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*Original Article*

# Longitudinal Changes in Serum 25-hydroxyvitamin D Levels after Periodontal Therapy in Patients with Chronic Periodontitis: A Single-arm Prospective Study

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## ABSTRACT

**Background and Objectives:** Chronic periodontitis is a prevalent inflammatory disease driven by dysbiotic subgingival biofilms, causing progressive destruction of the periodontal ligament and alveolar bone. Vitamin D (25-hydroxyvitamin D) may affect periodontal health via immunomodulatory and anti-inflammatory effects on host responses. However, if standard non-surgical or surgical periodontal therapy alters systemic vitamin D concentrations is still controversial due to previous conflicting evidence. This single-arm prospective study assessed longitudinal changes in serum vitamin D levels in patients with chronic periodontitis.

**Materials and Methods:** Thirty-two systemically healthy patients with chronic periodontitis were enrolled. Treatment included scaling and root planing, with surgical intervention as clinically indicated. Serum 25-hydroxyvitamin D was quantified via chemiluminescence immunoassay at the baseline, 1 m and 3 m. Repeated-measures ANOVA with Bonferroni adjustment was used ($\alpha = 0.05$).

**Results:** Mean serum vitamin D levels were 24.7(9.2), 22.8(7.9) and 20.6(8.6) ng/ml at the baseline, 1 m and 3 m, respectively. A significant time-dependent decrease was observed [$F(1.08,33.49) = 6.83$, $P = 0.012$, $\eta^2p = 0.181$]. The 3-m levels were significantly lower than the baseline ($P = 0.036$) and 1-m ($P = 0.044$) levels; the 1-m decrease was not significant ($P = 0.061$).

**Conclusions:** Within the limitations of this single-arm design, serum vitamin D significantly decreased post therapy. Without a control group or adjustment for seasonal, dietary or sunlight confounders, causal attribution is impossible. Periodontal therapy alone appears insufficient to increase systemic vitamin D, reinforcing the importance of independent nutritional assessment and targeted supplementation, particularly in patients with baseline insufficiency. These observational findings needs verification through randomized controlled trials.

**Keywords:** Vitamin D, 25-hydroxyvitamin D, Chronic periodontitis, Periodontal index

## Highlights

- Periodontitis is a multifactorial inflammatory disease induced by bacterial plaque. This is characterized by progressive destruction of the tooth-supporting structures, including the periodontal ligament and alveolar bone.
- The relationship between periodontal and systemic diseases may occur through two major mechanisms that often overlap.
- The vitamin plays essential biological roles in bone mineralization and preservation of calcified tissues, including teeth. In addition to its classical skeletal functions, vitamin D exerts pleiotropic immunomodulatory effects.
- Serum vitamin D significantly decreased post periodontal therapy.

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## Introduction

Periodontitis is a multifactorial inflammatory disease induced by bacterial plaque, containing *Porphyromonas gingivalis, Tannerella forsythia, Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans*, with their metabolic byproducts. This is characterized by progressive destruction of the tooth-supporting structures, including the periodontal ligament and alveolar bone (1). Chronic periodontitis progresses gradually if untreated, while aggressive periodontitis demonstrates rapid bone loss with a familial pattern. Based on the percentage of tooth involvement and attachment loss (mild, 1–2 mm; moderate, 3–4 mm; and severe: > 5 mm), chronic periodontitis can be localized or generalized (2). The relationship between periodontal and systemic diseases may occur through two major mechanisms that often overlap. The first involves residual oral infection acting as microbial reservoir for systemic pathogens, while the second involves inflammatory bacterial products and cytokines from diseased periodontal tissues entering systemic circulation, contributing to systemic inflammation and increasing the risk of comorbid inflammatory diseases (3).

