REVIEW ARTICLE

Periodontal Disease and Persistent Severe Vitamin D Deficiency in a Young Adult: A Concise Review with an Illustrative Case Report

Aida Meto[1,2,3,*] **and Drita Kordhoku**[1]

[1] Department of Dentistry, Faculty of Dental Sciences, University of Aldent, 1007 Tirana, Albania
[2] Department of Surgery, Medicine, Dentistry and Morphological Sciences with Interest in Transplant, Oncology and Regenerative Medicine, University of Modena and Reggio Emilia, 41125 Modena, Italy
[3] Department of Dental Research Cell, Dr. D. Y. Patil Dental College and Hospital, Dr. D.Y. Patil Vidyapeeth, Pimpri, 411018 Pune, India

Abstract

Background: Vitamin D plays an important role in bone metabolism, immune regulation, and periodontal health, although its independent contribution to periodontal disease remains uncertain. Aim: This report describes periodontal treatment outcomes in a young adult with periodontitis and persistent severe vitamin D deficiency, supported by a concise literature review. Case presentation: A 25-year-old female presented with gingival bleeding, dental hypersensitivity, tooth mobility, and radiographic alveolar bone loss. Baseline serum 25-hydroxyvitamin D [25(OH)D] was 8.3 ng/mL. Treatment included non-surgical periodontal therapy, restorative procedures, oral hygiene instruction, and medical referral for vitamin D supplementation. Results: At six months, bleeding on probing decreased from 78% to 22%, mean probing pocket depth decreased from 4.6 to 3.2 mm, and sites with probing depth $\geq$ 5 mm decreased from 18 to 5. However, serum 25(OH)D increased only to 8.9 ng/mL, indicating persistent severe deficiency. Supplementation adherence was inconsistent. Residual plaque and gingival inflammation remained evident. Incomplete clinical attachment level measurements prevented assessment of attachment gain. Conclusions: Periodontal inflammatory parameters improved following conventional dental treatment despite persistent severe vitamin D deficiency. However, the single-patient design and incomplete longitudinal measurements preclude causal conclusions regarding vitamin D status or supplementation. These findings emphasize conventional periodontal care, medical follow-up, and the need for further controlled investigations.

Keywords: alveolar bone loss, dental caries, oral health, periodontal disease, vitamin D deficiency.

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***Corresponding author:**
✉ Aida Meto
aida.meto@ual.edu.al

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1 Introduction

In daily clinical practice, periodontal disease remains an important concern in clinical dentistry, particularly among young adults who seek treatment only after experiencing pain, discomfort, or other noticeable oral symptoms. Irregular dental attendance and inadequate oral hygiene may allow inflammatory conditions to progress before professional care is received. Periodontitis is a multifactorial inflammatory disease associated with dysbiotic dental biofilms and a host-mediated response that can progressively damage the gingiva, periodontal ligament, cementum, and alveolar bone. Although dental biofilm is central to disease development, the severity and progression of periodontal destruction may also be influenced by behavioral, environmental, and systemic factors, including smoking, metabolic disorders, and nutritional status [1].

Among the nutritional factors investigated in periodontal research, vitamin D has received increasing attention because of its involvement in immune regulation, inflammatory responses, and bone metabolism. Vitamin D deficiency may result from limited sunlight exposure, inadequate dietary intake, impaired intestinal absorption, obesity, or disorders affecting hepatic and renal metabolism. Since low vitamin D levels may remain clinically unnoticed, circulating serum 25-hydroxyvitamin D [25(OH)D] is commonly used to assess vitamin D status. Marked deficiency can affect calcium–phosphate homeostasis and skeletal mineralization, raising questions about its possible relevance to alveolar bone health [2].

Vitamin D is more than a nutrient involved in calcium absorption. Following its synthesis in the skin or intake through diet, it undergoes hydroxylation in the liver to produce 25(OH)D, followed by conversion to its biologically active form, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], also known as calcitriol. Through interaction with the vitamin D receptor (VDR), calcitriol regulates biological processes in several tissues, including immune, epithelial, and bone-related cells. These functions provide a biological basis for investigating the relationship between vitamin D status and oral health [3].

Within periodontal tissues, vitamin D signaling may influence inflammatory regulation, antimicrobial defense, and alveolar bone remodeling. These processes are relevant to periodontitis, where persistent inflammation and altered bone metabolism contribute to the loss of periodontal support. Previous studies have explored associations between low serum vitamin D concentrations and periodontal inflammation, attachment loss, and alveolar bone destruction. However, the extent to which vitamin D deficiency independently influences periodontal disease progression or modifies the response to treatment remains uncertain [4, 5].

This uncertainty becomes particularly relevant when periodontal destruction is observed in young adults with concurrent nutritional deficiencies. Although systemic conditions may influence the host response, their presence does not necessarily establish a causal relationship with periodontal disease. Careful interpretation is therefore needed, especially when local risk factors and behavioral influences are also present.

The present study combines a clinical case report with a concise review of the literature to explore the potential relationship between vitamin D status and periodontal health. It describes the clinical presentation, conventional periodontal treatment, and six-month follow-up of a young adult patient with periodontitis and persistent severe vitamin D deficiency. Particular attention is given to the improvement in periodontal parameters despite the absence of biochemical correction of vitamin D deficiency. By considering these findings alongside the patient's local and behavioral risk factors and the available scientific evidence, this report presents a clinically relevant, hypothesis-generating observation that may inform future research, without attributing the periodontal findings or treatment response directly to vitamin D status.

1.1 Biological Mechanisms Linking Vitamin D to Periodontal Health

The biological relationship between vitamin D and periodontal health involves several biological pathways, particularly those associated with local vitamin D metabolism, immune regulation, antimicrobial defense, and alveolar bone remodeling [5, 6].

Vitamin D activity within periodontal tissues may extend beyond its systemic endocrine functions. Studies have shown that human gingival fibroblasts and periodontal ligament cells possess enzymes involved in vitamin D metabolism, suggesting that these tissues may participate in local vitamin D

