What's new for the clinician – summaries of recently published papers (July 2025)

SADJ JULY 2025, Vol. 80 No.6 P325-P329

Edited and Compiled by Prof V Yengopal, Faculty of Dentistry, University of the Western Cape

1. PREVENTING WHITE SPOT LESIONS AROUND ORTHODONTIC BRACKETS: EFFICACY OF PRE-REACTED GLASS-IONOMER BARRIER COAT VERSUS SILVER DIAMINE FLUORIDE

White spot lesions (WSLs) around orthodontic brackets are areas of enamel demineralization caused by acid produced by bacteria in dental plaque, especially when oral hygiene is poor during fixed orthodontic treatment. These lesions can appear as early as 4 weeks after bracket placement and commonly form on the buccal surfaces near the gingival margin, particularly around the lateral incisors and canines. Males tend to be affected more than females.

Orthodontists have long attempted, with limited success, to decrease demineralization. The preventive effects of dentifrices and/or home use of fluoride solutions, for example, have been established. However, patient compliance with the traditional preventive measures is problematic. It has been shown that 52.5% of the patients did not utilize fluoride solutions at home[1]

Remineralizing therapy has recently gained popularity, and many studies suggest that they are as effective as traditional restorative approaches.[1] More recently, bioactive substances with the power of remineralization at the deep area of the body of the lesion have been utilized. These include Silver Diamine Fluoride (SDF) and a surface reaction-type pre-reacted glass ionomer (S-PRG) fillers containing dental materials. PRG filler is an effective additive due to its capacity to liberate and replenish fluoride ions. It releases additional active ions when exposed to water or acidic solutions. These ions can modulate acidic environments, turning the surrounding environment into reduced coating material (PRG Barrier) to reduce dentin hypersensitivity and prevent cavities on smooth surface areas.[1]

Elshenaway and colleagues (2025)[1] reported on an in-vitro study that sought to compare the preventive potential of silver diamine fluoride (SDF) versus PRG barrier coat on the development of White spot lesions (WSLs) around orthodontic brackets regarding surface elemental analysis and microhardness. In addition, the effect of these materials on the shear bond strength of orthodontic brackets was evaluated. The first null hypothesis was that surface elemental analysis and microhardness will not change significantly after tested materials were applied. The second null hypothesis was that the applied, tested materials will not interfere with the shear bond strength (SBS) shear bond strength (SBS) of orthodontic brackets to the enamel.

Materials and methods

The materials that were used in this study are shown below.

This study was conducted as an in vitro experimental study using a split-tooth design. The specimens were designed to be surface characterized twice (before bonding and after bracket removal) by SEM, EDX, and microhardness. In addition, the effect of the tested materials on brackets' shear bond strength (SBS) was studied.

The estimated sample size of 21 samples per group was found to be the minimum needed. 105 extracted human permanent maxillary first premolars were chosen as the sample based on the specified inclusion and exclusion criteria. Teeth were examined under a stereomicroscope using 10x magnification. Sound completely formed maxillary first premolars typically removed for orthodontic

Material	Composition
Artificial saliva	(Na-3PO4 (3.90 mM), NaCl (4.29 mM), KCl (17.98 mM), CaCl2 (1.10 mM), MgCl2 (0.08 mM), H2SO4 (0.50 mM), NaHCO3 (3.27 mM)4
38% SDF solution	Silver particles and 38% (44,800 ppm) fluoride ion, which at pH 10 is 25% silver, 8% ammonia, 5% fluoride, and 62% water.
PRG Barrier Coat	Base: S-PRG filler based on fluoroboroaluminosilicate glass, Distilled water, Methacrylic acid monomer, and others Active: Phosphonic acid monomer, Methacrylic acid monomer, Bis-MPEPP, Carboxylic acid monomer, TEGDMA, Polymerization initiator, and others
Ortho Solo Primer	Highly filled light-cure adhesive, Bis-GMA resin
Grengloo (Two-way color change adhesive)	Uncured methacrylate ester monomers (20–38%), inert mineral fillers, fumed silica, activators, and preservative
Demineralizing solution	50 mMol acetic acid derivation, 2.25 mMol CaCl2 2H2O, 1.35 mMKH2PO4; 130 mm KCl 4
Remineralizing solution	(1.5 mMol Calcium Chloride-0.9 mMol Sodium Phosphate-150 mMol Potassium Chloride)

