cell biology. In vitro studies have demonstrated that 1,25-dihydroxyvitamin D3, the biologically active metabolite, inhibits IgE-mediated mast cell activation and suppresses the release of histamine, prostaglandin D2, and pro-inflammatory cytokines [18].

Our observation that vitamin D deficient patients required antihistamine up-dosing significantly more frequently (77.1%) than sufficient patients (27.8%) is clinically relevant and consistent with the findings of Rorie and colleagues, who demonstrated improved urticaria control when vitamin D supplementation was added to antihistamine therapy in deficient patients [19]. The relative treatment resistance observed in vitamin D deficient patients may relate to the enhanced mast cell reactivity and heightened inflammatory milieu associated with inadequate vitamin D signaling [20].

A particularly novel contribution of this study is the prospective evaluation of recurrence risk stratified by vitamin D status. The substantially higher 12-month recurrence rate in vitamin D deficient patients (71.4%) compared to sufficient patients (25.0%) and the identification of vitamin D deficiency as the strongest independent predictor of recurrence (OR 4.82) underscore the potential prognostic significance of vitamin D assessment. These longitudinal findings extend the cross-sectional observations reported by Chandrashekar and colleagues and provide a temporal dimension to the vitamin D–urticaria relationship [21].

The elevated total IgE levels and positive ASST prevalence observed in the vitamin D deficient group suggest a potential interaction between vitamin D status and autoimmune mechanisms in chronic urticaria. Vitamin D has been shown to promote immune tolerance through enhancement of regulatory T cell function and suppression of Th1 and Th17 pathways [22]. Deficiency may therefore facilitate the breakdown of immune tolerance and promote the generation of functional autoantibodies against IgE or the high-affinity IgE receptor (FcεRIα), which are implicated in the pathogenesis of autoimmune chronic urticaria [23].

The significant association between elevated CRP levels and vitamin D deficiency in our CU cohort further supports the concept that low vitamin D status contributes to a systemic pro-inflammatory state. This observation is consistent with epidemiological data from large population-based studies demonstrating an inverse relationship between vitamin D levels and inflammatory biomarkers [24].

Several limitations of this study warrant consideration. The observational design precludes the establishment of causality between vitamin D deficiency and disease severity or recurrence. The single-center setting may limit the generalizability of findings across diverse geographical and ethnic populations with varying baseline vitamin D levels. Additionally, the absence of a vitamin D supplementation intervention arm prevents us from determining whether correction of deficiency would directly improve clinical outcomes. Seasonal variation in vitamin D levels was not specifically controlled for, although enrollment occurred throughout the study period. Future randomized controlled trials investigating the therapeutic efficacy of vitamin D supplementation in vitamin D deficient CU patients are warranted to establish a definitive causal relationship and inform clinical guidelines [25].

Conclusion

This prospective cohort study demonstrates that vitamin D deficiency is highly prevalent among patients with chronic urticaria and is significantly and independently associated with increased disease severity, poorer treatment response, diminished quality of life, and substantially higher recurrence rates over a 12-month follow-up period. Serum 25-hydroxyvitamin D levels exhibited a strong inverse correlation with urticaria activity scores, and vitamin D deficiency emerged as the most powerful independent predictor of disease recurrence. These findings underscore the clinical importance of routine vitamin D assessment in patients with chronic urticaria and support the rationale for investigating vitamin D supplementation as an adjunctive therapeutic strategy. Integration of vitamin D status evaluation into the standard diagnostic workup for chronic urticaria may contribute to improved disease prognostication, individualized treatment planning, and enhanced long-term clinical outcomes.

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