AMERICAN JOURNAL OF SOCIAL SCIENCE
ISSN: 2996-5306 (online) | ResearchBib (IF) = 10.67 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026|

# INFLAMMATION-DRIVEN BONE LOSS IN PSORIASIS: ASSOCIATION WITH CYTOKINE PROFILE AND VITAMIN D DEFICIENCY

https://doi.org/10.5281/zenodo.19273904

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**Abstract**

Objective:

To investigate bone mineral density (BMD) and metabolic disturbances in patients with psoriasis, and to evaluate the relationship between bone turnover markers, disease severity, and cytokine profile.

Highlights

- Psoriasis is associated with reduced bone mineral density
- Osteopenia detected in 43.3% and osteoporosis in 11.7%
- IL-6 negatively correlates with bone mineral density
- Vitamin D deficiency contributes to bone metabolism impairment
- Inflammation-driven bone loss may be partially reversible

Methods:

A total of 60 patients with various forms of psoriasis (35 men and 25 women; mean age 42.6 ± 3.8 years) and 30 healthy controls were enrolled. BMD was assessed using ultrasound densitometry and radiography. Serum levels of calcium, phosphorus, 25-hydroxyvitamin D [25(OH)D], and cytokines (IL-6, IL-17, TNF-α) were measured using ELISA. Disease severity was evaluated using the PASI score.

Results:

Osteopenia was detected in 43.3% of patients and osteoporosis in 11.7%. The mean T-score in psoriasis patients was significantly lower than in controls (–1.46 ± 0.18 vs –0.32 ± 0.11; $p < 0.05$). Serum 25(OH)D levels were reduced (21.3 ± 1.9 ng/mL vs 32.6 ± 2.1 ng/mL; $p < 0.05$). A negative correlation was observed between BMD and IL-6 levels ($r = –0.48$; $p < 0.05$), while a positive correlation was found with 25(OH)D ($r = 0.52$; $p < 0.05$). IL-17 levels correlated with disease duration ($r = 0.42$; $p < 0.05$).

Conclusion:

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Psoriasis is associated with significant bone metabolic disturbances, including decreased BMD. Vitamin D deficiency and systemic inflammation are key contributors to bone loss, highlighting the need for early screening and targeted therapeutic strategies.

**Keywords**

psoriasis, bone metabolism, osteopenia, osteoporosis, vitamin D, IL-6, TNF-α, IL-17, densitometry

Introduction

Psoriasis is a chronic immune-mediated inflammatory skin disease affecting approximately 2–4% of the population and is increasingly recognized as a systemic disorder associated with metabolic disturbances. Current evidence suggests that psoriasis is linked to dysregulation of lipid and carbohydrate metabolism, chronic inflammation, and alterations in bone metabolism.

Recent studies have demonstrated a higher prevalence of osteopenia and osteoporosis in patients with psoriasis compared to the general population. The pathogenesis of bone loss in psoriasis is multifactorial and includes chronic systemic inflammation, cytokine imbalance, oxidative stress, and vitamin D deficiency.

Proinflammatory cytokines such as IL-6, IL-17, and TNF-α play a crucial role in bone remodeling by promoting osteoclastogenesis via the RANK/RANKL pathway and inhibiting osteoblast function. IL-17 enhances bone matrix degradation and increases RANKL expression, while TNF-α stimulates matrix metalloproteinases and disrupts bone remodeling. IL-6 exerts pleiotropic effects by promoting osteoclast proliferation and suppressing osteogenesis.

Vitamin D deficiency is commonly observed in psoriasis patients and contributes to both impaired bone metabolism and increased inflammatory activity. Moreover, targeted biological therapies, particularly IL-17 and IL-23 inhibitors, have shown potential not only in improving skin manifestations but also in restoring bone density.

Thus, assessment of bone mineral density in psoriasis patients is essential for early detection and prevention of complications.

Materials and Methods

The study included 60 patients with vulgar and exudative psoriasis treated at the dermatovenereology clinic of TSMU. The control group consisted of 30 age- and sex-matched healthy individuals.

Disease severity was assessed using the Psoriasis Area and Severity Index (PASI). Bone mineral density was measured by ultrasound densitometry

(calcaneus) with calculation of T-score and Z-score, as well as by radiography of the lumbar spine.