The goal of periodontal therapy is to preserve natural dentition by achieving and preserving periodontal health (4). Treatment encompasses a stepwise approach aimed at infection control and inflammation resolution. The initial phase involves supragingival and subgingival biofilm control through patient education, professional debridement, correction of overhanging restorations and elimination of risk factors such as smoking (4,5). When necessary, surgical intervention provides access for scaling and root planing and recontouring gingival morphology. While surgery yields better outcomes in deep pockets (> 5 mm), long-term difference between surgical and non-surgical therapy diminishes with appropriate oral hygiene (6,7).

The 25-hydroxyvitamin D or vitamin D, known as the "sunshine vitamin," is a secosteroid prohormone synthesized by skin cells upon ultraviolet exposure or achieved from dietary sources such as fish, red meat and eggs (8,9). The vitamin plays essential biological roles in bone mineralization and preservation of calcified tissues, including teeth (8). In addition to its classical skeletal functions, vitamin D exerts pleiotropic immunomodulatory effects, including regulation of antimicrobial peptide production, modulation of dendritic cell maturation and suppression of proinflammatory cytokine secretion (8). Genetic polymorphisms of the vitamin D receptor (**VDR**) have been associated with increased susceptibility to periodontitis, greater alveolar bone loss and further pronounced clinical attachment loss, further supporting a biologically likely role for vitamin D in periodontal health maintenance (10). Furthermore, individuals with sufficient vitamin D levels demonstrate better periodontal healing outcomes (11). Despite this mechanistic possibility, findings regarding the association between vitamin D deficiency and the onset or progression of periodontitis are inconsistent in the literature (12–16). The effect of periodontal therapy on systemic vitamin D concentrations has yielded conflicting results. Previous investigations demonstrated a significant decrease in serum vitamin D levels after non-surgical periodontal treatment, suggesting that the resolution of local inflammation may downregulate systemic vitamin D metabolism (17,18), In contrast, other studies reported increased serum or gingival crevicular fluid vitamin D concentrations post therapy, potentially reflecting decreased local consumption or improved tissue repair (9). These discordant findings might be attributable to differences in study designs, baseline vitamin D statuses, patient populations and timing of post-treatment assessments. Regarding these unresolved discrepancies, the present longitudinal study was designed to assess serial changes in serum 25-hydroxyvitamin D concentrations before and after comprehensive periodontal therapies in patients with chronic periodontitis. The study hypothesized that if periodontal treatment modulated systemic inflammation, this might be reflected in corresponding changes in circulating vitamin D levels over time.

## Materials and Methods

### Study Design and Ethical Considerations

This single-arm prospective longitudinal study was carried out on patients diagnosed with chronic periodontitis, who were referred to the Department of Periodontics, Guilan University of Medical Sciences, Rash, Iran. None of the participants was received prior periodontal therapy. The study design and implementation strictly adhered to the ethical principles outlined in the Declaration of Helsinki for medical research involving human subjects. Written informed consent was collected from all participants after a full explanation of the study objectives, procedures, potential risks and benefits. The research protocol was reviewed and approved by the Ethics Committee of Guilan University of Medical Sciences (IR.GUMS.REC.1402.574) and registered in the Iranian Registry of Clinical Trials (IRCT20240212060975N1). Confidentiality of patient information was served throughout the study and participants were free to withdraw at any stage without prejudice or effect on their subsequent care.

### **Sample Size** Calculation

Sample size was calculated based on a repeated-measures ANOVA design. Assuming moderate effect size, alpha level of 0.05, statistical power of 0.80 and three repeated measurements with correlation of 0.5 within time points, sample size was calculated as 28 participants using G*Power software v.3.1.9.7 (Universitat Dusseldorf, Germany). Accounting for an anticipated 10% attrition rate, a total of 32 participants were enrolled to ensure adequate statistical power for the primary outcome analysis.