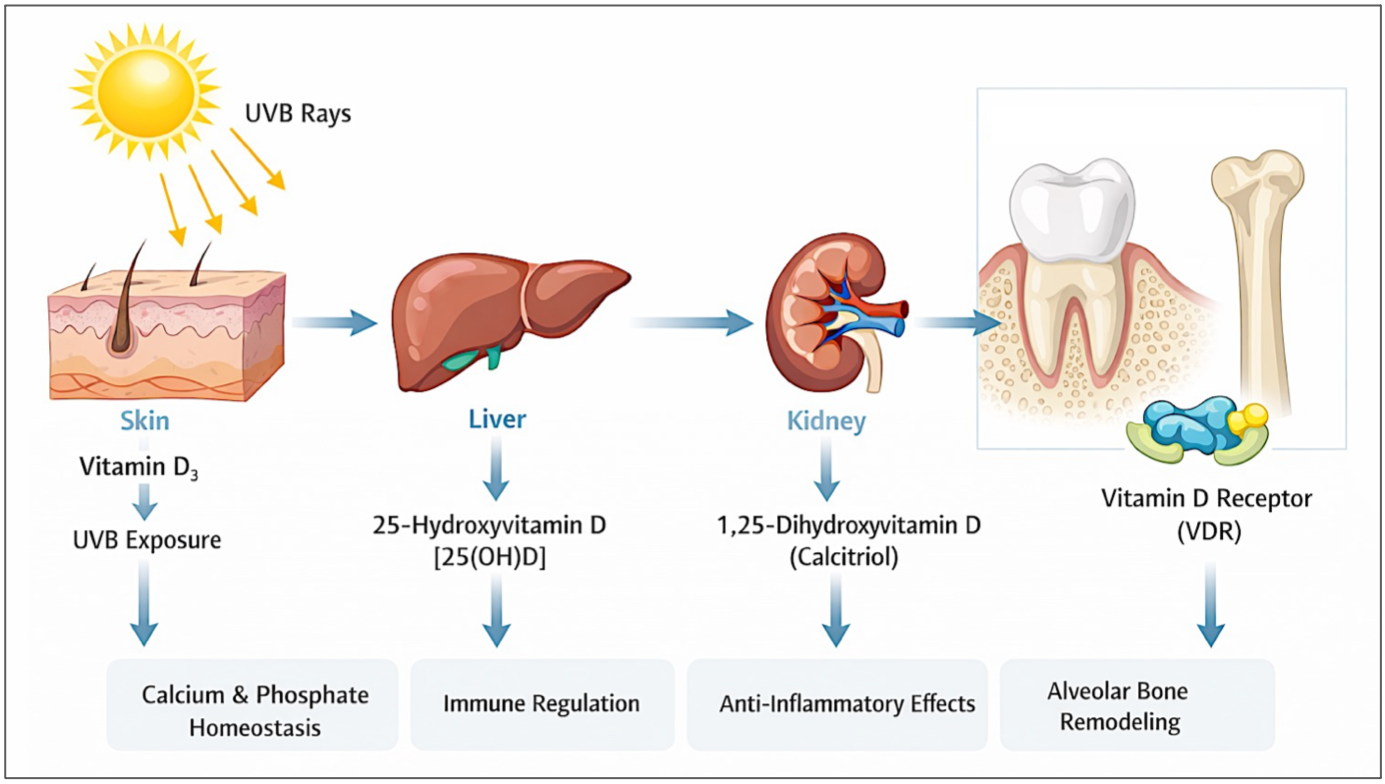


Figure 1. Vitamin D metabolism and biological activation. Cutaneous synthesis of vitamin D_3 following ultraviolet B (UVB) exposure is followed by hepatic conversion to 25(OH)D and renal activation to 1,25-dihydroxyvitamin D (calcitriol). The active metabolite binds to the vitamin D receptor (VDR) expressed in bone and periodontal tissues, regulating calcium–phosphate homeostasis, immune responses, inflammation, and alveolar bone remodeling relevant to periodontal health.

activation. This capacity may allow vitamin D to act through autocrine and paracrine mechanisms, influencing cellular activity and inflammatory regulation within the periodontal environment [6].

One of the most relevant functions of vitamin D is its role in immune regulation. Through VDR-mediated signaling, calcitriol can influence immune cell activity, modulate inflammatory mediator production, and stimulate antimicrobial peptides such as cathelicidin and defensins. These responses are important in periodontal tissues, where microbial challenges must be controlled without triggering excessive inflammation. Reduced vitamin D availability may alter aspects of this immune balance, although its direct contribution to periodontal disease in humans remains incompletely understood [7].

The host inflammatory response plays a central role in periodontal tissue destruction. Persistent activation of inflammatory pathways can increase the production of cytokines and other mediators involved in connective tissue degradation and alveolar bone resorption. This has encouraged interest in therapeutic approaches that address host-response regulation alongside conventional periodontal treatment [8]. Within this context, vitamin D has been investigated as a potential influence on periodontal inflammation, rather than as an established independent therapeutic agent [5].

Vitamin D is also involved in maintaining calcium–phosphate balance and regulating bone remodeling. Its actions influence osteoblast function, mineralization, and osteoclast activity through pathways involving receptor activator of nuclear factor kappa-B (RANK), RANK ligand (RANKL), and osteoprotegerin (OPG). These mechanisms are relevant to alveolar bone, where chronic periodontal inflammation can disturb the balance between bone formation and resorption. Altered vitamin D status may affect this regulatory environment, although the magnitude and clinical significance of its contribution remain uncertain [5, 7].

These mechanisms provide a biological rationale for investigating vitamin D as a potential modifying factor in periodontal disease (Figure 1). Nevertheless, periodontitis results from the interaction of microbial, host-related, behavioral, and systemic influences.

Vitamin D deficiency should therefore be considered within this broader context rather than as an isolated explanation for periodontal tissue destruction.

1.2 Clinical Evidence and Implications of Vitamin D Deficiency in Periodontal Care

Growing interest in the biological functions of vitamin D has encouraged research into its possible relationship with periodontal health. Epidemiological studies and systematic reviews have investigated whether circulating vitamin D concentrations are associated with the presence and severity of periodontitis. A systematic review and meta-analysis reported an association between vitamin D status and periodontal disease, suggesting that lower serum concentrations may be linked to less favorable periodontal findings. However, differences in study populations, diagnostic criteria, and methods of assessing vitamin D status make these results difficult to compare. Moreover, observational findings cannot determine whether vitamin D deficiency contributes directly to periodontal destruction or whether both conditions are influenced by shared risk factors [9].

The potential relevance of vitamin D extends beyond periodontal tissues. Its involvement in mineralization, immune regulation, and tissue homeostasis has prompted investigations into several aspects of oral health. A comprehensive review discussed possible associations between vitamin D deficiency and conditions affecting dental structures, periodontal tissues, and oral immune defense. Although these findings support the importance of considering nutritional and metabolic factors in oral health, the strength of the available evidence differs across conditions [10].

Vitamin D also plays an important role during dental development through its contribution to calcium–phosphate metabolism and mineralized tissue formation. Inadequate vitamin D status during early development has been associated with disturbances in dental mineralization, suggesting that its influence on oral health may begin in childhood, although these developmental effects concern dental hard tissues rather than periodontal support [11]. Experimental studies have also examined the effect of oral vitamin D3 on dental caries in vivo and in vitro [12]. Nevertheless, caries development is influenced by multiple dietary, microbial, behavioral, and environmental factors, and findings from individual studies do not establish vitamin D supplementation as an independent preventive or therapeutic measure.

Clinical investigations have further examined circulating vitamin D concentrations in individuals with and without periodontitis. Case-control studies have reported differences in vitamin D status between these groups, providing additional evidence of a possible association with periodontal health. However, such studies cannot establish whether reduced vitamin D concentrations precede periodontal disease or arise alongside other systemic and behavioral influences [13].

The possible long-term relevance of vitamin D status has also been explored in prospective studies investigating tooth loss. These investigations are important because tooth loss may reflect the cumulative effects of periodontal destruction and other oral conditions. However, dental caries, periodontal disease, access to care, treatment decisions, and individual patient characteristics can all influence tooth retention. Consequently, associations between serum 25(OH)D concentrations and subsequent tooth loss require careful interpretation [14].

Taken together, the available evidence suggests a possible relationship between vitamin D status and periodontal health, but important uncertainties remain regarding its clinical significance. This is particularly relevant in young adults presenting with substantial periodontal destruction alongside marked vitamin D deficiency. In such patients, nutritional and metabolic findings should be considered within a comprehensive assessment that also accounts for established local, behavioral, and systemic risk factors.

Persistent vitamin D deficiency may require medical attention even when periodontal inflammatory parameters improve following conventional treatment. Although routine vitamin D screening is not currently established as a standard component of periodontal assessment, targeted evaluation may be appropriate when independent medical indications or recognized risk factors for deficiency are present. When deficiency is identified, coordination between dental and medical professionals can support appropriate treatment, patient adherence, and biochemical monitoring (Figure 2).

