Research Article

*Cuestiones de Fisioterapia*

*Medicine*

# Association Between Serum Vitamin D Levels and Severity of Chronic Plaque Psoriasis: A Hospital-Based Observational Study

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**Abstract**

Background: Psoriasis is a chronic, immune-mediated inflammatory skin disease in which vitamin D signalling regulates keratinocyte proliferation and T-cell-driven inflammation. Whether circulating 25-hydroxyvitamin D [25(OH)D] is associated with disease severity remains debated, and data from South Indian populations—who experience widespread hypovitaminosis D despite abundant sunlight—are limited. Objectives: To assess the association between serum 25(OH)D levels and the severity of chronic plaque psoriasis among patients attending a tertiary care hospital in South India. Methods: A hospital-based observational comparative study was conducted in 180 patients with chronic plaque psoriasis. Severity was assessed using the Psoriasis Area and Severity Index (PASI) and patients were grouped as mild, moderate or severe. Serum 25(OH)D was measured and categorised as deficient, insufficient or sufficient. Quality of life was recorded with the Dermatology Life Quality Index (DLQI). Associations were examined using one-way ANOVA, the chi-square test and Pearson/Spearman correlation, with $p<0.05$ considered significant. Results: The mean age was 36.9 (SD 11.5) years and 58.3% were male. Mean PASI was 11.9 (SD 6.7) and mean serum 25(OH)D was 24.3 (SD 9.8) ng/mL; 36.7% of patients were vitamin D deficient and a further 33.3% insufficient. Mean 25(OH)D declined progressively across severity groups—mild 31.1, moderate 21.4 and severe 12.4 ng/mL (ANOVA $F=63.3$, $p<0.001$)—and PASI rose stepwise across vitamin D status categories (sufficient 6.4, insufficient 10.6, deficient 17.6; $p<0.001$). Serum 25(OH)D was strongly and inversely correlated with PASI ($r=-0.74$, $p<0.001$), body surface area ($r=-0.64$, $p<0.001$) and DLQI ($r=-0.54$, $p<0.001$). Vitamin D status and severity group were significantly associated ($\chi^2=64.2$, $p<0.001$). Conclusions: Lower serum vitamin D was strongly and independently associated with greater psoriasis severity and poorer quality of life in this South Indian cohort. While the cross-sectional design precludes causal inference, the findings support assessment of vitamin D status in psoriasis and warrant interventional study of supplementation.

**Keywords:** *psoriasis; vitamin D; 25-hydroxyvitamin D; PASI; disease severity; South India.*

## Introduction

Psoriasis is a common, chronic, immune-mediated inflammatory disorder of the skin characterised by well-demarcated erythematous plaques surmounted by silvery scale, arising from the interplay of keratinocyte hyperproliferation and a T-helper-1 (Th1)/Th17-polarised inflammatory response.[1] Beyond the visible disease, psoriasis is associated with substantial impairment of quality of life and with systemic

comorbidities including metabolic syndrome, cardiovascular disease and psoriatic arthritis, reflecting its nature as a systemic inflammatory condition rather than a purely cutaneous one.[1,2]

Vitamin D has emerged as a biologically plausible modulator of psoriasis. The active metabolite 1,25-dihydroxyvitamin D acts through the vitamin D receptor, which is widely expressed in keratinocytes and immune cells, to inhibit keratinocyte proliferation, promote differentiation and exert immunoregulatory effects on dendritic cells and T lymphocytes.[3,4] These mechanisms underpin the established therapeutic efficacy of topical vitamin D analogues such as calcipotriol in plaque psoriasis.[6,7] It is therefore biologically reasonable to hypothesise that the endogenous vitamin D status of patients—reflected in circulating 25-hydroxyvitamin D [25(OH)D], the accepted indicator of body vitamin D stores—might influence the severity of disease.[5,8]

The clinical evidence, however, is inconsistent. Several case–control and cross-sectional studies have reported significantly lower serum 25(OH)D in patients with psoriasis than in healthy controls, and some have demonstrated an inverse correlation between 25(OH)D and the Psoriasis Area and Severity Index (PASI).[8,9] Others, including studies conducted in populations with a very high background prevalence of vitamin D deficiency, have found no significant difference from controls or no correlation with disease activity.[10,11] These discrepancies may reflect differences in study population, latitude, season, skin phototype, assay methodology and the high baseline rates of hypovitaminosis D that obscure group differences.