### Participants

Diagnosis of chronic periodontitis was carried out by a dental specialist based on comprehensive clinical and radiographic assessments demonstrating periodontal ligament and alveolar bone loss. Inclusion criteria were (a) presence of subgingival calculus and chronic inflammation with mild to moderate progression, (b) predominantly horizontal patterns of alveolar bone loss

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affecting at least 30% of sites, (c) probing pocket depths ≥ 4 mm with bleeding on probing at a minimum of six non-adjacent teeth, (d) age of 18–65 years old, and (e) possession of at least 16 natural teeth (excluding third molars).

Exclusion criteria were (a) systemic disorders affecting bone metabolism (e.g., uncontrolled diabetes mellitus, osteoporosis, hyperparathyroidism and renal osteodystrophy), (b) history of malignancy within the past five years, (c) current pregnancy or lactation, (d) use of medications affecting calcium or vitamin-D metabolism within the previous six months (including systemic antibiotics, bisphosphonates, barbiturates, cardiac glycosides, cholestyramine, glucocorticoids, anticonvulsants and laxatives), (e) current or prior vitamin-D supplementation exceeding 400 IU $d^{-1}$ within the three months prior enrollment, (f) history of periodontal therapy within the last 12 months, (g) smoking more than ten cigarettes per day, (h) body mass index ≥ 30 kg per m², and (i) conditions limiting the ability to provide informed consent or comply with the study procedures. All participants were in generally good systemic health as verified using medical history review and, when indicated, consultation with the participant's primary care physician.

**Intervention**

A total of 32 eligible patients were selected for scaling and root planing (SRP) and periodontal surgery was carried out when indicated.

**Serum** Vitamin-**D Assessment**

Peripheral venous blood samples (5 ml) were collected from each participant at three time points of (a) baseline (immediately prior to initiation of SRP), (b) 1-m post treatment (4 w ±1 after completion of the active treatment phase), and (c) 3-m post treatment (12 w ±1 after completion of the active treatment phase). All blood samples were collected between 7:00 and 9:00 AM after an overnight fast of at least 8 h to minimize diurnal variation and dietary effects. Samples were transported to the central laboratory within 2 h of collection, centrifuged at 3,000 rpm for 10 min and serum aliquots were stored at -80 °C until analysis. Serum 25-hydroxyvitamin D [25(OH) D] concentrations were assessed using commercially available chemiluminescence immunoassay (Abbott, USA). The assay included a detection range of 8.8–385.5 ng $ml^{-1}$, with intra-assay and inter-assay coefficients of variation of less than 2.6–4.1 and 3.0–5.1%, respectively, as reported by the manufacturer. All samples from each participant were analyzed in a single batch at the end of the study to eliminate inter-assay variability. Vitamin-D status was categorized based on the Endocrine Society Clinical Practice Guidelines as deficiency (< 20 ng $ml^{-1}$), insufficiency (20–29 ng $ml^{-1}$) and sufficiency (≥ 30 ng $ml^{-1}$) (3).

**Statistical Analysis**

All statistical analyses were carried out using IBM SPSS Statistics for Windows v.26.0 (IBM USA). Normality of continuous variables was assessed using Shapiro-Wilk test. Data were expressed as mean ±SD (standard deviation) for normally distributed continuous variables, median with interquartile range (IQR) for non-normally distributed variables and frequency with percentage for categorical variables.

Repeated-measures analysis of variance (ANOVA) was used to assess differences in serum 25(OH)D concentrations between the three time points (the baseline, 1 m and 3 m). Pairwise comparisons between the time points were carried out using Bonferroni post-hoc correction to control for Type I error. Statistical significance was set at a two-tailed *P*-value < 0.05.

## Results

**Participant Characteristics and Treatment Completion**

A total of 32 patients (18 females, 56.3%; 14 males, 43.7%) with a mean age of 46.03 y ±12.66 (range of 24–65 y) were enrolled and completed the study protocol. No participants withdrew or were lost to follow-up, resulting in a 100% retention rate. Baseline demographic and clinical characteristics are summarized in Table 1. The mean body mass index (BMI) was 25.9 kg per m² and six patients (18.7 %) reported a history of light smoking (≤ 5 cigarettes $d^{-1}$). All participants reported no vitamin-D supplementation within the last 3 m and continued consistent dietary and physical activity habits throughout the study.