The following case illustrates the coexistence of periodontitis and persistent severe vitamin D deficiency in a young adult. It highlights the improvement in periodontal parameters following conventional treatment despite the absence of

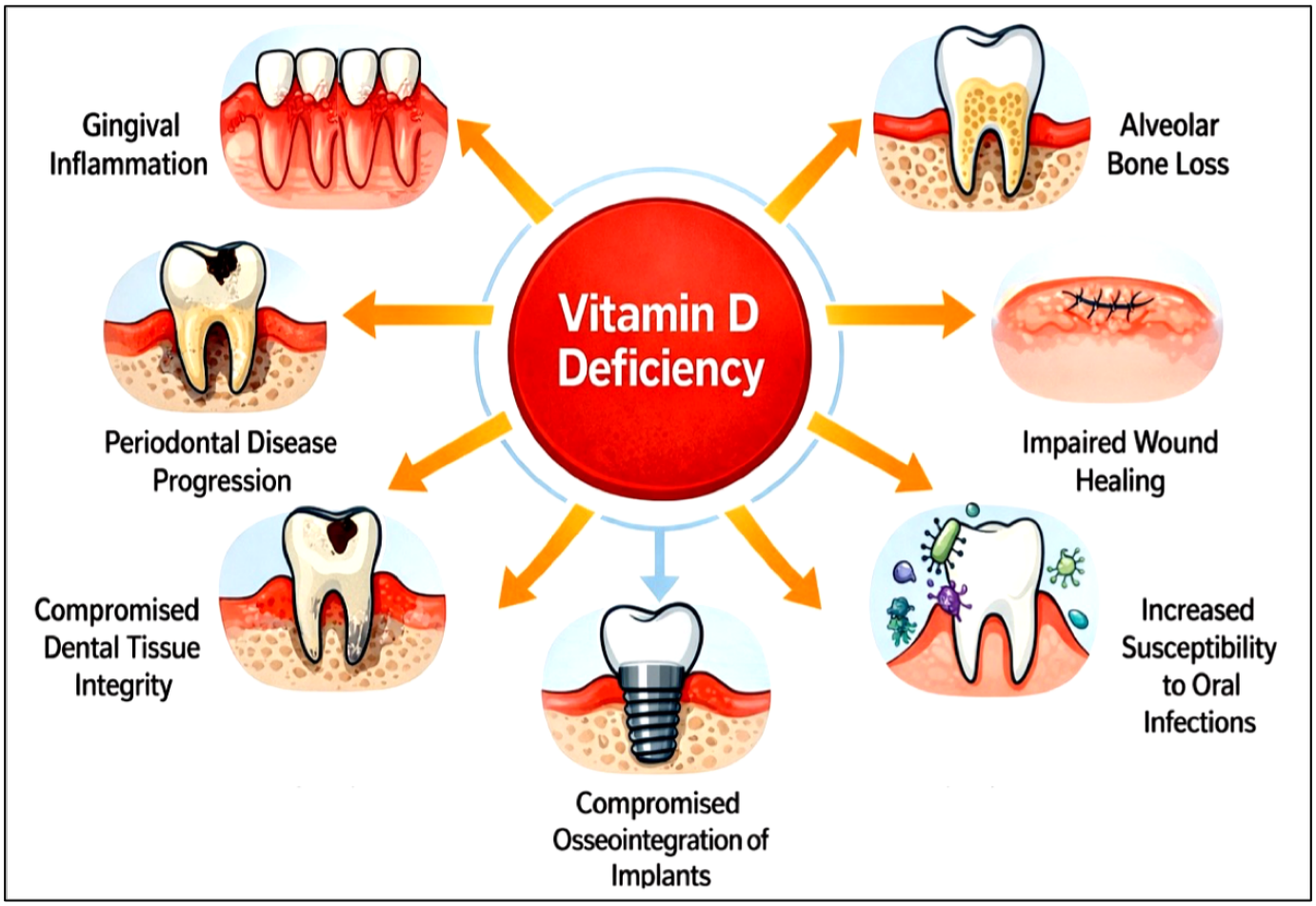


Figure 2. Relationship Between Vitamin D Deficiency and Oral Disease Manifestations. Schematic representation of the biological and clinical associations between vitamin D deficiency and oral disease manifestations. Hypovitaminosis D is linked to enhanced gingival inflammation, accelerated periodontal breakdown, and increased alveolar bone resorption. It is further associated with impaired wound healing, heightened susceptibility to oral infections, increased caries risk, compromised epithelial and connective tissue integrity, and reduced osseointegration capacity of dental implants, reflecting its role in immune modulation, bone metabolism, and tissue homeostasis.

biochemical correction, underscoring the need for cautious interpretation of the relationship between vitamin D status and periodontal outcomes.

2 Clinical Case

2.1 Case Report Design and Reporting Framework

This manuscript presents a single-patient clinical case supported by a concise review exploring the potential relationship between vitamin D deficiency and periodontal health. The case is reported in accordance with the CARE guidelines, covering patient information, clinical findings, diagnostic assessment, treatment, and follow-up.

Clinical data were obtained from dental records, periodontal examinations, radiographic images, laboratory results, and follow-up assessments. The accompanying literature review provides the scientific context for interpreting these findings.

Given the single-patient design, the observations are presented descriptively and cannot establish a causal relationship between vitamin D deficiency and periodontal destruction or determine the independent effects of vitamin D supplementation on treatment outcomes.

2.2 Patient Information and Ethics

A 25-year-old female patient presented to the Dental Clinic of Aldent University in Tirana, Albania. She reported spontaneous gingival bleeding, pain during chewing, and generalized dental hypersensitivity. These symptoms had worsened over several days and were severe enough to impair oral hygiene practices and mastication.

The recent worsening of symptoms in this young patient prompted consideration of possible systemic or metabolic contributing factors.

Written informed consent was obtained from the patient before inclusion in this report. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Aldent University (Protocol No. 4/4; 15 January 2025).

2.3 Medical, Dental, and Lifestyle History (Risk Assessment)

A detailed medical, dental, and lifestyle history was obtained to identify factors potentially relevant to the patient's periodontal condition. The patient reported no known systemic diseases, regular medications, or allergies, including no previous diagnosis of endocrine, metabolic, autoimmune, or gastrointestinal disorders that might affect nutrient absorption. However, undiagnosed systemic conditions could not be excluded based on medical history alone.

Her dental history revealed irregular attendance, with the last professional cleaning performed approximately one year before presentation. She had never received periodontal treatment or followed a regular maintenance program. These findings suggested limited professional periodontal monitoring. Gingival discomfort, dental hypersensitivity, and pain during chewing may also have affected her ability to maintain effective daily oral hygiene.

Regarding lifestyle, the patient reported limited sunlight exposure due to predominantly indoor activities and a low intake of vitamin D-rich foods, including fatty fish, fortified dairy products, and eggs. She was not taking vitamin D or multivitamin supplements. Subsequent laboratory testing revealed a serum 25(OH)D concentration of 8.3 ng/mL, indicating marked deficiency.

Additional factors relevant to periodontal risk, including tobacco exposure, glycemic status, family history, body mass index, interdental cleaning habits, and parafunctional activity, were not sufficiently documented for retrospective assessment. These limitations were considered when interpreting the patient's clinical presentation and the potential contribution of systemic factors.

