



(CASE REPORT)



Therapeutic Role of Vitamin D Supplementation in Periodontitis: A Case-Based Approach

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Abstract

Generalized periodontitis is a common chronic inflammatory disease that may be aggravated by systemic conditions such as vitamin D deficiency. This report describes a 43-year-old male who presented with bleeding gums, discomfort during chewing, and tooth mobility, with clinical and radiographic findings consistent with Stage III, Grade A generalized periodontitis. The management plan combined nonsurgical and surgical periodontal therapy with adjunctive vitamin D supplementation. On follow-up, the patient showed marked improvements in clinical attachment and reduction in inflammation, underscoring the importance of assessing and addressing vitamin D status to optimize periodontal treatment outcomes even in populations where deficiency may be overlooked.

Keywords: Periodontitis; Vitamin D; Bone metabolism; Antimicrobial peptides

1. Introduction

Periodontitis is a chronic inflammatory condition that progressively damages the tissues supporting the teeth, including the gingiva, periodontal ligament, and alveolar bone. It is primarily triggered by microbial imbalance in dental plaque, but its progression is heavily influenced by host immune responses and systemic factors. Generalized periodontitis, which affects multiple teeth across the mouth, is one of the most common forms and can significantly impair oral function and quality of life [1].

Vitamin D has emerged as a key factor in maintaining periodontal health due to its dual role in local tissue regulation and systemic immune modulation. Locally, vitamin D contributes to the integrity of the gingival epithelium and helps regulate inflammatory mediators [2]. Systemically, it supports bone metabolism and immune surveillance, both of which are essential for periodontal stability [3]. The presence of vitamin D receptors (VDRs) in gingival tissues and immune cells such as macrophages and T lymphocytes suggests that vitamin D functions not only as a nutrient but also as a signalling molecule in periodontal defense mechanisms [4].

In individuals with generalized periodontitis, persistent inflammation leads to the degradation of connective tissue and alveolar bone. Vitamin D deficiency may worsen this process by impairing the resolution of inflammation and reducing the regenerative capacity of periodontal structures [2]. Research has shown that vitamin D can inhibit nuclear factor-kappa B (NF- κ B) signalling, a pathway responsible for producing inflammatory cytokines and matrix metalloproteinases (MMPs), which contribute to tissue breakdown [2].

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Vitamin D also influences the activity of osteoblasts and osteoclasts, thereby regulating bone remodelling. Since bone loss is a hallmark of periodontitis, maintaining adequate vitamin D levels is crucial for preserving bone density. A study found that individuals with higher serum 25hydroxycholecalciferol levels had significantly less alveolar bone loss, indicating a protective effect of vitamin D on periodontal tissues [3].

Additionally, vitamin D enhances the production of antimicrobial peptides such as cathelicidin and defensins, which help control bacterial populations in the oral cavity [5]. This antimicrobial action complements mechanical periodontal therapies and may reduce bacterial load in periodontal pockets. Patients with sufficient vitamin D levels have shown better clinical outcomes following periodontal treatment, including reduced bleeding on probing and improved attachment levels [6].

Despite abundant sunlight, vitamin D deficiency remains widespread due to factors such as skin pigmentation, limited outdoor activity, and dietary habits [7]. This highlights the importance of routine screening and supplementation, especially for individuals with chronic inflammatory conditions like generalized periodontitis.

Incorporating vitamin D assessment into periodontal diagnostics offers a more comprehensive approach to patient care. Identifying and correcting deficiencies may enhance the effectiveness of conventional therapies and reduce the risk of disease recurrence [2]. As research continues to uncover the molecular pathways influenced by vitamin D, its role in periodontal medicine is becoming increasingly significant, bridging the gap between nutrition, immunology, and oral health.

2. Case report

A 43-year-old male patient named Mr. Shanmugam reported to the Department of Periodontics at RVS Dental College & Hospital with a primary complaint of bleeding gums in the anterior region, especially during brushing, which had persisted for the past year. He also experienced discomfort during chewing and noted occasional mobility of his teeth.

His medical history was non-contributory, with no systemic illnesses reported. Dental history revealed the extraction of tooth 16 five years prior and a recent oral prophylaxis performed one month ago. He brushed once daily using horizontal strokes for approximately four minutes and replaced his toothbrush every three months. His father had a history of diabetes and hypertension.