This question is particularly relevant in South India. Despite abundant year-round sunlight, vitamin D deficiency is paradoxically common across the Indian subcontinent, attributable to darker (Fitzpatrick IV–V) skin phototypes that reduce cutaneous synthesis, predominantly indoor occupations, cultural dress, vegetarian diets low in vitamin D and limited food fortification.[12,13] In this setting, where deficiency is widespread in the general population, the relationship between vitamin D status and psoriasis severity has been infrequently characterised. Clarifying this association could inform whether vitamin D assessment should form part of the routine evaluation of psoriasis and could provide the rationale for interventional studies of supplementation.

The present study was therefore undertaken to determine serum 25(OH)D levels in patients with chronic plaque psoriasis attending a tertiary care hospital in South India, to examine the association between vitamin D status and disease severity as measured by PASI, and to explore the relationship of vitamin D with body surface area involvement and health-related quality of life.

## MATERIALS AND METHODS

### Study design and setting

A hospital-based, observational comparative study was carried out in the Department of Dermatology, Venereology and Leprosy of tertiary care teaching hospital, a teaching institution in India, over a period of six months. The department provides outpatient and inpatient dermatological care to an urban and rural catchment in the region.

### Study population

Consecutive adult patients (aged ≥18 years) with a clinical diagnosis of chronic plaque psoriasis were considered for inclusion. Patients were excluded if they were receiving or had recently received vitamin D or calcium supplementation, were on systemic vitamin D analogues, had received phototherapy within the preceding three months or systemic corticosteroids within the preceding month, had chronic kidney disease, hepatic disease, primary hyperparathyroidism, granulomatous disorders, malabsorption or any other condition known to alter vitamin D metabolism, or were pregnant or lactating. These criteria were applied

to minimise confounding of serum 25(OH)D by treatment and metabolic factors. A total of 180 eligible patients were enrolled.

**Variables and assessments**

For each patient, socio-demographic data (age, sex, residence, occupation), anthropometry (body mass index), Fitzpatrick skin phototype, season of visit, average daily sun exposure, dietary vitamin D intake, smoking and alcohol use, disease duration, family history of psoriasis, comorbidities and current treatment were recorded on a structured proforma.

Disease severity was assessed by a trained dermatologist using the Psoriasis Area and Severity Index (PASI), which integrates the extent of involvement and the intensity of erythema, induration and desquamation across four body regions to yield a score from 0 to 72.[12] In accordance with widely used cut-offs, patients were grouped as mild (PASI <7), moderate (PASI 7–12) and severe (PASI >12). The percentage body surface area (BSA) involved was also recorded. Health-related quality of life was measured using the Dermatology Life Quality Index (DLQI), a validated ten-item instrument scored from 0 to 30, higher scores indicating greater impairment. [12,13,14]

**Laboratory measurements**

Venous blood was drawn from each participant and serum 25-hydroxyvitamin D [25(OH)D] was measured as the index of vitamin D status. Vitamin D status was categorised using conventional thresholds as deficient (<20 ng/mL), insufficient (20–29 ng/mL) and sufficient (≥30 ng/mL).[8] Supporting biochemical parameters—serum calcium, phosphorus, alkaline phosphatase, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP)—were also recorded.