2.4 Local and Behavioral Risk Factors

Clinical examination revealed visible plaque accumulation, particularly in the anterior regions, along with multiple active carious lesions. Irregular dental attendance and the absence of previous periodontal treatment suggested insufficient professional plaque control and periodontal monitoring. In addition, dental hypersensitivity and discomfort during chewing may have made daily oral hygiene more difficult.

These findings highlight the relevance of local biofilm accumulation and behavioral factors to the patient's periodontal condition. Although marked vitamin D deficiency was also identified, its independent contribution to periodontal destruction could not be determined. The clinical presentation was therefore interpreted within a multifactorial context, with particular consideration given to the documented local and behavioral risk factors.

2.5 Clinical findings

Intraoral examination revealed generalized gingival erythema and marginal swelling in both arches, with bleeding on probing at multiple sites (Figures 3 and 4). Visible plaque accumulation and several active carious lesions, particularly in the anterior region, were also observed. The patient reported dental hypersensitivity and discomfort during mastication.



Figure 3. Frontal intraoral view showing generalized gingival erythema and marginal inflammation, with visible plaque accumulation and carious lesions affecting the anterior teeth.

At baseline, the mean probing pocket depth (PPD) was 4.6 mm, with 18 sites measuring ≥ 5 mm. Bleeding on probing (BOP) was recorded at 78% of the examined sites, and four teeth exhibited Grade II–III mobility. Clinical attachment loss (CAL) was documented, although complete site-specific measurements and a mean CAL value were unavailable for retrospective analysis.

Panoramic radiography revealed horizontal alveolar bone loss across several regions, with localized areas



Figure 4. Intraoral photographs documenting periodontal probing of the mandibular anterior teeth, with immediate bleeding on probing, indicative of active gingival inflammation and periodontal disease.

suggestive of vertical defects (Figure 5). Together with the clinical findings, these observations supported the diagnosis of periodontitis.



Figure 5. Panoramic radiograph demonstrating generalized horizontal alveolar bone loss with localized vertical defects, findings disproportionate to the patient's age and dental history.

However, definitive staging and grading according to the current periodontal classification could not be established because complete interdental CAL measurements, radiographic bone-loss percentages relative to root length, tooth-loss history attributable to periodontitis, and relevant progression factors were not sufficiently documented.

2.6 Periodontal Examination and Measurement Protocol

Periodontal examinations were performed at baseline and at the six-month follow-up by the same clinician (D.K.), using a graduated Williams periodontal probe and a consistent clinical assessment protocol. Probing was carried out at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual). The examination included PPD, CAL, BOP, and tooth mobility, together with radiographic assessment of alveolar bone loss. PPD was measured from the gingival margin to the base of the periodontal pocket, while CAL was determined from the cementoenamel junction to the same point. BOP was recorded as the percentage of examined sites showing bleeding following probing.

The clinical records provided baseline and follow-up data on mean PPD, the number of sites with PPD ≥ 5 mm, BOP percentage, and tooth mobility. Panoramic radiographs were also reviewed to assess the overall distribution and visible pattern of alveolar bone loss. These radiographic findings were interpreted alongside the clinical examination to support the evaluation of the patient's periodontal condition.

As this case was managed in a university dental clinic, maintaining regular recall appointments was an important consideration during follow-up. Although the same examination protocol was used at both visits, the retrospective records were not equally complete for all periodontal parameters. In particular, the absence of detailed site-specific CAL measurements prevented a reliable comparison of mean attachment levels over time. Numerical plaque scores were also unavailable, so plaque deposits visible in the clinical photographs were described qualitatively rather than assigned estimated values.

Furthermore, the available documentation did not establish whether formal examiner calibration had been performed or provide a complete record of the total number of teeth and periodontal sites included in the summary measurements. Together with the limitations of panoramic radiography, these gaps prevented a more detailed periodontal classification

and restricted the interpretation of changes between baseline and follow-up. Therefore, the findings presented in this report were limited to measurements that could be verified from the original clinical records, allowing the patient's periodontal condition to be described without overstating the available evidence.

3 Diagnostic Approach and Clinical Interpretation

3.1 Periodontal Diagnosis and Classification

The periodontal diagnosis was assessed according to the 2017 World Workshop classification of periodontal and peri-implant diseases and conditions, which considers disease severity, treatment complexity, progression, and extent of periodontal involvement [15].

At baseline, the patient presented with clinical and radiographic findings consistent with periodontitis. The examination revealed a mean PPD of 4.6 mm, with 18 sites measuring $\geq$ 5 mm. BOP was recorded at 78% of the examined sites, reflecting widespread gingival inflammation. Four teeth showed Grade II–III mobility, indicating reduced periodontal support. Clinical photographs also revealed visible plaque accumulation, gingival erythema, marginal inflammation in both dental arches, and multiple carious lesions, particularly in the anterior region.

Panoramic radiographs showed alveolar bone loss affecting several regions of the dentition, predominantly following a horizontal pattern, with localized areas suggestive of vertical defects. Together with the clinical findings of attachment loss and increased probing depths, these observations supported the diagnosis of periodontitis.

Under the current classification system, periodontitis is characterized by stage, grade, and extent. Staging reflects the severity of periodontal destruction and the complexity of treatment, while grading considers the available evidence of disease progression and relevant risk factors. Although the findings in this patient indicated established periodontal tissue destruction, the retrospective documentation did not contain sufficient information to assign a reliable stage or grade. In particular, complete site-specific CAL measurements, standardized radiographic bone-loss percentages, and information on tooth loss attributable to periodontitis were unavailable. The proportion of affected teeth could not be determined with confidence.

The condition was therefore described as periodontitis involving multiple regions of the dentition, without assigning a definitive stage, grade, or extent. This diagnostic approach reflects the available clinical evidence while avoiding classifications that could not be adequately supported by the original records. The baseline periodontal, oral health, and biochemical findings are summarized in Table 1.

3.2 Clinical Interpretation and Contributing Factors

The presence of periodontal tissue destruction at a relatively young age prompted further consideration of factors that might influence the patient's oral health. Visible plaque accumulation, untreated carious lesions, irregular dental attendance, and the absence of previous periodontal therapy were identified as relevant local and behavioral factors. These circumstances may have contributed to persistent gingival inflammation and delayed periodontal care.

The patient's medical and lifestyle history also revealed limited sunlight exposure and a low reported intake of vitamin D-rich foods, providing a rationale for evaluating serum vitamin D status. Laboratory testing showed a baseline serum 25(OH)D concentration of 8.3 ng/mL.

For consistency, vitamin D status was interpreted using the thresholds established in the 2011 Endocrine Society clinical practice guideline, which defines deficiency as serum 25(OH)D below 20 ng/mL, insufficiency as 21–29 ng/mL, and sufficiency as $\geq$ 30 ng/mL [16]. This guideline does not define a separate category of severe deficiency; in this report, the term "severe" is used descriptively for serum 25(OH)D below 10 ng/mL. Serum 25(OH)D concentrations remained markedly low at both baseline (8.3 ng/mL) and six months (8.9 ng/mL), despite the prescription of vitamin D supplementation.

Vitamin D plays an important role in bone metabolism and immune regulation, and its potential relationship with periodontal health has attracted increasing scientific interest. Nevertheless, the findings of this single case cannot establish whether vitamin D deficiency contributed to the development or severity of periodontal destruction. The periodontal diagnosis was therefore based on the clinical and radiographic evidence, independently of the patient's biochemical status.

From a clinical perspective, the coexistence of periodontitis and marked vitamin D deficiency highlighted the importance of addressing both oral

Table 1. Baseline Periodontal and Oral Health Assessment.

