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CASE SERIES

Vitamin D in the Treatment of Recalcitrant Oral Lichen Planus: A Case Series

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Video abstract

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Background: Oral lichen planus (OLP) is a chronic mucocutaneous inflammation. Although the aetiology of OLP was unknown, dysregulation of T lymphocyte function (CD8 T lymphocytes) in autoimmune disease was considered a contributing factor. Vitamin D deficiency was found to exacerbate the condition by increasing oral keratinocyte apoptosis, which led to worsened clinical manifestations and delayed healing in OLP patients. Moreover, OLP management was challenging due to its frequent recurrences. Vitamin D receptor (VDR) has anti-inflammatory and immunomodulatory properties, along with a regulatory effect on keratinocyte proliferation and differentiation; thus, suggesting its possible role in the treatment of OLP.

Purpose: This case report analyzed recalcitrant OLP lasting over one year and highlighted the significant role of vitamin D in enhancing wound healing.

Case Presentation: Two female patients, aged 50 and 58 years, complained of pain in the left and right cheeks for one and three years ago. Both patients had received previous therapies but had shown no improvement. Intraoral examination revealed erosive lesions with Wickham striae on the left and right buccal mucosa. We diagnosed both patients with erosive and reticular OLP.

Case Management: The first-line treatment for both patients was corticosteroid mouthwash and anti-inflammatory therapy. An examination of vitamin D 25 (OH) levels was carried out as a risk factor screening due to the clinical manifestations being quite severe and persistent. The examination results indicated a vitamin D deficiency; both patients received vitamin D replacement therapy. The last control showed that patients' subjective and objective complaints experienced significant changes.

Conclusion: Vitamin D deficiency could increase the risk of OLP. Vitamin D examination and administration were important considerations in therapeutic decisions for OLP patients because they could accelerate the healing process.

Keywords: immune response, oral lichen planus, vitamin D deficiency, wound healing

Introduction

Oral lichen planus (OLP) is a chronic mucocutaneous inflammation that occurs on the oral mucosa.[1–5] The prevalence of OLP ranges from 0.1% to 2.5% of the world's population. OLP is categorized as an oral potentially malignant disorder (OPMD) by the World Health Organization (WHO) because 0.5–2% of cases can undergo malignant transformation.[1–3,6–8] The global prevalence of OLP displays geographical variation, with the highest rates in South-Central America (1.74%) and the lowest in India (0.49%). Individuals over 40 years of age have a nearly 3.5-fold higher prevalence of OLP.[9] OLP predominantly affects women, with a prevalence rate of approximately 60 to 69%, especially among those aged 30 to 50.[2,7] However, there is an increased risk of cases of erosive and/or atrophic lesions, tongue lesions, greater intake of alcohol/tobacco, accompanying hepatitis C virus infection, and elderly females in an age range of 60–70 years.[9] OLP is regarded as a psychosomatic disorder. OLP is associated with higher rates of anxiety, depression, and stress-related exacerbations. Elevated salivary or urinary cortisol levels in OLP patients further support this association.[10–12]

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The etiopathogenesis of OLP is not yet fully understood, but it is suspected to be related to autoimmunity due to the dysregulation of T lymphocyte function (CD8 T lymphocytes), which results in damage and apoptosis of basal keratinocyte cells on the epithelial surface. Additionally, other predisposing factors are suspected to be related to psychological stress, systemic diseases (hypertension, diabetes mellitus, and hepatitis C), medications (antidiabetics, antihypertensives, and hepatitis), dental materials, and hormonal factors. Other factors include microelement deficiencies, such as vitamin B12, folic acid, and vitamin D.[3–5,13]

Vitamin D is an essential steroid hormone that is fat-soluble. Sunlight exposure, skin synthesis, food, and supplements are sources of Vitamin D.[14,15] Vitamin D is involved in the maintenance of homeostasis in a variety of biological systems, including the acceleration of wound healing, aiding bone formation, contributing to the regulation of the cardiovascular system, suppressing tumor growth, exhibiting antibacterial properties, inhibiting the production of parathyroid hormone, and enhancing calcium absorption.[13–15] Additionally, vitamin D, which has an immunosuppressive and immunomodulatory effect, regulates the production of inflammatory cytokines and immunoglobulins by binding with vitamin D receptors (VDR) from activated T and B lymphocytes and inhibiting antigen presentation to T lymphocytes. This affects homeostasis in the oral cavity and inhibits the development of autoimmune diseases.[7,15,16]