www.sada.co.za / SADJ Vol. 80 No.6

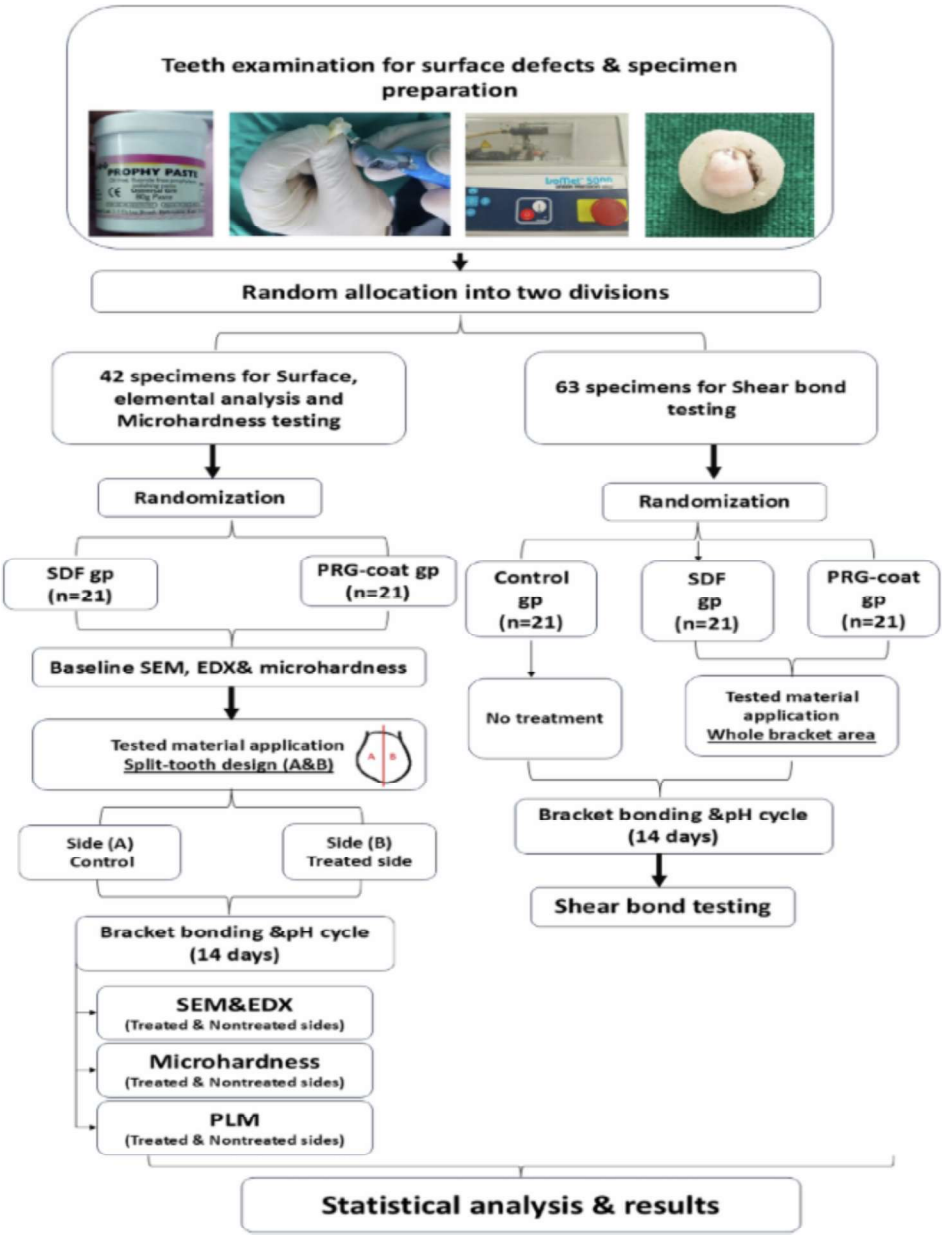
treatment were the inclusion criterion. The following teeth met the exclusion criteria: those with evident buccal flaws, microcracks, erosions, caries, or restorations.