Clinical parameter	Baseline findings
Patient age	25 years
Periodontal condition	Clinical findings consistent with periodontitis
Gingival condition	Gingival erythema and marginal inflammation involving both arches
Mean probing pocket depth (PPD)	4.6 mm
Sites with PPD $\geq$ 5 mm	18 sites
Bleeding on probing (BOP)	78%
Clinical attachment loss (CAL)	Reported clinically; complete site-specific measurements unavailable
Tooth mobility	Four teeth presenting Grade II–III mobility
Plaque accumulation	Visible in clinical photographs; quantitative plaque score unavailable
Gingival recession	Complete site-specific measurements unavailable
Furcation involvement	Not documented in the available records
Suppuration	Not documented in the available records
Radiographic bone loss	Alveolar bone loss involving multiple regions, predominantly horizontal
Vertical bone defects	Localized areas suggestive of vertical defects
Radiographic bone-loss percentage	Not established from standardized measurements
Dental caries	Multiple active carious lesions, particularly in the anterior region
Missing teeth	Number and causes of missing teeth not established from the available records
Periodontal stage	Not definitively established
Periodontal grade	Not definitively established
Extent and distribution	Multiple regions involved; precise percentage of affected teeth unavailable
Serum 25(OH)D	8.3 ng/mL

and general health needs. Treatment planning focused on conventional periodontal therapy, plaque control, management of carious lesions, and medical referral for evaluation and treatment of vitamin D deficiency. The persistently low serum 25(OH)D concentration at follow-up was interpreted as a lack of documented biochemical improvement rather than successful correction of the deficiency.

Overall, this case illustrates the importance of considering local, behavioral, and systemic findings together when evaluating periodontal health, while recognizing that their coexistence does not necessarily establish a causal relationship.

3.3 Systemic and Metabolic Assessment

The patient's medical history and lifestyle were reviewed to identify possible systemic or nutritional factors that could influence her periodontal health. She reported no known endocrine, metabolic, autoimmune, or gastrointestinal disorders and was not taking medications known to affect bone metabolism or immune function. Nevertheless, undiagnosed systemic conditions could not be completely excluded.

Given her young age and the severity of periodontal destruction, further assessment of her metabolic and nutritional status was considered important. The markedly low serum 25-hydroxyvitamin D [25(OH)D] level detected at baseline raised additional clinical concerns. However, vitamin D deficiency was regarded as a concurrent systemic finding, as its direct contribution to periodontal destruction could not be established.

Additional laboratory investigations, including calcium, phosphate, parathyroid hormone, alkaline phosphatase, renal and liver function, and blood glucose, could help identify potential metabolic abnormalities. A complete blood count and further nutritional assessment might also be useful, depending on the patient's clinical findings.

During the six-month follow-up, serum 25(OH)D was the only biochemical parameter reassessed, remaining severely deficient. No other laboratory results were available to clarify whether metabolic disturbances, nutritional deficiencies, or impaired vitamin D absorption contributed to this persistent deficiency.

The patient was therefore referred for medical evaluation and appropriate management of vitamin D deficiency. Continued collaboration between dental and medical professionals was recommended to monitor her systemic health and guide further investigations when necessary. This approach ensured that the deficiency received appropriate clinical attention while avoiding unsupported conclusions about its role in periodontal disease.

4 Treatment Plan and Implementation

An individualized treatment plan was developed to address the patient's periodontal condition, active carious lesions, and associated oral symptoms, alongside the management of her vitamin D deficiency (Table 2). Dental care focused on controlling plaque-related inflammation through non-surgical periodontal therapy, improving daily oral hygiene, and restoring affected teeth. At the same time, medical referral was arranged to address the identified systemic deficiency. Treatment was delivered over several appointments, with periodontal reassessment and supportive care incorporated into the six-month follow-up period. Dental outcomes and vitamin D status were evaluated separately to allow a clearer interpretation of the patient's clinical and biochemical responses.

4.1 Systemic Management: Vitamin D Supplementation and Medical Follow-Up

Following the detection of severe vitamin D deficiency, the patient was referred to her family physician for medical evaluation and treatment. Oral cholecalciferol (vitamin D_3) was prescribed at a dose of 50,000 IU once weekly for eight weeks, followed by a recommended maintenance dose of 2,000 IU daily. Regular medical follow-up and laboratory monitoring were advised to evaluate her response to supplementation.

However, during the six-month follow-up, the patient reported irregular adherence to the prescribed treatment. Serum 25(OH)D was reassessed only at the six-month visit, showing a minimal increase from 8.3 ng/mL at baseline to 8.9 ng/mL. These findings confirmed that severe vitamin D deficiency persisted. No adverse effects related to supplementation were reported, although the lack of intermediate laboratory assessments limited the evaluation of treatment response and safety.

Apart from the prescribed vitamin D supplementation, the available records did not indicate the use of additional nutritional supplements, such as calcium or magnesium, during this period. Information about other medications potentially affecting vitamin D absorption or metabolism was also incomplete, preventing further assessment of their possible influence on the patient's vitamin D status.

The persistent deficiency, together with the patient's inconsistent supplementation, made it difficult to evaluate the effectiveness of the prescribed regimen. Continued medical supervision was therefore recommended to reassess her vitamin D status, address treatment adherence, and determine whether further investigations or adjustments were needed.

4.2 Periodontal Therapy: Control of Infection and Inflammation

Non-surgical periodontal treatment was initiated to reduce bacterial deposits, control gingival inflammation, and improve periodontal health. Treatment involved supragingival and subgingival instrumentation, including scaling and root planing with ultrasonic and manual instruments.

The active phase was completed over two appointments. The first session, performed one week after the baseline examination, involved the first and second quadrants, while the remaining quadrants were treated during the second session, two weeks after baseline. Local anesthesia was administered when necessary to improve comfort, particularly in areas with deeper periodontal pockets or increased sensitivity.

Oral hygiene instructions were provided throughout treatment, with particular emphasis on effective daily plaque removal. As a short-term adjunct to mechanical cleaning, the patient was instructed to rinse with 0.12% chlorhexidine twice daily for 14 days, as documented in the clinical records. The early response to treatment was assessed at six weeks, followed by supportive periodontal care at three months and a final clinical evaluation at six months. These visits focused on monitoring gingival inflammation, bleeding on probing, periodontal pocket depths, and the patient's ability to maintain oral hygiene.

4.3 Assessment and Management of Tooth Mobility

At the initial examination, four teeth presented Grade II–III mobility, reflecting reduced periodontal support. The initial management focused on controlling inflammation and removing local bacterial deposits before evaluating the need for further intervention.

Tooth mobility was monitored during follow-up, and the six-month records indicated an improvement, with only one tooth presenting Grade I mobility. However, the documentation did not identify the individual teeth involved or provide complete tooth-specific mobility measurements at both examinations. It also did not establish whether additional procedures, such as occlusal adjustment or periodontal splinting, had been performed.

The improvement was therefore reported as an

Table 2. Chronological Summary of Periodontal Treatment and Follow-Up.