Vitamin D deficiency is a non-communicable condition that has been identified as a worldwide pandemic, affecting 15.7% of the world's population. Studies indicate that the incidence of vitamin D insufficiency in Indonesia varies between 60% and 90%.[15,17,18] Vitamin D deficiency can increase the risk of higher cancer rates and cause various inflammatory disease conditions, such as rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, and OLP. Research shows that VDR levels in OLP patients are nearly 50% lower in the oral mucosa, likely due to an immune response.[7] Gholizadeh et al reported that the total vitamin D 25 levels in saliva were significantly lower in patients with OLP compared to the control group.[16] When compared to the control group, Ana et al also discovered that serum vitamin D levels were much lower in people with OLP, especially erosive-type OLP, and in people who had oral cancer because of erosive OLP.[7] However, patients with vitamin D deficiency are usually clinically asymptomatic and often unaware of it, resulting in slow healing of the disease lesions. Additionally, the treatment of OLP is quite challenging because it requires long-term therapy, a high recurrence rate, and a short remission period, making risk factor screening essential.[17,19] Therefore, this case report aims to analyze the important role of vitamin D in accelerating the healing of persistent OLP.

Case Series

Case 1

A 50-year-old female patient came to the Department of Oral Medicine, Padjadjaran University Dental Hospital, with complaints of sore and burning sensations on the left and right cheeks one year ago. The patient experienced discomfort, particularly when consuming spicy and hot foods, making eating and chewing challenging. The patient had used mouthwash but had not shown any improvement. The patient denied having a history of recurrent canker sores, as well as drug and food allergies. The Visual Analogue Scale (VAS) evaluation of pain severity shows a score of 5, indicating that the patient has experienced moderate discomfort. An extra oral exam showed no abnormalities. Intra-oral examination revealed reddish erosion lesions with non-scrapable striae on the left and right posterior buccal mucosa, maxilla posterior, and mandibula anterior gingiva (Figure 1). The patient was diagnosed with erosive and reticular oral lichen planus. The Depression Anxiety Stress Scale 21 (DASS-21) examination shows normal results. The patient was prescribed 10 mg of prednisone mixed with a 0.025% hyaluronic acid mouthwash 3 times a day (twice each in the morning, in the afternoon, and at night) at a dosage of 10 mL, petroleum jelly applied thinly three times a day on the upper and lower lips, and multivitamins.

One week later, the complaints on the cheek had significantly decreased, and the patient could eat comfortably. Intraoral examination shows that the lesions on the gingiva have healed, and the lesions on the left and right buccal mucosa have improved. We referred the patient for a complete hematology laboratory examination, cortisol hormone test, and total immunoglobulin E (IgE) allergy test to identify risk factors for OLP. Patients also undergo vitamin D testing not only to look for risk factors but also because the lesions tend to be persistent and heal slowly. The results of the complete hematology

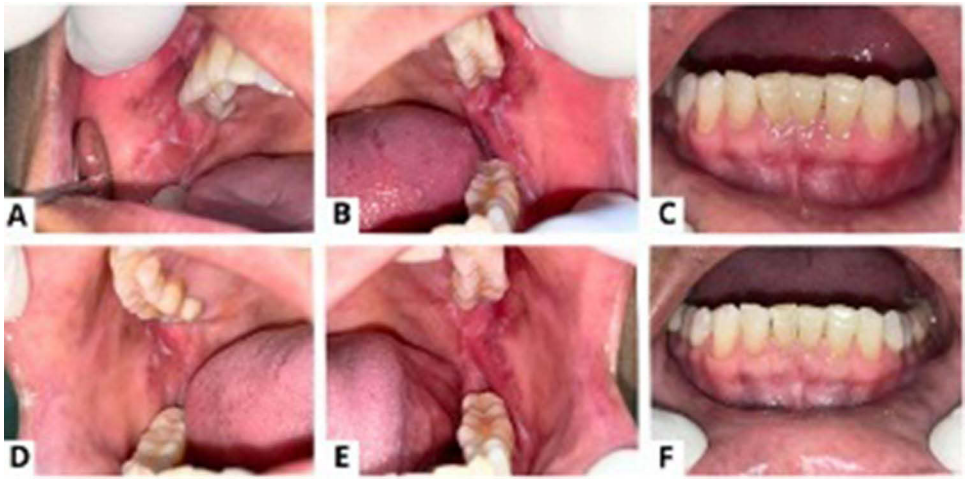

Figure 1 Clinical documentations at 1st Visit (**A**–**C**); The clinical photos from the last visit demonstrated improvement in the erosion lesion at both the buccal mucosa and attach gingiva (black arrow) (**D**–**F**).