**Table 1.** Comparison of serum vitamin-D levels in patients with chronic periodontitis before and after periodontal therapy

| Variables | Mean (SD) (ng/mL) | Statistical Comparison | P-value |
|---|---|---|---|
| Baseline (Pre-treatment) | 24.7 ( 9.2) | - | - |
| 1 Month Post-treatment | 22.8 (7.9) | Compared with Baseline | 0.061 |
| 3 Months Post-treatment | 20.6 (8.6) | Compared with Baseline | 0.036 |
| | | Compared with 1 Month | 0.044 |
| Repeated-Measures ANOVA | F(1.08, 33.49) = 6.83 | $\eta^2p$ = 0.181 | 0.012 |

**Note:** Partial eta squared ($\eta^2p$) = 0.181 indicates a large effect size (small = 0.01–0.06; medium = 0.06–0.14; large > 0.14); Values are presented as mean (SD); ANOVA: ANOVA: Analysis of variance; $\eta^2p$: Partial eta squared; SD: standard deviation; P-value of less than 0.05, was considered statistically significant.

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**Serum Vitamin-D Levels Over Time**

Mean serum 25-hydroxyvitamin D [25(OH)D] concentrations at the baseline, 1-m post treatment and 3-m post treatment were 24.7 ±9.2 ng/ml, 22.8 ±7.9 ng/ml and 20.6 ±8.6 ng/ml, respectively. At the baseline, ten patients (31.25%) were vitamin-D deficient (< 20 ng/ml), 12 patients (37.5%) were vitamin-D insufficient (20–29 ng/ml) and ten patients (31.25%) were vitamin-D sufficient (≥ 30 ng/ml). By 3-m post-treatment, the number of deficient patients increased to 18 (56.25%), insufficient patients decreased to ten (31.25 %) and sufficient patients decreased to five (12.5%).

Repeated-measures ANOVA demonstrated a statistically significant difference in mean serum vitamin-D levels within the three time points [F (1.08, 33.49) = 6.83, $P$ = 0.012, $\eta^2p$ = 0.181], indicating a progressive decrease over time. The effect size (partial eta squared = 0.181) corresponded to a large effect magnitude according to Cohen's conventions ($\eta^2p$ > 0.14). Bonferroni-adjusted pairwise comparisons revealed that the mean vitamin-D concentration at 3 m post treatment was significantly lower than the two others; however, this decrease did not reach statistical significance ($P$ = 0.061) (Figure 1). The most pronounced decrease in serum vitamin D occurred between the 1-m and 3-m time points, suggesting a continued downward trajectory beyond the immediate post-treatment time.

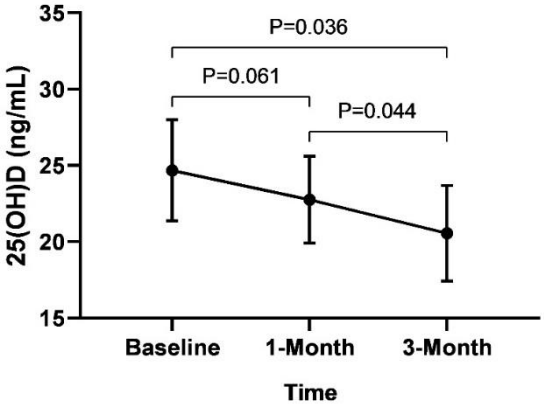



**Figure 1.** Mean serum vitamin-D levels before, 1 m after and 3 m after periodontal therapy. 25(OH)D, 25-Hydroxyvitamin D (vitamin D); ANOVA, analysis of variance. Data are present as mean ±95% confidence interval. *P*-values were calculated using repeated-measures analysis of variance (ANOVA) and Bonferroni post-hoc tests for pairwise comparisons. *P*-value of less than 0.05 was considered statistically significant.