Treatment phase	Time	Clinical procedures and assessment
Initial examination	Week 0	Medical and dental history, periodontal examination, radiographic assessment, and baseline clinical documentation
First periodontal treatment session	Week 1	Supragingival and subgingival instrumentation of the first and second quadrants using manual and ultrasonic instruments
Second periodontal treatment session	Week 2	Completion of periodontal instrumentation in the remaining quadrants
Early periodontal reassessment	Week 6	Clinical reassessment of periodontal inflammation and response to non-surgical therapy
Supportive periodontal care	Month 3	Periodontal maintenance and reinforcement of oral hygiene instructions
Final clinical reassessment	Month 6	Periodontal examination, assessment of treatment response, and follow-up evaluation
Vitamin D assessment	Baseline	Serum 25(OH)D: 8.3 ng/mL
Vitamin D supplementation	During the observation period	Cholecalciferol prescribed by the family physician; patient-reported adherence was inconsistent
Vitamin D reassessment	Month 6	Serum 25(OH)D: 8.9 ng/mL; severe deficiency persisted

observed clinical finding following periodontal care, without assigning it to a particular intervention or assuming that lost periodontal support had been restored.

4.4 Caries Management and Functional Restoration

Restorative treatment was included in the overall care plan to address the active carious lesions identified at baseline, particularly in the anterior region. According to the clinical records, restorations were performed on teeth 1.1, 1.2, 1.3, 2.1, 2.2, 3.4, and 3.5, using the Fédération Dentaire Internationale (FDI) numbering system. These procedures were carried out alongside periodontal therapy to improve oral health, reduce dental discomfort, and support normal function. Although endodontic treatment had initially been considered, no endodontic procedures were performed during the documented treatment period.

At the six-month follow-up, the patient reported reduced dental hypersensitivity and greater comfort during mastication. These patient-reported improvements were considered alongside the clinical findings when evaluating the overall response to treatment.

4.5 Patient Education and Behavioral Support

Patient education was an essential part of periodontal treatment, particularly because of the patient's previous irregular dental visits and limited experience with professional periodontal care. During the initial appointments, personalized instructions focused on effective toothbrushing and interdental cleaning, with particular attention to areas of increased plaque accumulation and gingival inflammation, especially in the anterior region.

At follow-up visits, the patient reported greater comfort during toothbrushing and increased attention to her daily oral hygiene. She also described following the recommended cleaning practices more regularly than before treatment. These self-reported improvements suggested greater awareness of her oral health and a positive change in daily care habits. However, as adherence was not evaluated using standardized questionnaires or daily hygiene records, the consistency of these practices could not be objectively confirmed.

The importance of regular periodontal maintenance was also explained to help reduce the risk of recurrent inflammation and support long-term periodontal stability. In addition, the patient received guidance on dietary sources of vitamin D, adherence to the supplementation prescribed by her family physician, and the importance of periodic medical evaluation and laboratory monitoring.

Despite these recommendations, the patient continued to experience difficulties maintaining regular vitamin D supplementation. This emphasized the need for ongoing patient support and close collaboration between dental and medical professionals to encourage treatment adherence and promote both periodontal and general health.

4.6 Supportive Periodontal Care and Follow-Up

Following non-surgical periodontal therapy, the patient underwent clinical reassessment at six weeks, supportive periodontal care at three months, and a final evaluation at six months. These visits focused on monitoring periodontal healing, reinforcing oral hygiene instructions, and identifying areas requiring further attention.

At the six-month examination, reductions in bleeding on probing and mean probing pocket depth indicated a favorable clinical response to periodontal treatment. However, residual plaque deposits and gingival inflammation were still observed, suggesting that plaque control remained incomplete. Oral hygiene guidance was reinforced, particularly in areas with persistent plaque accumulation. Although the patient reported improvements in her daily oral hygiene practices, the absence of objective adherence records made it difficult to assess their consistency.

Continued supportive periodontal care was recommended to preserve the clinical improvements, manage residual inflammation, and reduce the risk of disease progression. However, follow-up was documented only through six months, and no further maintenance visits or long-term periodontal outcomes were available.

5 Outcomes and Follow-Up

5.1 Clinical Periodontal Outcomes

At the six-month follow-up, the patient showed improvement in several recorded periodontal parameters following non-surgical periodontal therapy, restorative treatment, and reinforcement of oral hygiene practices. BOP decreased from 78% at baseline to 22%, while mean PPD decreased from 4.6 mm to 3.2 mm. The number of sites with PPD ≥ 5 mm was also reduced from 18 to 5, indicating fewer sites with increased probing depths.

Tooth mobility showed a favorable change in the available records. Initially, four teeth presented Grade II–III mobility, whereas only one tooth was recorded with Grade I mobility at the six-month examination. However, because paired tooth-specific measurements were unavailable, the extent of improvement in individual teeth could not be established.

The reduction in BOP was consistent with decreased gingival inflammation, while the lower PPD values suggested an improvement in the recorded periodontal condition. Nevertheless, these changes should not be interpreted as evidence of clinical attachment gain or periodontal regeneration, since complete site-specific CAL and gingival recession measurements were unavailable. Changes in gingival inflammation and gingival margin position may also influence probing depth measurements.

Clinical photographs taken at follow-up showed reduced gingival redness compared with baseline, although residual plaque deposits, localized calculus accumulation, and gingival inflammation remained visible, particularly in the anterior regions (Figure 6). As quantitative plaque scores were not documented, changes in plaque accumulation could only be assessed visually.

These findings indicate a favorable but incomplete clinical response to conventional periodontal treatment. Continued oral hygiene reinforcement and supportive periodontal care remained necessary to address residual inflammation and maintain the improvements observed during the six months.

5.2 Radiographic Findings

Panoramic radiographs obtained at baseline and at the six-month follow-up were reviewed to assess the general appearance of the dentition and alveolar bone support. The follow-up image continued to show alveolar bone loss affecting multiple regions of the dentition, consistent with the periodontal destruction identified at the initial examination (Figure 7).

However, the radiographs were not obtained using a standardized longitudinal imaging protocol. Differences in patient positioning, projection, and image geometry may influence the apparent height and contour of the alveolar bone, limiting the reliability of direct comparisons between examinations.

For this reason, the available images could not establish whether subtle changes in alveolar bone levels or new periodontal defects had occurred during the observation period. The radiographic findings were therefore interpreted descriptively rather than as definitive evidence of bone regeneration or stabilization. More accurate longitudinal assessment would require standardized site-specific radiographs together with complete periodontal charting.

5.3 Patient-Reported Outcomes

At the six-month follow-up, the patient reported reduced dental hypersensitivity, less discomfort during mastication, and greater comfort when brushing her teeth. These changes suggested an

Table 3. Periodontal findings at baseline and six-month follow-up.

Parameter	Baseline	Six-month follow-up	Descriptive change
BOP	78%	22%	Decrease of 56 percentage points
Mean PPD	4.6 mm	3.2 mm	Decrease of 1.4 mm
Sites with PPD ≥5 mm	18	5	Decrease of 13 recorded sites
Tooth mobility	Four teeth with Grade II-III mobility	One tooth with Grade I mobility	Fewer teeth recorded with mobility; tooth-specific paired data unavailable
Mean CAL	Not available	Not available	Cannot be determined
Quantitative plaque score	Not available	Not available	Cannot be determined
Gingival recession	Complete site-specific measurements unavailable	Complete site-specific measurements unavailable	Cannot be determined

***Note:** Values represent descriptive findings from a single patient. The total number of examined teeth and periodontal sites, as well as the precise method used to calculate mean PPD, could not be verified from the retrospective records. Consequently, the mean PPD and the number of sites with PPD ≥ 5 mm are reported as summary values taken from the records and could not be cross-checked against each other. Changes in probing depth should not be interpreted as clinical attachment gain in the absence of paired CAL and gingival recession measurements. No inferential statistical analysis was performed.

improvement in her daily oral comfort following periodontal treatment. However, although toothbrushing had become easier, no detailed information was available regarding brushing frequency, interdental cleaning, or consistent adherence to the recommended oral hygiene practices.