examination, cortisol hormone, and IgE allergy test show normal results. However, the vitamin D test showed a mild vitamin D deficiency of 21 ng/mL (Normal value < 30 ng/mL) (Table 1). We instruct the patient to continue using the mouthwash as usual and start taking vitamin D3 (1000 IU) once daily in the morning for one month. The third visit showed that the subjective complaints had disappeared, with the VAS assessment result indicating a scale of 0. Intraoral examination shows that the

Table 1 Comparison of Clinical and Laboratory Findings of Both Patients

	Case 1	**Case 2**
Gender	Female	Female
Age	50 years	58 years
VAS before treatment	5 (moderate)	7 (severe)
VAS after treatment	0 (normal)	2 (mild)
DASS-21	Normal	Moderate depression, severe anxiety, and mild stress
Duration of complaint	1 years ago	3 years ago
Systemic disease	Denied	Diabetes
Clinical examination	Erythematous erosion with non-scrapable striae on the left and right posterior buccal mucosa, maxilla posterior, and mandibula anterior gingiva	Erosive lesions with striae that cannot be scraped off on the left and right buccal mucosa spreads to the lower labial mucosa
Diagnosis	Erosive and reticular oral lichen planus	Erosive and reticular oral lichen planus
Hematology laboratory	Normal	• ESR: 40 mm/hour (High) • IgE level: of 925.85 KIU/L (High)
Vitamin D level	21 ng/mL (mild deficiency)	16 ng/mL (moderate deficiency)
Treatment	• Prednison 10 mg mixed with 0.025% hyaluronic acid mouthwash • Petroleum jelly • Vitamin D3 1000IU • Multivitamins	• Prednison 10 mg mixed aquadest mouthwash • Hyaluronic acid gel • Petroleum jelly • Vitamin D3 1000IU • Multivitamins

Abbreviations: VAS, Visual analogue scale; DASS-21, Depression Anxiety Stress Scale 21; ESR, Erythrocyte sedimentation rate; IgE, Immunoglobulin E.

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lesions have significantly improved (Figure 1). The patient was advised to continue the prescribed mouthwash regimen and initiate oral supplementation with vitamin D3 (1000 IU) once daily in the morning for one month.

Case 2

A 58-year-old female patient came to the Department of Oral Medicine, Padjadjaran University Dental Hospital, with complaints of canker sores inside of her cheeks and lower lips for the past 3 years. It is causing significant discomfort, including pain, soreness, burning sensations, and numbness, especially when consuming spicy foods, which may exacerbate the condition. The patient experiences pain that limits her ability to open her mouth wide. The patient encounters minor challenges while eating, particularly with tougher foods. The complaints have not improved even after being treated with antibiotics. The patient has no history of recurrent ulcers and drug or food allergies, but has a history of diabetes and routinely takes glimepiride. The VAS assessment shows a score of 7, indicating severe pain.

An extraoral examination revealed dry and exfoliative lips, accompanied by red erosions with well-defined striae that were painful to touch or when opening the mouth (Figure 2). Intraoral examination of the right buccal mucosa that goes to the lower labial mucosa and the left buccal mucosa shows erosive lesions with striae that cannot be scraped off (Figure 2). The lesion was painful with no spontaneous bleeding. The patient was diagnosed with erosive and reticular oral lichen planus. The DASS-21 examination showed that the patient is experiencing moderate depression, severe anxiety, and mild stress. A complete blood count showed a high erythrocyte sedimentation rate (ESR) with a value of 40 mm/hour (normal value < 20 mm/hour) and a high total IgE level with a value of 925.85 KIU/L (normal value < 150 KIU/L). We instructed the patient to rinse with 5 mg of prednisone mixed with 10 mL of aquadest three times a day (twice each in the morning, in the afternoon, and at night) and apply a thin layer of 0.2% hyaluronic acid gel on the lower lip lesions and buccal mucosa, take multivitamins, and apply petroleum jelly to the upper and lower lips three times a day. We also refer patients to the periodontics department for scaling and recommend managing stress as well as consulting a psychologist or psychiatrist.