On clinical examination, the extraoral findings were unremarkable. Intraorally, the patient presented with generalized rolled-out marginal gingiva, bulbous interdental papillae, and gingival recession classified as Class I in multiple teeth, Class II in tooth 26, and Class III in tooth 46. The gingiva appeared soft, edematous, and tender, with generalized bleeding on probing. Tooth mobility was noted as Grade I in teeth 31, 32, 42, and 12, and Grade II in teeth 41 and 13. The fremitus test was positive, and inadequate attached gingiva was observed in relation to teeth 31 and 41. The tension test was also positive.

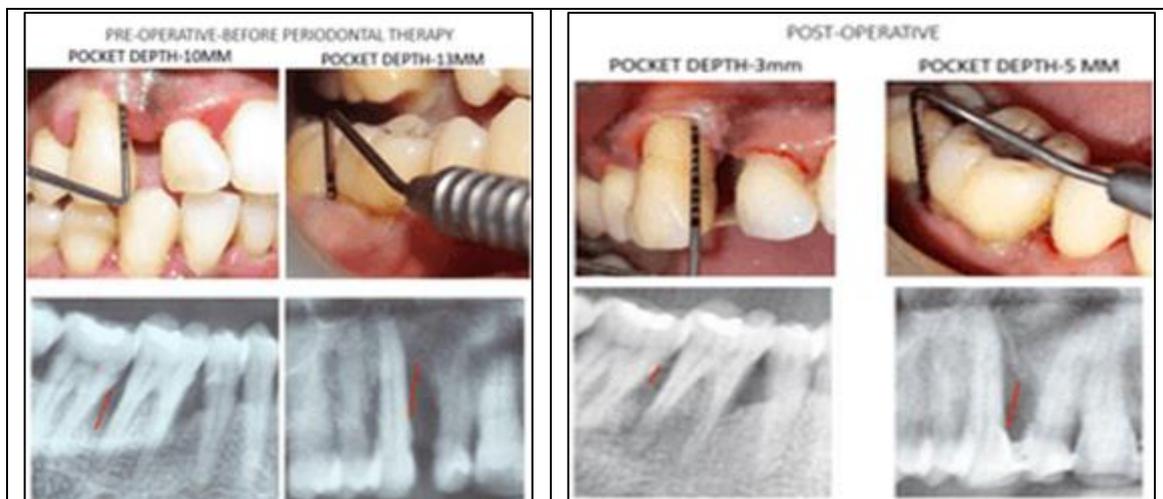


Figure 1 Pre operative image

Figure 2 Post operative image

Radiographic evaluation using full-mouth IOPA and OPG revealed generalized horizontal bone loss, with Grade I furcation involvement in molars including 17, 26, 27, 28, 37, and 38. Tooth 16 was missing, and loss of contact was noted between teeth 12 and 13, as well as 41 and 42.



Figure 3 OPG of the patient

Blood investigations showed no systemic abnormalities, and all parameters were within normal limits.

Table 1 Serum vitamin d level pre and post operative values

Values	Specimen	Parameter	Observed value	Unit
PRE OPERATIVE VALUE	SERUM	25-OH VITAMIN D	13.9	ng/mL
POST OPERATIVE VALUE	SERUM	25-OH VITAMIN D	34	ng/mL

Based on the clinical and radiographic findings, the diagnosis was chronic generalized periodontitis according to the 1999 classification, and Stage III, Grade A periodontitis as per the 2018 classification. The overall prognosis was fair, although individual prognosis for teeth 41 and 13 was considered poor.

The treatment plan was structured into four phases. Phase I involved etiologic therapy, including full-mouth scaling and root planing, oral hygiene instruction, full-mouth curettage, coronoplasty of teeth 31 and 41, and splinting of teeth 13 to 23 and 33 to 43. Phase II included surgical intervention with open flap debridement in all quadrants and regenerative procedures in relation to teeth 13, 21, 41, 46, and 47. Phase III focused on restorative care, specifically the replacement of missing tooth 16. Phase IV emphasized maintenance, with re-evaluation scheduled after 3–4 weeks and regular recall visits every three months to monitor oral hygiene, gingival inflammation, pocket depth, clinical attachment levels, tooth mobility, and occlusion.