## Discussion

The present single-arm prospective longitudinal study assessed serial changes in serum 25-hydroxyvitamin D concentrations in patients with chronic periodontitis after comprehensive periodontal therapy. The principal finding included significant progressive decreases in serum vitamin-D levels over a 3-m monitoring time, with mean concentrations decreasing from 24.7 ng/ml at the baseline to 20.6 ng/ml at 3-m post treatment ($P$ = 0.012). This decrease was most pronounced between the 1-m and 3-m time points, suggesting a sustained downward trajectory rather than a transient post-treatment fluctuation. Importantly, these changes occurred despite clinically significant improvements in all periodontal parameters.

The decrease in serum vitamin D after periodontal therapy was similar to that of several previous investigations. Liu et al. (19) reported decreased serum 25-hydroxyvitamin $D_3$ levels in patients with aggressive periodontitis after initial non-surgical therapy, accompanied by decreased local and systemic interleukin-1β (IL-1β) concentrations. Similarly, Ribeiro LSF et al. (18) demonstrated that periodontal healing outcomes were associated to baseline vitamin-D status, with lower serum levels observed post treatment. Yuce HB et al. (20) also documented decreases in local and systemic 25-hydroxyvitamin D in patients with periodontitis and rheumatoid arthritis (RA) following periodontal intervention. The convergence of these findings in diverse populations and study designs supported the hypothesis that periodontal therapy, while effective in decreasing local inflammation, might be associated to decreased systemic vitamin-D concentrations while samples were collected during the winter and spring to decrease the effects of environment.

In contrast, Costantini et al. observed that patients with periodontitis demonstrated higher levels of proinflammatory cytokines and lower vitamin-D concentrations in saliva, compared with healthy individuals (21). Similarly, Faramarzi et al. detected significantly lower serum vitamin-D levels within affected individuals (24.98 ng/ml) than within controls (35.61 ng/ml). The discrepancies between these studies and the present study might originate from methodological differences. The aforementioned investigations used healthy control groups, whereas the present research followed a single-group design, including patients with chronic periodontitis (22). Furthermore, Costantini et al. and Faramarzi et al. (21,22) carried out single-time-point assessments without therapeutic intervention, whereas the current study monitored serum vitamin-D levels longitudinally at the baseline, 1 m and 3 m after treatment.

In contrast to the present findings, Ghalwash et al. reported a significant post-treatment increase in serum and gingival crevicular fluid (GCF) vitamin-$D_3$ levels, accompanied by a significant decrease in fibroblast growth factor 23 (FGF23) after non-surgical periodontal therapy. Their results indicated that vitamin $D_3$ was positively associated to periodontal health, whereas FGF23 correlated with periodontal inflammation (9). The discrepancy between their findings and the present ones might be attributed to differences in study design population characteristics, baseline vitamin-D status, dietary habits or environmental factors such as sunlight exposure.

The mechanistic basis for the reported decrease in serum vitamin D after periodontal therapy warranted careful monitoring. Vitamin D plays a vital role in modulating inflammatory cytokines, including the downregulation of IL-1β, tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) through nuclear factor-κB (NF-κB) pathway inhibition (23). Since IL-1β and other

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proinflammatory cytokines typically decrease following effective periodontal therapy, the concurrent decrease in circulating vitamin D might reflect the downregulation of systemic inflammatory pathways during the healing phase. This interpretation is similar to "consumption hypothesis," wherein active inflammation drives increased local utilization and catabolism of vitamin D and resolution of inflammation decreases this demand, potentially leading to lower circulating levels as balance is re-established. However, this is a hypothesis that needs direct verification through simultaneous measurement of inflammatory biomarkers.