On the second visit, the mouth complaints had improved. The patient can now open their mouth wider and eat more comfortably. The patient is currently avoiding spicy and hot foods. The lip lesions showed improvement upon extraoral examination. The intraoral examination revealed that the lesion on the right buccal mucosa had extended to the lower labial mucosa, while the left buccal mucosa showed some improvement, although it was not yet significant. A vitamin D examination was conducted due to the suspicion of risk factors for vitamin D deficiency, considering the persistence of OLP lesions. The results of the vitamin D test indicate that the patient has moderate vitamin D deficiency (16 ng/mL) (Table 1). We instruct the patient to continue using the mouthwash as before and to take 1000 IU of vitamin D four times a day after meals. The next visit showed that the complaints in the oral cavity had significantly decreased, with a VAS score of 2, indicating mild pain. The intraoral examination also revealed a significant reduction in the lesions (Figure 1).

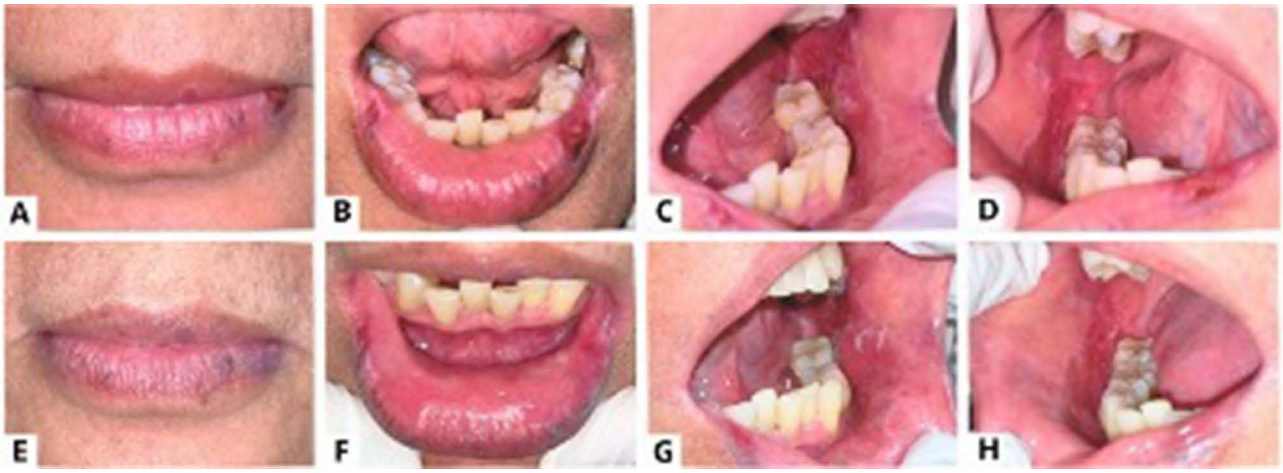

Figure 2 Clinical Photos at 1st Visit (**A–D**); Clinical photos at the last visit, the OLP lesion showed improvement (**E–H**).

The patient was advised to continue the prescribed mouthwash regimen and initiate oral supplementation with vitamin D3 (1000 IU) four times a day.

Discussion

Oral lichen planus (OLP) is a chronic inflammatory condition of the oral mucosa mediated by the immune system.[1–5] OLP is more prevalent in women than in men among adults. Most patients complain of a burning sensation, pain, sensitivity, and discomfort in the oral cavity, especially when eating certain foods.[3,20–22] OLP presents a variety of oral clinical manifestations, including reticular, plaque, and papule types, which are typically asymptomatic, as well as atrophic, erosive, and bullous types, which are typically symptomatic and can progress to malignancy. The buccal mucosa and the lateral aspect of the tongue typically exhibit the lesions. Approximately 10% of OLP patients present with exclusive gingival lesions, most commonly as erythematous lesions causing desquamative gingivitis, the typical form of gingival lichen planus.[3,20–22] OLP lesions usually last a long time with episodes of exacerbation and high recurrence, especially in erosive-type OLP. In this case, both patients are female, over 50 years old, and complain of pain and burning in the buccal mucosa, especially when eating spicy food.