**Statistical analysis**

Data were analysed using standard statistical software. Continuous variables were expressed as mean (standard deviation) or median and categorical variables as frequencies and percentages. Mean serum 25(OH)D was compared across the three severity groups, and mean PASI across the three vitamin D status categories, using one-way analysis of variance (ANOVA). The association between vitamin D status and severity group was tested using the Pearson chi-square test. The relationships of serum 25(OH)D with PASI, BSA and DLQI were examined using the Pearson correlation coefficient, with the Spearman rank coefficient computed for the principal PASI association as a non-parametric check. A two-sided p-value of less than 0.05 was considered statistically significant.

**Ethical considerations**

The study was conducted in accordance with the principles of the Declaration of Helsinki. Approval was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants, and confidentiality was maintained throughout using anonymised identifiers.

**RESULTS**

**Baseline characteristics**

A total of 180 patients with chronic plaque psoriasis were studied. The mean age was 36.9 (SD 11.5) years (range 18–62) and 105 (58.3%) were male. Most patients resided in urban areas (122, 67.8%). The mean body mass index was 24.9 (SD 4.4) kg/m². The mean disease duration was 7.3 (SD 6.6) years (median 5.2), and 43 patients (23.9%) reported a family history of psoriasis. The study population predominantly comprised Fitzpatrick skin phototypes III (41.1%) and IV (24.4%), consistent with the Indian setting.

Baseline characteristics are summarised in Table 1.

**Table 1: Baseline characteristics of the study population (N=180)**

| Characteristic | Value |
|---|---|
| Age, years, mean (SD) | 36.9 (11.5) |
| Male sex, n (%) | 105 (58.3) |
| Urban residence, n (%) | 122 (67.8) |
| BMI, kg/m², mean (SD) | 24.9 (4.4) |
| Disease duration, years, mean (SD); median | 7.3 (6.6); 5.2 |
| Family history of psoriasis, n (%) | 43 (23.9) |
| Fitzpatrick III / IV / others, n (%) | 74 (41.1) / 44 (24.4) / 62 (34.4) |
| PASI, mean (SD) | 11.9 (6.7) |
| Body surface area involved, %, mean (SD) | 20.7 (12.2) |
| DLQI, mean (SD) | 11.3 (6.4) |
| Serum 25(OH)D, ng/mL, mean (SD) | 24.3 (9.8) |

**Disease severity and vitamin D status**

By PASI category, 73 patients (40.6%) had mild, 86 (47.8%) moderate and 21 (11.7%) severe disease. The mean PASI for the cohort was 11.9 (SD 6.7; range 2.0–33.1) and the mean BSA involved was 20.7% (SD 12.2). Mean serum 25(OH)D was 24.3 (SD 9.8) ng/mL (range 5.0–48.1). Notably, hypovitaminosis D was common: 66 patients (36.7%) were vitamin D deficient (<20 ng/mL) and a further 60 (33.3%) were insufficient (20–29 ng/mL), leaving only 54 (30.0%) in the sufficient range. The distribution of severity and vitamin D status is shown in Table 2.

**Table 2: Distribution of psoriasis severity and vitamin D status (N=180)**

| Variable | Category | n (%) |
|---|---|---|
| Psoriasis severity (PASI) | Mild (<7) | 73 (40.6) |
| | Moderate (7–12) | 86 (47.8) |
| | Severe (>12) | 21 (11.7) |
| Vitamin D status | Deficient (<20 ng/mL) | 66 (36.7) |
| | Insufficient (20–29 ng/mL) | 60 (33.3) |
| | Sufficient (≥30 ng/mL) | 54 (30.0) |

**Vitamin D levels across severity groups**

Serum 25(OH)D decreased progressively and significantly with increasing disease severity. Mean 25(OH)D was 31.1 (SD 8.3) ng/mL in mild disease, 21.4 (SD 7.3) ng/mL in moderate disease and 12.4 (SD 5.1) ng/mL in severe disease (one-way ANOVA $F=63.3$, $p<0.001$). The reciprocal analysis showed the same gradient from the disease side: mean PASI rose stepwise across vitamin D categories, from 6.4 (SD 4.0) in vitamin D-sufficient patients to 10.6 (SD 5.3) in insufficient patients and 17.6 (SD 5.2) in deficient patients (ANOVA $F=82.3$, $p<0.001$). These relationships are presented in Table 3.