After extraction, a hand scaler was used to remove calculus and soft deposits from the teeth. The teeth were carefully washed with distilled water after being cleaned with fluoride- and oil-free pumice (Prophy paste). Teeth crowns were separated at CEJ using a diamond saw. Each tooth's coronal portion was subsequently embedded in self-cured acrylic resin blocks with buccal surfaces facing upward. The teeth were housed in laboratory-prepared artificial saliva in an incubator at 37 ° C, which was changed daily until the experiment was finalized. Fig 1 provides details of the methodology in a table format.

For elemental analysis and microhardness, the buccal surfaces of 21 specimens from each group were divided vertically into two halves by a permanent marker. The control side (section A) received no treatment in these specimens. The other half was the test side, where the enamel surface would be treated with its respective remineralizing anti-cariogenic agent (section B).

For shear bond testing, three groups ($n=21$/group) were allocated randomly: the Control group, which received no treatment before Athe whole bracket area; and the PRG-coat group, which received a PRG-barrier coat over the entire bracket area.

All groups' samples underwent remineralization/demineralization pH cycles for 14 days. Each cycle consisted of four phases: a demineralizing phase lasting 120 minutes, a washing phase lasting 30 seconds, a remineralizing phase lasting 60 minutes, and a final washing for 30 seconds. The specimens were subjected to the remineralizing solution for a 6-hour "night" period. The pH of both solutions was measured using a pH meter.



Surface characterization

Specimens designated for surface evaluation by SEM, microhardness, and *Polarized light microscopic examination* (PLM) were assessed as follows:

- **Scanning electron microscopy (SEM) and quantitative elemental analysis (weight %) by EDX spectrometry**

Sample surfaces on sections A and B were examined using **SEM attached** with EDX Unit SEM. *For SEM evaluation*, the samples were carefully dried and gold-plated. Then, the samples were fixed to investigate the enamel surface. In addition, Both Calcium (Ca) and Phosphorus (P) content at the enamel surface of each specimen were analyzed quantitively as weight percentage using EDX. This step was done for all specimens at baseline and after bracket removal for non-treated (section A) and treated sides (section B).

- **Microhardness**

Microhardness was measured using a Digital Vickers Microhardness testing machine using 300 gm force for 10 seconds with a Vickers' diamond indenter and 10X objective length. Three indentations were made on the surface of each specimen, and the average was calculated. This step was done for all specimens at line baseline and after bracket removal (for treated and non-treated sides).

- **Polarized light microscopic examination (PLM)**

Each tooth was sectioned vertically in a buccolingual direction utilizing a diamond saw to split up section A from section B. Each half could be analyzed individually under PLM. The images were captured using a PLM built-in camera via image software LAS EZ version 3.0.0.

- **Shear bond strength (SBS) evaluation**

A universal testing machine (INSTRON 3365) was used to measure SBS. The debonding force was measured in Newtons (N) and then divided by bracket base area (10.25 mm^2) to calculate SBS in MPa.

To ensure consistency in data collection, each examiner independently assessed the same samples under standardized conditions. The degree of agreement between examiners was evaluated using Cohen's kappa (κ) test, yielding a kappa value of 0.9, which reflects excellent agreement.

Results

To better understand the alterations occurring after treatment application, the researchers captured an SEM image of a typical, intact enamel surface (Baseline) before treatment. The normal enamel surface appeared smooth in architecture with a layer of a prismatic enamel covering its external surface. Side A of both SDF and PRG groups (untreated side) depicted the typical etching pattern irregular with uneven depressions and type-I pattern which include removal of the rod body and maintenance of rod boundary and interrod area. However, side B of SDF (treated side) showed enamel remineralization that covered almost the whole enamel surface with many large calcium crystals combined. Tiny pores between the calcium crystallites were also depicted in small areas. Moreover, side B of PRG (treated side) revealed enamel remineralization, which covered almost the enamel surface with many large calcium deposits coalesced with small porosities between the calcium crystallites.

The data obtained from EDX spectrometry was Ca and P weight %, so the researchers calculated Ca/P ratios for each group based on their content. Paired T-test results of both groups showed significant differences between Ca/P ratios at baseline and non-treated sides ($P=0.000^*$). In addition, the treated side of both groups showed significantly higher Ca/P ratios than those of non-treated sides ($P=0.000^*$). Regarding the SDF group, the Ca/P ratio of the treated side did not differ significantly from the baseline ratio ($T=1.6$, $P=0.121$). For the PRG-coat group, the Ca/P ratio of the treated side showed a significantly lower Ca/P ratio than baseline ratios ($T=3.4$, $P=0.003^*$). Notably, non-significant differences were found in the mean difference of Ca/P ratios between treated and baseline groups according to T-test outcomes ($T=0.99$, $P=0.32$).