Despite being treated by physicians, these complaints have been ongoing for 1–3 years without any spontaneous remission. These recalcitrant conditions are due to the complexity of finding the exact etiology of OLP. The second case demonstrates some risk factors of OLP, such as psychological stress, allergies, and diabetes. However, these factors were untreated. Physiological disorders, including stress, anxiety, and depression, can induce various alterations in the human metabolic and endocrine systems, notably an increase in cortisol levels. The adrenal cortex secretes cortisol, a 21-carbon glucocorticoid, to regulate the metabolism of proteins, carbohydrates, fats, and water, thereby influencing the nervous system and stress response. Previous research has shown that cortisol levels in the serum, urine, and saliva of OLP patients are significantly higher than control group. Therefore, psychological stress is more common in OLP patients.[23–26]

This case series reveals that systemic disorders, especially diabetes mellitus, have a substantial effect on how severe OLP is and how effectively it recovers. Diabetes may elevate the incidence of OLP due to the presence of human leukocyte antigen (HLA), specifically HLA-28, in both conditions. Recent research has observed elevated serum levels of interleukin 8 (IL-8) in both oral lichen planus (OLP) and diabetes, indicating a relationship between the two conditions, both independently and in their interconnectedness.[27]

The second patient with diabetes experienced more pain on the VAS scale (7, severe) than the first patient, who did not have a systemic ailment (VAS 5, moderate). Diabetes mellitus is known to induce low-grade inflammation that lasts a long time, difficulties with the immune system, and poor healing of wounds. All of these things make the symptoms of OLP worse and last longer.[27] The diabetic patient had a high ESR, which means there was inflammation in the body, and a high total IgE, suggesting an allergy or sensitivity. High levels of ESR and IgE in people with diabetes are associated with chronic inflammation and immunological activation, which is a major factor contributing to delayed wound healing and prolonged clinical symptoms in OLP.[27,28]

The diabetic patient also had a worse vitamin D deficiency (16 ng/mL, moderate deficiency) than the non-diabetic patient (21 ng/mL, mild deficiency). Diabetes mellitus is associated with lower vitamin D levels because diabetic nephropathy, insulin resistance, and greater body fat levels make vitamin D less accessible. 19 Vitamin D is particularly crucial for keeping the immune system in equilibrium, combating inflammation, generating new blood vessels, and growing keratinocytes. Diabetics with low vitamin D may have lesions that take longer to heal and worsen symptoms.[7,14,22]

The pathogenesis of OLP involves an immunological process. T cells trigger apoptosis in the basal cells of the oral epithelium, activating a protein cascade through lymphoepithelial interactions, including soluble cytotoxic molecules, ultimately leading to the rupture of the lamina propria. The first phase of OLP consists of dendritic cells, particularly Langerhans cells. These cells primarily function by presenting antigens obtained from external or internal sources to T lymphocytes. The T cells primarily implicated in the pathogenesis of OLP are T-helper (Th) cells, particularly Th-1, Th-2, and Th-17.[3,13]

Micronutrient deficiencies, such as vitamin D deficiency, can also lead to an increased occurrence of OLP.[7,14,22] It is challenging to subjectively and clinically identify vitamin D deficiency because patients typically do not exhibit specific symptoms, as evidenced in both cases where a vitamin D examination revealed the condition. Vitamin D deficiency was

detected in both patients despite different backgrounds. This confirms that vitamin D insufficiency is a common, underestimated risk factor for chronic OLP, especially when clinical lesions are persistent and recalcitrant. As demonstrated in the second case, diabetes mellitus may exacerbate vitamin D deficiency due to metabolic changes, but even in the absence of systemic disease (first case), mild vitamin D deficiency was found. This emphasizes that screening for vitamin D levels should be considered routine in OLP patients to identify modifiable risk factors early.

Vitamin D is a steroid hormone that performs various physiological functions by binding to the VDR. Vitamin D/VDR signaling demonstrates anti-inflammatory properties by downregulating Th-1, upregulating Th-2 response, and suppressing the expression of microRNA 802 (miR-802). The VDR receptor protects against microbial invasions of connective tissue because bacterial lipopolysaccharides can activate pro-inflammatory cytokines.[7,14,22] Research has also found that vitamin D/VDR signaling inhibits the Nuclear Factor-kappa Beta (NF-κB) pathway and increases the production of MiR-26,27 a/b. These factors lead to the suppression of oral keratinocyte apoptosis in epithelial cells and reduce the likelihood of OLP lesions developing. Vitamin D deficiency in OLP can disrupt the protective effects of vitamin D/VDR and decrease the number of Th-2 cells, particularly those involved in inflammatory pathways such as Th-1 and Th-17, thereby increasing oral keratinocyte apoptosis and leading to more severe clinical manifestations of erosions or ulcerations.[7,14,22]