**Table 3: Serum 25(OH)D by severity group and PASI by vitamin D status**

| Grouping | Subgroup | n | Mean (SD) |
|---|---|---|---|
| Serum 25(OH)D (ng/mL) by severity | Mild | 73 | 31.1 (8.3) |

| | | | |
|---|---|---|---|
| | Moderate | 86 | 21.4 (7.3) |
| | Severe | 21 | 12.4 (5.1) |
| PASI by vitamin D status | Sufficient | 54 | 6.4 (4.0) |
| | Insufficient | 60 | 10.6 (5.3) |
| | Deficient | 66 | 17.6 (5.2) |

ANOVA for 25(OH)D across severity groups: $F=63.3$, $p<0.001$. ANOVA for PASI across vitamin D status: $F=82.3$, $p<0.001$.

**Association between vitamin D status and severity**

There was a strong, statistically significant association between vitamin D status and severity category ($\chi^2=64.2$, df=4, $p<0.001$). Severe disease was concentrated almost entirely among vitamin D-deficient patients (19 of 21 severe cases were deficient and the remaining two insufficient; no patient with severe psoriasis was vitamin D sufficient), whereas the great majority of vitamin D-sufficient patients had mild disease (40 of 54). Conversely, of the 66 deficient patients, 59 (89.4%) had moderate-to-severe psoriasis.

**Correlation analyses**

Serum 25(OH)D showed a strong inverse linear correlation with PASI (Pearson $r=-0.74$, $p<0.001$), confirmed by the non-parametric Spearman coefficient ($\rho=-0.74$, $p<0.001$). Vitamin D was also significantly and inversely correlated with the extent of disease measured by body surface area ($r=-0.64$, $p<0.001$) and with quality-of-life impairment measured by DLQI ($r=-0.54$, $p<0.001$), indicating that lower vitamin D accompanied both more extensive disease and greater patient-reported burden. In contrast, PASI was not correlated with disease duration ($r=0.004$, $p=0.96$), suggesting that severity in this cohort related to current vitamin D status rather than simply to how long patients had had the disease. The correlation findings are summarised in Table 4.

**Table 4: Correlation of serum 25(OH)D and related variables with clinical measures**

| Variable pair | Pearson r | p-value |
|---|---|---|
| Serum 25(OH)D vs PASI | −0.74 | <0.001 |
| Serum 25(OH)D vs PASI (Spearman ρ) | −0.74 | <0.001 |
| Serum 25(OH)D vs BSA involved | −0.64 | <0.001 |
| Serum 25(OH)D vs DLQI | −0.54 | <0.001 |
| PASI vs disease duration | 0.004 | 0.96 |

**Discussion**

In this hospital-based observational study of 180 Indian patients with chronic plaque psoriasis, lower serum vitamin D was strongly and consistently associated with greater disease severity. Mean 25(OH)D fell progressively from mild to moderate to severe disease, PASI rose stepwise from vitamin D-sufficient to deficient patients, and serum 25(OH)D correlated inversely with PASI ($r=-0.74$), body surface area ($r=-0.64$) and quality-of-life impairment ($r=-0.54$). The association between vitamin D status and severity category was highly significant, with severe disease occurring almost exclusively in deficient patients. Taken together, these internally consistent findings—convergent across continuous correlation, categorical comparison and reciprocal grouping—make a strong case for a genuine relationship between vitamin D status and psoriasis severity in this population.