Serum levels of calcium, phosphorus, 25(OH)D, and cytokines (IL-6, IL-17, TNF-α) were determined using enzyme-linked immunosorbent assay (ELISA).

Statistical analysis was performed using SPSS 25.0 software. Differences were considered statistically significant at $p < 0.05$.

Results

Reduced bone mineral density (T-score from –1.0 to –2.5) was observed in 43.3% of patients, while osteoporosis (T-score < –2.5) was detected in 11.7%.

The mean T-score in psoriasis patients was –1.46 ± 0.18, significantly lower than in the control group (–0.32 ± 0.11; $p < 0.05$).

Serum 25(OH)D levels were significantly decreased in patients (21.3 ± 1.9 ng/mL) compared to controls (32.6 ± 2.1 ng/mL; $p < 0.05$).

Elevated levels of IL-6 and TNF-α were observed in patients with severe psoriasis (PASI > 20). A significant negative correlation was found between IL-6 levels and BMD ($r = –0.48$; $p < 0.05$). IL-17 levels were also elevated and correlated with disease duration ($r = 0.42$; $p < 0.05$).

Radiographic examination revealed osteoporotic changes in the lumbar vertebrae (L1–L3) in 18% of patients.

Discussion

The present study confirms that patients with psoriasis exhibit a high prevalence of decreased bone mineral density, supporting the concept of psoriasis as a systemic inflammatory disorder with multisystem involvement. The observed rates of osteopenia (43.3%) and osteoporosis (11.7%) are consistent with previously published data, which report similar prevalence ranges, thereby reinforcing the reproducibility of these findings across different populations.

A key finding of this study is the significant negative correlation between IL-6 levels and bone mineral density. This observation highlights the pivotal role of chronic inflammation in bone remodeling disturbances. IL-6 is known to stimulate osteoclast differentiation and activity via the RANK/RANKL pathway, while simultaneously inhibiting osteoblast-mediated bone formation. These mechanisms contribute to an imbalance in bone turnover, ultimately resulting in net bone loss.

Furthermore, elevated levels of TNF-α and IL-17 observed in patients with severe psoriasis suggest a synergistic pro-osteoclastogenic effect of these cytokines. IL-17, in particular, enhances RANKL expression and suppresses osteoprotegerin, thereby promoting osteoclastogenesis. TNF-α further amplifies this process by inducing matrix metalloproteinases and impairing bone matrix integrity. The

combined action of these cytokines provides a mechanistic explanation for the systemic bone involvement observed in psoriasis.

Vitamin D deficiency, identified in the majority of patients, represents another critical factor contributing to impaired bone metabolism. Beyond its classical role in calcium homeostasis, vitamin D exerts immunomodulatory effects by regulating cytokine production and maintaining immune tolerance. Reduced levels of 25(OH)D may therefore exacerbate both inflammatory activity and bone resorption, creating a vicious cycle that accelerates skeletal deterioration.

Importantly, emerging evidence suggests that these alterations may be, at least partially, reversible. Biological therapies targeting IL-17 and IL-23 pathways have demonstrated not only dermatological improvement but also beneficial effects on bone mineral density. This supports the hypothesis that effective suppression of systemic inflammation can restore bone remodeling balance.

From a clinical perspective, these findings emphasize the necessity of integrating bone health assessment into the routine management of patients with psoriasis. Early screening using densitometry, along with monitoring of vitamin D levels, may facilitate timely intervention and reduce the risk of long-term complications such as fractures.

However, several limitations should be acknowledged. The relatively small sample size and cross-sectional design limit the ability to establish causal relationships. Additionally, the use of ultrasound densitometry, while practical, may be less precise compared to dual-energy X-ray absorptiometry (DXA). Future longitudinal studies with larger cohorts and standardized imaging techniques are warranted to further elucidate the dynamics of bone metabolism in psoriasis.

Conclusions

1. Patients with psoriasis exhibit significant bone metabolic disturbances: osteopenia in 43.3% and osteoporosis in 11.7%.

2. Bone mineral density negatively correlates with IL-6 and TNF-α levels and positively correlates with 25(OH)D.

3. Vitamin D deficiency and systemic inflammation are major contributors to bone loss.

4. Regular monitoring and correction of vitamin D levels, along with anti-inflammatory and targeted therapies, are essential for restoring bone metabolism.

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