Figure 4 Papilla preservation flap with placement of bone graft and gtr irt 12,13

3. Discussion

Vitamin D synthesis primarily occurs in the skin, where ultraviolet B (UVB) rays transform 7-dehydrocholesterol into cholecalciferol (vitamin D₃) [7]. While this photochemical reaction is the dominant source, dietary intake from foods like oily fish, egg yolks, fortified products, and supplements also contributes. After absorption, vitamin D₃ is metabolized in the liver by 25-hydroxylase, producing 25-hydroxyvitamin D [25(OH)D], which serves as the main circulating form and is commonly used to evaluate vitamin D status. This intermediate is then converted in the kidneys by 1 α -hydroxylase into its active form, 1,25-dihydroxycholecalciferol, or calcitriol. Calcitriol binds to vitamin D receptors (VDRs) found in various tissues, including bone, immune cells, and gingival structures, where it regulates gene expression related to mineral balance, immune function, and cellular differentiation [4].

A deficiency in vitamin D may arise due to limited sun exposure, poor nutrition, gastrointestinal malabsorption, chronic renal or hepatic disorders, obesity, or medications that disrupt its metabolism. When serum 25(OH)D levels fall below 20 ng/mL, deficiency is diagnosed; levels between 20–30 ng/mL are considered insufficient [7]. Low vitamin D impairs calcium absorption, leading to hypocalcemia and triggering secondary hyperparathyroidism. Elevated parathyroid hormone (PTH) levels promote bone resorption to stabilize calcium levels, which can result in reduced bone density, osteopenia, osteoporosis, and alveolar bone degradation [3]. Additionally, vitamin D deficiency weakens immune regulation, increasing vulnerability to infections and chronic inflammatory diseases, including periodontal conditions [2].

In periodontal pathology, insufficient vitamin D intensifies disease progression through several pathways. It elevates the production of inflammatory cytokines like IL-1 β , IL-6, and TNF- α , which contribute to tissue breakdown and bone loss, while suppressing anti-inflammatory mediators such as IL-10 [5]. The innate immune system is also compromised, as vitamin D deficiency reduces antimicrobial peptides like cathelicidin and β -defensins, which are vital for controlling oral pathogens [5]. This imbalance fosters a dysbiotic subgingival biofilm, accelerating periodontal tissue destruction.

Vitamin D is essential for maintaining bone homeostasis in the periodontium. It stimulates osteoblasts and inhibits osteoclasts, preserving alveolar bone structure [3]. In deficient states, this equilibrium is disrupted, leading to increased bone resorption and impaired healing. Genetic variations in the VDR gene have been linked to heightened susceptibility to periodontitis and may influence individual responses to vitamin D therapy [4].

Therapeutically, vitamin D supplementation has demonstrated benefits when combined with conventional periodontal treatments like scaling and root planing (SRP). Patients receiving adjunctive vitamin D show improved clinical attachment, reduced bleeding on probing, and better overall gingival health compared to those undergoing SRP alone [6]. It may also enhance the success of regenerative procedures and dental implants by promoting bone formation and minimizing peri-implant inflammation.

Managing vitamin D deficiency typically involves supplementation with either ergocalciferol (D₂) or cholecalciferol (D₃), with dosages adjusted based on severity and patient-specific factors. Mild cases may respond to daily doses of 800–2,000 IU, while more severe deficiencies might require 50,000 IU weekly for several weeks, followed by maintenance therapy. In individuals with renal impairment or enzymatic defects affecting activation, active forms like calcitriol or alfacalcidol may be necessary. Regular monitoring of serum 25(OH)D levels ensures effective treatment and prevents toxicity. Lifestyle changes, including improved diet and weight management, also support optimal vitamin D levels.

4. Conclusion

Vitamin D is far more than just a nutrient—it's a key player in maintaining the delicate balance between oral health and systemic wellness. From its complex metabolic journey through the skin, liver, and kidneys to its active role in regulating immune responses and bone remodeling, vitamin D influences nearly every stage of periodontal health. When levels drop, the consequences ripple through the body: inflammation intensifies, bone resorption accelerates, and the oral microbiome shifts toward disease. In the context of periodontal therapy, correcting vitamin D deficiency isn't just supportive—it can be transformative. Supplementation has shown real promise in improving clinical outcomes, reducing inflammation, and enhancing healing. With the rise of chairside testing and personalized care, we now have the tools to integrate nutritional assessment into routine dental practice. Addressing vitamin D status is no longer optional—it's an essential part of comprehensive periodontal management.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Written informed consent was obtained from the patient.

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