These results are biologically coherent. The active vitamin D metabolite suppresses keratinocyte

hyperproliferation, promotes differentiation and exerts immunomodulatory effects that dampen the Th1/Th17 axis central to psoriasis, including the regulation of dendritic cell and T-cell function.[3,4] The same pathways explain why topical vitamin D analogues are effective antipsoriatic agents.[6,7] A relative deficiency of substrate for these regulatory actions could plausibly permit more florid disease, and our observation that severity tracked current vitamin D status rather than disease duration is consistent with an active, state-dependent influence rather than a mere epiphenomenon of chronicity.

Our findings align with a substantial body of clinical literature. Studies from several countries have reported lower serum 25(OH)D in psoriasis and an inverse correlation with PASI; notably, a Indian study reported a significant negative correlation between 25(OH)D and PASI together with raised inflammatory and oxidative-stress markers in more severe disease.[8] Case–control studies have similarly documented significantly reduced 25(OH)D in psoriatic patients relative to controls.[9] At the same time, the literature is not unanimous: some studies have found no significant difference from controls or no correlation with activity, particularly in populations where vitamin D deficiency is so prevalent in the general population that any disease-related difference is difficult to detect.[10,11] Our cohort, drawn from a region with widespread background hypovitaminosis D,[13] nonetheless demonstrated a clear gradient, which may reflect the wide spread of severity captured and the exclusion of patients on vitamin D supplementation, phototherapy or other treatments that raise 25(OH)D or modify disease.[15,16]

The high overall burden of hypovitaminosis D in our patients—70% deficient or insufficient—deserves emphasis in the South Indian context. Despite plentiful sunshine, deficiency is common across India owing to pigmented skin reducing cutaneous synthesis, indoor lifestyles, dietary patterns low in vitamin D and limited fortification.[13] In psoriasis specifically, additional disease-related factors such as photoavoidance, the chronic inflammatory state and, in some, obesity may compound the deficiency. The strong inverse association with DLQI further indicates that the patients with the lowest vitamin D were also those bearing the greatest psychosocial burden, reinforcing the clinical relevance of the finding beyond the visible plaque count.

From a practical standpoint, these data support incorporating assessment of vitamin D status into the evaluation of patients with moderate-to-severe psoriasis, particularly in regions of high background deficiency. They also provide a clear rationale for adequately powered, randomised interventional studies to test whether correction of deficiency with oral vitamin D, as an adjunct to standard therapy, improves disease severity—an approach suggested but not established by small uncontrolled reports of high-dose supplementation.[10]

**Limitations**

Several limitations must be acknowledged. First, and most importantly, the cross-sectional, observational design establishes association but cannot prove causation; the direction of the relationship cannot be determined, since severe inflammatory disease and its associated behaviours (photoavoidance, reduced outdoor activity) could themselves lower vitamin D. Second, the study was hospital-based and did not include a non-psoriatic comparison group, so the cohort's vitamin D levels cannot be benchmarked against the local general population. Third, although key confounders were addressed through exclusion criteria, residual confounding by season, sun exposure, body mass index, diet and skin phototype was not formally adjusted in a multivariable model and should be in future work. Fourth, a single serum 25(OH)D measurement may not capture long-term vitamin D status. Finally, the findings derive from a single centre and may not be generalisable to other settings. Prospective and interventional studies, ideally multicentre and with multivariable adjustment, are needed to confirm and extend these observations.

## Conclusion

Among South Indian patients with chronic plaque psoriasis, lower serum vitamin D was strongly and significantly associated with greater disease severity by PASI, more extensive body surface involvement and poorer quality of life, with severe disease occurring almost exclusively in vitamin D-deficient patients. Hypovitaminosis D was highly prevalent in this cohort. While the observational design precludes causal conclusions, the consistency and strength of the association, together with the established immunoregulatory role of vitamin D in psoriasis, support routine assessment of vitamin D status in patients with moderate-to-severe disease and justify well-designed interventional trials of supplementation as an adjunct to standard treatment.

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