Baseline hardness for both groups was significantly higher than the treated sides, which was significantly higher than the non-treated sides ($P=0.000^*$) and ($P=0.000^*$) in accordance with the Paired T-test. In comparing SDF with PRG-coat groups, the T-test showed non-significant differences in mean differences between treated and baseline values ($T=0.32$, $P=0.74$).

Mean Shear bond strength (SBS) values showed a non-significant difference between the groups ($F=2.51$, $P=0.089$), with the SDF group showing slightly higher values followed by PRG-coat groups than the control group.

Polarized light microscope (PLM) for Baseline normal enamel showed a standard prismatic surface enamel layer with almost typical homogenous subsurface enamel reflecting normal mineralization and birefringence of enamel. However, side A of SDF (untreated side) and side A of PRG (untreated side) showed surface demineralization with a positive birefringent demineralized enamel band extending beneath an intact surface layer.

The treated side of SDF showed widely distributed areas of remineralized enamel, small, demineralized regions, and an evident remineralized surface enamel layer. The treated side of PRG showed alternative areas of remineralized enamel together with small, demineralized areas and elimination of demineralization with the appearance of a surface remineralized layer.

Conclusion

The researchers found that applying either SDF varnish or PRG-barrier coat before bonding orthodontic brackets could effectively prevent the development of WSL and achieve surface enamel protection. In addition, the two applied varnishes showed slightly higher shear bond strength of orthodontic brackets compared to the control group, with the SDF slightly higher than PRG.

Implications for practice

These findings indicate that incorporating these protective agents in orthodontic practice may enhance enamel preservation without compromising bracket adhesion.

REFERENCE

1. Elshenawy EA, Alawy SB, Alghonemy WY, El Dosoky AI. Preventing white spot lesions around orthodontic brackets: efficacy of pre-reacted glass-ionomer barrier coat versus silver diamine fluoride: an in vitro study. BDJ open. 2025 May 5;11(1):45.

www.sada.co.za / SADJ Vol. 80 No.6

2. THE ROLE OF VITAMIN D DEFICIENCY IN THE DEVELOPMENT AND SEVERITY OF ORAL LICHEN PLANUS: A CASE-CONTROL STUDY

Lichen planus (OLP) is a chronic inflammatory, autoimmune ailment, primarily affecting the skin, oral and genital mucosa, and with a potential for undergoing malignant alterations. Oral involvement is a common occurrence, and in 15–35% of cases, oral mucosa may be the only affected site of the disease. Oral lichen planus (OLP) represents the mucosal counterpart of the cutaneous LP and typically presents with episodes of exacerbation and remission. OLP has an age and gender predilection, primarily affecting females over 40 years of age[1].

Recent studies have reported that the malignant transformation rate of OLP ranges from 0.44% to 2.28% [1]. However, there is an increased risk of malignant potential in cases of erosive and/or atrophic lesions, tongue lesions, greater intake of alcohol/tobacco, and an accompanying hepatitis C virus infection.

OLP lesions characteristically manifest as bilaterally symmetrical reticular lesions on the buccal mucosa, tongue, and gingiva, although, involvement of the palatal mucosa, lips, and floor of the mouth is infrequently seen.

OLP may manifest a plethora of clinical forms, and range from reticular, erosive, atrophic, plaque-like, papular, and bullous lesions. Generally, reticular lesions are the commonest, and the bullous/papular forms are the rarest oral presentations. The most common reticular form of OLP is asymptomatic, whereas, the atrophic, erosive, and bullous forms usually cause pain, burning sensations, difficulty in mastication and speech, and deteriorated oral hygiene. These forms are also associated with negative psychosocial outcomes due to the chronic, uncertain clinical patterns and potential for malignant transformation, thus affecting the patient's quality of life[1].