OLP treatment aims to alleviate symptoms in patients, reduce recurrence, and decrease exacerbations. These goals can be achieved by administering an immunosuppressive or anti-inflammatory agent, which is corticosteroids. It is the gold standard for the treatment of OLP by suppressing T lymphocyte activity. Corticosteroids function by entering the target organ cells and binding to specific receptors in the cytoplasm. Subsequently, the steroid-receptor complex becomes active and enters the nucleus, binding to specific sites on the DNA. Corticosteroids decrease inflammation through several mechanisms, including inhibiting the leakage of leukocytes and plasma components, thereby reducing edema; preserving cell membrane integrity by preventing excessive cellular swelling; inhibiting lysosomal release from granulocytes; blocking phagocytosis; and stabilizing lysosomal membranes within cells.[29–32] Topical corticosteroids have fewer side effects but have the same or even higher efficacy than systemic steroids, making them the first line of treatment for OLP. Empirical evidence shows that mouthwashes are beneficial for patients with extensive symptomatic OLP, where lesions are unreachable for applying ointments or gels.[31,32] Therefore, in this case, the therapy chosen was the use of topical corticosteroids, specifically prednisone 10 mg, which was used as a mouthwash and administered three times a day. This therapy effectively reduced subjective and objective complaints in both patients.

An additional therapy to reduce inflammation and accelerate the healing of lesions in this case is the use of topical medication containing hyaluronic acid (HA). HA is a glycosaminoglycan composed of a polysaccharide that consists of disaccharide units formed from N-acetylglucosamine and D-glucuronic acid. HA is crucial in numerous biological processes, including cell signaling, morphogenesis, matrix organization, tissue hydration, lubrication, wound healing, gene expression regulation, and cell proliferation. Commercially, sodium hyaluronate, polyvinylpyrrolidone (PVP), and glycyrrhizic acid combine to form HA. Sodium hyaluronate forms a protective layer on the oral mucosa, enhancing tissue hydration and promoting healing processes. PVP is a hydrophilic polymer characterized by its mucoadhesive and film-forming properties, which contribute to improved tissue hydration. Glycyrrhetinic acid, a metabolite of glycyrrhizin found in licorice root, exhibits anti-inflammatory properties that facilitate cell proliferation and re-epithelialization by promoting the proliferation of basal keratinocytes. Due to its high viscosity, HA inhibits the entry of bacteria and viruses into the hyaluronate-rich pericellular matrix and protects wound healing from damage inflicted by free radicals.[15,32–35] HA significantly improves healing and tissue regeneration in both mouthwash and gel formulations. The patient's dry and exfoliating lips were treated with petroleum jelly, which serves as a protective agent that prevents transepidermal water loss, thereby maintaining skin moisture.[36]

In addition to the above-mentioned pharmacotherapy, our patients were also given the vitamin D supplement. This approach was considered after the initial treatment involving corticosteroids, HA, and petroleum jelly had not shown improvement in clinical signs, although the subjective symptoms were reduced. Follow-up to the laboratory results showing mild and moderate deficiencies, the patients were prescribed vitamin D, as indicated. Patients with moderate deficiency 25(OH) vitamin D levels less than 20 ng/mL (like the second case) can receive vitamin D3 therapy of 4000–6000 IU per day for 8 weeks, followed by maintenance therapy of 1000 IU per day. Meanwhile, patients with

vitamin D 25(OH) levels above 20–30ng/mL (mild deficiency, as in the first case) can receive maintenance therapy of 1000–2000 IU per.[35] In both cases, supplementation of vitamin D therapy plays a crucial role in maintaining adequate vitamin D levels in the body, which in turn enhances the main therapy and promotes optimal healing of OLP lesions. Based on a systematic review by Paria et al, the use of vitamin D supplements has shown significant improvements in OLP patients.[14] Vitamin D therapy has been demonstrated in several cases to accelerate the healing process for various oral conditions, including major recurrent aphthous stomatitis (RAS), traumatic ulcerative granuloma with stromal eosinophilia (TUGSE), xerostomia, and burning mouth syndrome.[15,37]