As no standardized patient-reported outcome questionnaire or oral health-related quality-of-life assessment was used, these improvements were considered subjective observations rather than objectively measured changes in quality of life or oral hygiene behavior. A comparison of baseline and follow-up findings is presented in Table 3.

5.4 Vitamin D Reassessment and Clinical Interpretation

At the six-month follow-up, serum 25(OH)D showed only a minimal increase, from 8.3 ng/mL at baseline to 8.9 ng/mL, confirming that severe vitamin D deficiency persisted throughout the observation period.

The patient reported irregular adherence to the prescribed supplementation, which may have contributed to the limited biochemical response. However, the lack of intermediate laboratory measurements, comprehensive metabolic testing, and complete information on other medications or supplements prevented a clear explanation for the persistent deficiency. Therefore, other possible contributing factors could not be excluded.

The absence of additional metabolic testing and complete information on other medications or supplements also limited the interpretation of the patient's vitamin D response. Although inconsistent supplementation was reported, other possible factors affecting vitamin D status could not be excluded. Therefore, the persistent deficiency should not be attributed to poor treatment adherence alone.

The observed improvements were considered in relation to the periodontal treatment and supportive care provided, although the individual contribution of each intervention could not be determined. Further medical evaluation was recommended to investigate the persistent deficiency and guide appropriate management.

This case documents an improvement in periodontal inflammatory parameters without a corresponding improvement in vitamin D status. Given the single-patient design and limitations of the available clinical records, these findings should be interpreted cautiously and cannot support conclusions about a causal relationship between vitamin D deficiency and periodontal treatment outcomes.

6 Discussion

The present case describes a young adult patient with periodontitis and marked vitamin D deficiency, highlighting the importance of considering both local and systemic factors during periodontal assessment. The most relevant observation was the difference between the clinical and biochemical responses over



Figure 6. Six-month clinical follow-up after conventional periodontal treatment. The photographs show improvement in gingival appearance compared with baseline, together with residual plaque and calculus deposits and persistent gingival inflammation. The images illustrate partial inflammatory control rather than complete periodontal stabilization.



Figure 7. Panoramic radiograph obtained at the six-month follow-up. The image provides an overview of the dentition and alveolar bone support at follow-up. Because the baseline and follow-up panoramic radiographs were not obtained using a standardized longitudinal protocol, their comparison cannot reliably establish changes in alveolar bone height, the absence of new periodontal defects, or definitive stabilization of periodontal support.

six months. Periodontal inflammatory parameters improved following conventional dental treatment, while serum 25(OH)D remained markedly reduced. This finding raises an important clinical question regarding the relationship between vitamin D status and periodontal health, although a causal association cannot be established from a single case.

Periodontitis develops through a complex interaction between the dental biofilm and the host inflammatory response [8]. While bacterial accumulation initiates the inflammatory process, individual susceptibility and the extent of periodontal destruction may be influenced by several behavioral, immune, and systemic factors [1]. Vitamin D has attracted particular interest because of its involvement in bone metabolism, immune regulation, and antimicrobial defense [5, 7]. These biological functions provide a plausible basis for investigating its relationship with periodontal disease, without necessarily establishing vitamin D deficiency as an independent cause of periodontal destruction [5, 9].

Vitamin D contributes to calcium and phosphate homeostasis and supports normal bone mineralization and remodeling [5, 7]. These processes are relevant to alveolar bone, where the balance between bone formation and resorption is affected by periodontal inflammation. Alterations in vitamin D status may influence this balance, although the clinical consequences depend on several interacting factors. Observational studies have reported associations between lower serum 25(OH)D concentrations and

unfavorable periodontal findings, including greater attachment loss, increased probing depths, and tooth loss [5, 9, 13, 14]. However, these associations do not establish whether vitamin D deficiency precedes periodontal deterioration or contributes directly to disease progression, particularly because periodontal disease and low vitamin D status may share common risk factors [1].

Beyond its skeletal functions, vitamin D participates in the regulation of innate and adaptive immune responses. Its active metabolite, calcitriol, acts through vitamin D receptors expressed in several immune and periodontal cell types. Experimental evidence suggests that vitamin D signaling can influence the production of inflammatory mediators, including interleukin-1 β, interleukin-6, and tumor necrosis factor- α, while supporting antimicrobial responses through peptides such as cathelicidin and defensins [5, 7]. Human gingival fibroblasts and periodontal ligament cells also express enzymes involved in vitamin D metabolism, which may allow local activation within periodontal tissues [6]. These mechanisms are relevant to periodontal tissue homeostasis because excessive inflammatory activity contributes to connective tissue breakdown and alveolar bone resorption, which has encouraged interest in host-modulation strategies [8]. Nevertheless, the extent to which these biological effects translate into measurable clinical benefits remains uncertain [5, 7].

Clinical research has also explored whether vitamin D supplementation, alone or in combination with calcium, may influence periodontal outcomes. Cross-sectional investigations have examined associations between supplementation status and periodontal parameters, while longer-term studies have evaluated changes in periodontal health among individuals receiving vitamin D and calcium supplementation [17, 18]. Although these findings provide a rationale for further investigation, differences in study design, participant characteristics, baseline nutritional status, and treatment protocols limit the conclusions that can be drawn. In particular, observational associations should not be interpreted as evidence that supplementation independently improves periodontal treatment outcomes.

The broader role of nutrition in periodontal health has also been discussed in relation to vitamins C and D. Both nutrients participate in biological processes relevant to tissue maintenance, inflammatory regulation, and repair, although their mechanisms differ. Available reviews suggest that nutritional assessment may complement periodontal management, particularly when deficiencies are suspected or confirmed. However, nutritional intervention should remain supportive of, rather than a replacement for, conventional periodontal treatment and effective daily plaque control [19].

In the present patient, several established local and behavioral factors were documented, including visible plaque accumulation, active carious lesions, irregular dental attendance, and the absence of previous periodontal therapy. These findings provide a clinically relevant context for the periodontal inflammation and tissue destruction observed at baseline. Although the patient also presented with severe vitamin D deficiency, the available evidence does not allow its independent contribution to be distinguished from that of the documented local factors. Furthermore, incomplete information regarding smoking status, glycemic control, family history, body mass index, and other metabolic parameters limited a more comprehensive assessment of the patient's periodontal risk profile.

Following non-surgical periodontal therapy, oral hygiene instruction, restorative treatment, and professional follow-up, the recorded periodontal parameters showed a favorable response. Bleeding on probing and mean probing pocket depth decreased, and fewer sites presented increased probing depths. Nevertheless, clinical photographs continued to show residual plaque and calculus deposits and localized gingival inflammation. The six-month outcome is therefore best interpreted as partial inflammatory control rather than complete periodontal stabilization. Similarly, reductions in probing depth cannot be taken as evidence of clinical attachment gain or periodontal regeneration without corresponding site-specific measurements.

The radiographic findings require comparable caution. Although panoramic radiographs were available at baseline and follow-up, they had not been obtained using a standardized longitudinal imaging protocol. Differences in positioning and projection may influence the apparent alveolar bone levels, making subtle changes difficult to assess reliably. Consequently, these images could not confirm the absence of further bone destruction or demonstrate definitive stabilization of periodontal support.