An alternative, though not mutually exclusive, explanation involves vitamin-D binding protein (VDBP). The VDBP, which transports vitamin D and its metabolites, has been shown to increase in patients with aggressive periodontitis, compared with healthy individuals (24,25). This mechanism might partly explain the decrease in circulating vitamin D after treatment, as osteoclastic activity and local inflammatory turnover decrease during resolution. It is also important to address the role of FGF23, which Ghalwash et al. (9) identified as inversely correlated with vitamin-D status. The FGF23 regulates phosphate homeostasis and vitamin-D metabolism by suppressing renal 1α-hydroxylase activity; thereby, decreasing active vitamin-D production. Periodontal inflammation might affect FGF23 expression through inflammatory cytokines and treatment-linked decreases in inflammation could theoretically modify FGF23 levels, with downstream effects on vitamin-D metabolism. The absence of FGF23 assessment in the present study represents a limitation that should be addressed in further investigations.

While the biological mechanisms described previously provide possible explanations for the observed decrease, several alternative explanations must be addressed. Regarding the single-arm design of this study, the authors could not exclude the possibility that the observed decrease reflected seasonal variation, changes in dietary vitamin-D intake or decreased sunlight exposure during the study. Overall, the present findings indicated that although periodontal therapy effectively decreased inflammation and improved periodontal health, it did not appear to enhance systemic vitamin-D levels. This suggested that preserving adequate vitamin-D status might need adjunctive nutritional or lifestyle interventions in addition to periodontal treatment.

A key strength of this study is linked to its longitudinal design, standardized clinical procedures and use of repeated serum assessments, which allowed temporal assessment of treatment effects. However, absence of a control group, limited sample size and lack of assessment of confounding factors such as sunlight exposure, diet and seasonal variation might affect the reported outcomes. Further investigations should use multicenter randomized controlled trials (RCT) with larger and further diverse populations, incorporating serum and gingival crevicular fluid analyses. Such studies can help delineate the local versus systemic dynamics of vitamin D in periodontal healing and clarify if supplementation can enhance therapeutic outcomes in vitamin-D deficient individuals.

**Conclusion**

Within the limitations of this single-arm prospective longitudinal study, serum 25-hydroxyvitamin D concentrations demonstrated progressive and statistically significant decreases over a 3-m time after comprehensive periodontal therapy. This decrease occurred despite clinically meaningful improvements in all periodontal parameters, verifying treatment efficacy. The decrease was similar to that of several previous reports but in contrast to others, highlighting the need of further investigations. Although the mechanistic basis was speculative, possible explanations included decreased systemic inflammation leading to decreased vitamin-D consumption or altered VDBP-mediated transport. Critically, periodontal therapy alone did not seem to enhance systemic vitamin-D status, reinforcing the importance of independent nutritional assessment and targeted supplementation, particularly in vitamin-D deficient or insufficient patients. Clinicians should monitor vitamin-D status as a part of comprehensive periodontal care, especially in populations at risk for the vitamin deficiency. Further RCTs with larger, further diverse populations and comprehensive mechanistic assessments are warranted to clarify the causal relationships between periodontal therapy, inflammation resolution and vitamin-D metabolism.

**Declarations**

**Ethics approval and consent to participate**

Ethical approval to carry out the present study was granted by the Ethics Committee of Guilan University of Medical Sciences (ethical code: IR.GUMS.REC.1402.574) and all participants were fully informed about the aim of the study and the voluntary nature of participation. This study was carried out in accordance with the ethical principles outlined in Declaration of Helsinki.

**Consent for publication**

Not applicable.

**Clinical trial number**

This study registered in the Iranian Registry of Clinical Trials https://irct.behdasht.gov.ir/trial/75491 with registration no. IRCT20240212060975N1.

## Financial disclosure

Data present in this study are available on request from the corresponding author.

**Declaration of competing interest**

The authors declare no known competing financial interest or personal relationship that could affect this study.

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CRediT authorship contribution statement

Conceptualization :, Data curation :, Formal analysis :, Funding acquisition : No funding, Investigation :, Methodology :, Project administration :, Resources : FMGH, MK, FJ, Software : Supervision :, Validation :,

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