In this case series, both patients showed clinical improvement after receiving therapy, but the healing response varied. The first patient without systemic disease experienced complete resolution of pain symptoms (VAS reduced from 5 to 0) and full mucosal healing within one month of topical corticosteroid mouthwash, hyaluronic acid, and low-dose vitamin D supplementation (1000 IU daily). In contrast, the second patient, who had diabetes mellitus and moderate vitamin D deficiency, showed slower improvement, with a VAS score reduced from 7 to 2 and partial healing of lesions despite a higher dose of vitamin D (1000 IU, four times daily). The therapy was adjusted based on individual patient conditions. For the non-diabetic patient, standard corticosteroid mouthwash and low-dose vitamin D were sufficient. Meanwhile, for the diabetic patient with high ESR and IgE, persistent lesions, and systemic comorbidities, higher-frequency vitamin D supplementation and additional stress management were required. The present study shows that individualized treatment planning is essential in OLP, considering both systemic factors and laboratory profiles.

Vitamin D is very important for healing wounds on mucous membranes because it controls the growth of keratinocytes, changes immunological responses (changing Th1/Th17 to Th2 dominance), lowers pro-inflammatory cytokines, and helps new blood vessels form at the wound site.[14] Vitamin D reduces inflammation by inhibiting the proliferation of monocytes and T cells. This inhibits the secretion of inflammatory cytokines such as C-reactive protein (CRP), IL-6, and Tumor Necrosis Factor-α (TNF-α), while enhancing the synthesis of IL-10, which facilitates the healing process in the body. Vitamin D deficiency can increase TNF-α levels, leading to increased fibroblast apoptosis and decreased fibroblast proliferation, which results in a worsened lesion healing process. TNF-α also increases the production and activity of matrix metalloproteinase (MMP), especially MMP-9. MMP breaks down matrix proteins and growth factors, which are both important for healing wounds. As a result, the healing process becomes fragmented and intermittent, leading to chronic inflammation. Moreover, a vitamin D deficiency increases the susceptibility to infections by reducing macrophage phagocytosis, which in turn reduces the inhibition of interferon-mediated macrophage activation. Vitamin D plays a crucial role in the process of angiogenesis, which involves the formation of new blood vessels in the lesion area to supply oxygen and nutrients. Therefore, a vitamin D deficiency can slow down the formation of new blood vessels around the oral wound, thereby prolonging the recovery time.[15,38] Vitamin D deficiency can slow down various stages of lesion healing, prolong recovery time, and increase the risk of complications such as infections.

Adding vitamin D to the treatment sped up the healing process in both cases, especially when the first corticosteroid therapy alone was not enough. For the non-diabetic patient, correcting a modest deficiency was enough to rid them of all of their symptoms. In the meantime, the diabetic patient needed a greater dose to make up for both the deficiency and the slower healing of wounds that comes with diabetes. These results back up what we already know: vitamin D supplementation is not only advantageous but also necessary for treating OLP that is recalcitrant, extending the time between remissions, lowering the chances of recurrence, and maybe lowering the risk of malignant transformation.

Using vitamin D supplements led to a significant improvement in the clinical symptoms of oral lichen planus (OLP), which is consistent with other research that suggests it can be effective as an additional treatment. Several studies have looked into how vitamin D affects OLP and what causes its impacts on the immune system (Table 2). Most of the trials before this one used high-dose bolus regimens (50,000–200,000 IU monthly or single doses), but our cases showed clinical improvement with routine daily replacement dosing.[6,39–41] This suggests that getting enough serum may be more important than the dosing approach itself. Shoukheba (2020) also showed that using topical steroids with large doses of vitamin D lowered TNF-α levels and pain scores by a lot.[42] This evidence supports the idea that vitamin D has immunomodulatory and anti-inflammatory effects in the development of OLP.