An important aspect of this case was the persistence of vitamin D deficiency despite the prescribed

supplementation. Serum 25(OH)D increased only from 8.3 ng/mL at baseline to 8.9 ng/mL at six months, while the patient reported inconsistent adherence to treatment. The periodontal improvement therefore occurred without documented biochemical correction of vitamin D status. Although conventional periodontal care may have contributed substantially to the observed clinical response, the individual effects of the interventions cannot be separated. Likewise, the findings do not demonstrate an additional periodontal benefit from vitamin D supplementation.

The limited biochemical response also emphasizes the importance of appropriate medical follow-up. Cholecalciferol is commonly prescribed to treat vitamin D deficiency, while calcifediol, its 25-hydroxylated metabolite, has been investigated as an alternative formulation because of differences in its pharmacokinetic characteristics and its ability to increase circulating 25(OH)D concentrations [20–23]. These differences may be relevant in selected patients, particularly when persistent deficiency requires further medical assessment. However, calcifediol was not administered in the present case, and no conclusions regarding its potential benefit can be drawn from the patient's outcome. Decisions about supplementation should be guided by medical indications, treatment adherence, possible underlying metabolic conditions, and appropriate biochemical monitoring rather than periodontal findings alone.

Several limitations should be considered when interpreting this case. Although probing pocket depth, bleeding on probing, and tooth mobility were recorded at baseline and follow-up, complete paired site-specific clinical attachment level measurements, gingival recession data, and quantitative plaque scores were unavailable. These gaps limited the evaluation of changes in periodontal attachment and prevented a reliable determination of disease stage, grade, and extent. The absence of standardized radiographic comparisons also restricted the assessment of changes in alveolar bone support.

The systemic evaluation was also incomplete. Apart from serum 25(OH)D, no additional laboratory results were available during follow-up to investigate possible metabolic or nutritional abnormalities. Information on other medications and supplements, including calcium and magnesium, was limited, making it difficult to assess their potential influence on vitamin D status.

The patient's irregular adherence to vitamin D supplementation and the lack of intermediate biochemical monitoring further limited the interpretation of the persistent deficiency and its possible relationship with periodontal outcomes.

In addition, adherence to daily oral hygiene recommendations was not evaluated using standardized methods. Although the patient reported greater comfort during toothbrushing and showed improvements in periodontal inflammatory parameters, these observations could not confirm consistent home care. Residual plaque deposits at six months also indicated that plaque control remained incomplete.

Finally, as this report describes a single patient who received multiple dental interventions, the contribution of individual treatments could not be determined. The findings should therefore be interpreted as clinical observations rather than evidence of a causal relationship between vitamin D status and periodontal treatment outcomes.

Future studies should investigate whether vitamin D deficiency influences periodontal progression or modifies the response to conventional treatment, particularly in patients with documented nutritional deficiencies. Well-designed longitudinal studies and randomized controlled trials would help clarify whether correction of vitamin D deficiency offers additional periodontal benefits beyond established therapy [5, 9, 17, 18]. Further research may also examine appropriate indications for vitamin D testing, supplementation strategies, and the clinical relevance of different formulations [20–23]. Integrating biochemical measurements with periodontal outcomes, immune markers, and microbiological findings could provide a more complete understanding of the potential interactions between systemic nutritional status and periodontal inflammation [8].

6.1 Clinical Recommendations and Practice Integration

From a clinical perspective, this case emphasizes the value of a comprehensive periodontal assessment that considers oral hygiene, behavioral factors, medical history, and relevant systemic findings. Vitamin D testing may be considered when there are independent clinical indications or risk factors for deficiency, particularly in patients reporting limited sunlight exposure, inadequate dietary intake, or other circumstances suggesting altered vitamin D status. However, the available evidence does not support

routine vitamin D screening solely on the basis of a periodontal diagnosis.

When deficiency is identified, collaboration with the patient's physician is important to investigate possible contributing factors, provide appropriate treatment, and monitor the biochemical response. Patient education should also address the importance of adherence to prescribed medical treatment, adequate nutrition, daily plaque control, and regular periodontal maintenance.

Proposal for Vitamin D Assessment in Patient Record

1. Serum 25(OH)D test conducted?

☐ Yes ☐ No

Result: _____ ng/mL Date: _____

2. Vitamin D status

Vitamin D status	Serum 25(OH)D concentration
Deficiency	<20 ng/mL
Insufficiency	20 to <30 ng/mL
Sufficiency	≥30 ng/mL

☐ Deficiency (<20 ng/mL) ☐ Insufficiency (20 to <30 ng/mL) ☐ Sufficiency (≥30 ng/mL)

3. Supplementation recommended?

☐ Yes ☐ No

Recommended dosage: _____

Duration of treatment: _____

4. Additional clinical notes

_____

_____

_____

Figure 8. Proposed template for documenting vitamin D status in the dental patient record. The structured template includes serum 25(OH)D measurements, the classification framework used to interpret vitamin D status, prescribed supplementation, treatment duration, adherence, and relevant clinical notes. It is intended to support interdisciplinary communication and consistent documentation when vitamin D assessment is clinically indicated. Vitamin D measurements should be interpreted within the appropriate medical context and should not independently determine periodontal diagnosis, prognosis, or treatment decisions.

To support clinical documentation, Figure 8 presents a proposed template for recording vitamin D-related information within the dental patient record. The template is intended as a practical aid when vitamin D assessment is clinically indicated, rather than as a validated diagnostic tool or a substitute for established periodontal risk assessment.

Overall, this case illustrates the need to interpret periodontal and systemic findings together while recognizing the limits of the available evidence. The improvement in periodontal inflammatory parameters despite persistent vitamin D deficiency reinforces the importance of conventional periodontal care and highlights the need for further research to clarify the possible modifying role of vitamin D in periodontal disease.

7 Conclusion

This case highlights the coexistence of periodontitis and persistent severe vitamin D deficiency in a young adult patient. Following conventional periodontal treatment, improvements in bleeding on probing and probing pocket depth were observed over six months, although residual plaque accumulation and gingival inflammation indicated that inflammatory control remained incomplete. Despite these clinical improvements, serum 25(OH)D remained markedly reduced, increasing only from 8.3 to 8.9 ng/mL, without achieving biochemical correction.

Periodontal improvement was observed despite the absence of documented biochemical correction of vitamin D deficiency, although the contribution of supplementation and other individual treatment components could not be determined. Furthermore, the limitations of the available clinical and radiographic records prevented a definitive assessment of changes in periodontal attachment and alveolar bone support.

While vitamin D may have biological relevance to periodontal health, its specific contribution to disease progression and treatment response remains uncertain and requires further investigation through well-designed clinical studies.

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Conflicts of Interest

Aida Meto served as an Editor-in-Chief of the *Journal of Oral and Dental Approaches* at the time of manuscript submission. To ensure the integrity of the peer-review process, Aida Meto was not involved in the editorial handling, peer review, or decision-making process for this manuscript, which was handled independently

by another editor. The remaining authors declare no conflicts of interest.

AI Use Statement

The authors declare that no generative AI was used in the preparation of this manuscript.

Ethical Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Aldent University (Protocol No. 4/4; 15 January 2025).

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Aida Meto is a specialist in orthodontics and endodontics and holds a PhD in Clinical and Experimental Medicine from the University of Modena and Reggio Emilia, Italy. In 2025, she earned the Italian National Scientific Qualification (ASN) as an Associate Professor. She lectures at Aldent University (AL) and the University of Modena (IT), with more than 80 peer-reviewed publications. Her research centers on oral microbiology, biofilm control, and advanced dental materials. She also serves as a reviewer and member of the editorial board for several international journals. (Email: aida.meto@ual.edu.al)