The current results support the scientific basis for routinely testing OLP patients for vitamin D deficiency, especially those with persistent or stubborn lesions, since a lack of vitamin D may cause keratinocyte apoptosis and keep

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Table 2 Summary of Studies on Vitamin D Supplementation in OLP Patients

Author & Year	Study Design	Sample Size	Intervention	Results
Razi A et al (2018)[40]	RCT	100 samples; women; 35–45 years/ Perimenopausal; 4 weeks follow up	OLP patients with vitamin D serum levels < 30 ng/mL were divided into 2 groups: Group A: Steroid therapy Group B: Supplemented vitamin D (50.000IU)	The subject in group B showed improvements in the clinical appearance of the lesion between week 1 and week 4.
Gupta J et al (2019)[6]	Observational study	106 samples; all ages; both genders; 12 weeks follow up	OLP patients were divided into 3 groups based on Vitamin D levels, and history of Stress: Group 1: Stress and mild vitamin D deficiency (> 15 ng/mL); topical steroid and counselling Group 2: Severe vitamin D deficiency (< 15 ng/mL); topical steroid and vitamin D (60.000IU) Group 3: Stress and severe vitamin D deficiency (< 15 ng/mL); Topical steroid, vitamin D (60.000 IU), and counselling	There was a significant improvement in subjective and objective symptoms in vitamin D-supplemented patients with or without psychological counseling and topical steroid use for a short time.
Shoukheba (2020)[42]	RCT	30 samples; women; 45–65 years / post-menopausal; 2,4,6 weeks follow up	OLP patients with vitamin D serum levels < 30 ng/mL were divided into 2 groups: Group 1: topical steroid Group 2: topical steroid and vitamin D (200.000IU)	Both treatment modalities produced a statistically significant reduction in pain scores (VAS) and TNF-α levels.
Abdulcader Riyaz et al (2024)[39]	RCT	90 samples; both genders; 25–60 years; 12 weeks follow up	OLP patients were divided into 3 groups based on Vitamin D levels Group 1: Vitamin D level < 15 ng/mL; vitamin D (60.000 IU) Group 2: Vitamin D level 15–20 ng/mL; vitamin D (60.000 IU) Group 3: Vitamin D level > 30 ng/mL; vitamin D (60.000IU)	The improvement in Group I symptoms was statistically significant. The burning feeling improved with regular vitamin D supplements.
Shalaby R et al (2024)[41]	RCT	40 samples; 4 weeks follow up	OLP patients with vitamin D serum levels < 30 ng/mL were divided into 2 groups: Group A: systemic steroid and vitamin D (60.000IU) Group B: systemic steroid	The vitamin D group significantly reduced pain (VAS) and IFN-γ levels compared with the control group.

Abbreviations: RCT, Random Clinical Trial; VAS, Visual analogue scale3.

inflammation going. Fixing a vitamin D deficiency may improve how well treatment works, lower the chance of recurrence, and maybe even lengthen the time between remissions.

This case series has several limitations that should be acknowledged to ensure a balanced interpretation of the findings. Firstly, the small sample size, which involves only two patients, limits the generalizability of the results to broader populations with OLP. Secondly, the lack of control groups prevents direct comparisons with cases that did not receive treatment or those that did not use vitamin D, which is crucial for understanding the true effectiveness of vitamin D. Additionally, the method used to estimate vitamin D levels was restricted to serum 25(OH) vitamin D testing, without assessing active metabolite levels or VDR expression in oral mucosal tissues, which could offer further information about the local mechanisms of action. Furthermore, due to the inherent design of case series, establishing cause and effect is not possible, and factors such as stress, diet, and sun exposure were not considered. Future randomized controlled trials with larger sample sizes and comprehensive assessments are necessary to validate these preliminary observations and clarify the role of vitamin D in managing OLP.

Both patients expressed relief upon finally understanding an underlying factor contributing to their persistent symptoms. The first patient felt grateful that her pain resolved completely after years of discomfort, while the second patient, although still experiencing mild pain, felt reassured knowing that her condition was improving with treatment tailored to her systemic health. They highlighted that having an explanation for their condition and being involved in the treatment plan improved their confidence in managing their oral health. For clinicians, these cases highlight the importance of screening for vitamin D deficiency and systemic conditions such as diabetes in patients with recalcitrant OLP. Addressing modifiable risk factors like vitamin D insufficiency alongside standard therapy may enhance treatment outcomes and improve patients' quality of life. Future studies with larger samples are recommended to strengthen the evidence for incorporating vitamin D assessment and supplementation in OLP management protocols.

Conclusion

Vitamin D deficiency increased the risk of OLP. Examination and administration of vitamin D were important considerations in therapeutic decisions for OLP patients, because they could accelerate the healing process.

Consent Statements

Two patients, who provided written informed consent, approved the publication of this case report. The institution has also approved the publication of these articles.

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Disclosure

The authors report no conflicts of interest in this work.

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