Despite breakthrough research and substantial knowledge advancements, the etiopathogenesis of OLP is still ambiguous, and OLP is regarded as a chronic T-cell-mediated disorder of unknown etiology. However, a plethora of multifactorial predisposing factors, such as autoimmunity, microorganisms, infective agents, drugs and dental materials, nutritional deficiencies, psychological stress, and genetic predisposition may also have a role to play.

Treatment strategies are focused on precluding the excruciating symptoms, hastening the remission of erosive lesions, enhancing the asymptomatic periods, diminishing the malignant transformation risk, and maintaining good oral hygiene and dental status. Several pharmacological and non-pharmacological treatment regimens have been advocated for the management of OLP. The pharmacological therapeutic modalities used in the treatment of OLP include corticosteroids (topical, intralesional, and systemic steroids), Immunosuppressants (tacrolimus, azathioprine, cyclosporin, and mycophenolate mofetil,), immunomodulators (levamisole and thalidomide), and retinoids. Various non-pharmacological regimens, such as Light amplification by stimulated emission of radiation (LASER) therapy, photodynamic therapy, and Psoralen plus ultraviolet-A radiation (PUVA) therapy are also used in OLP treatment[1].

In recent years, the relationship between vitamin D (VD) and immunologically mediated diseases has drawn increasing attention. VD, a fat-soluble vitamin, exerts its action through VD receptors (VDR), which are abundantly present on T-lymphocytes. There is emerging evidence about the role of active VD (25-hydroxy VD3) in the control of immune reactions. To date, the responsibility of serum vitamin D level (SVDL) as a possible risk element in the OLP development and its advancement into oral squamous cell carcinoma is unclear.

Furthermore, a recent meta-analysis revealed that OLP participants experience a greater incidence of stress, anxiety, and depression. Shalaby et al (2025) [2] reported on a study that sought to compare SVDL between OLP patients and healthy controls as well as between OLP subtypes representing different severities of the condition. In addition, the current study compared SVDL in OLP lesions that showed signs of dysplasia in histopathologic examination (Dysplastic) and those with no signs of dysplasia (non-dysplastic) to ascertain whether VD deficiency ought to be regarded as a contributing factor for the onset, progression, to more severe forms, or probably malignant transformation of OLP. Additionally, psychological assessment utilizing the Depression Anxiety and Stress Scale (DASS-21 scale), and socioeconomic status using Kuppuswamy's scale was performed.

Methodology

This was an Observational case-control study that followed the STROBE guidelines. Seventy participants were assigned into two groups. Thirty-five patients in Group A had a clinical picture that matches the clinical diagnosis of OLP and histological findings that confirm the diagnosis, mainly liquefaction degeneration of the basal cells and a band-like area of lymphocytic infiltration limited to the upper part of connective tissue, without exclusion of dysplasia. Clinically, patients were classified into three main types: reticular (including papular or plaque type), atrophic, and bullous erosive ulcerative OLP. Histologically, any signs of epithelial dysplasia were recorded, so lesions were further divided into dysplastic and non-dysplastic OLP. Group B included 35 healthy volunteers which were matched by sex, age, and dietary habits (whether they were vegetarian or non-vegetarians).

Exclusion criteria involved were Patients receiving any medication including corticosteroids for the management of OLP or any other condition for the past 6 months; Individuals who have been using multivitamins, different types of VD supplements, or medications that might alter VD measures; Patients with oral lichenoid lesions including lesions caused by contact with amalgam, reaction to drug or in the setting of graft versus host disease; Patients who smoke and/or use smokeless tobacco because additional keratotic lesions caused by tobacco products could mislead the examiners; Pregnant females or individuals with any systemic medical condition; and when VD ≥ 100 ng/ml as it is considered VD toxicity.

To gather information, each participant filled out a structured questionnaire. Age, sex, country, early living circumstances

(rural or urban), systemic medical conditions, history of drug intake, consumption of VD supplements, dietary habits, psychological assessment applying the DASS-21 scale, and socioeconomic status using Kuppuswamy's scale were among the details it contained. Also, investigators collected 5 ml of peripheral venous blood from all participants. For the quantification of VD3, the enzyme-linked immunosorbent assay (ELISA) was employed to analyze all samples concurrently in accordance with the manufacturer's instructions. A competitive ELISA-based technique was performed to measure the level of 25-OH VD3/D2 in blood samples.

According to the Society of Clinical Endocrinology, the blood level of 25(OH)D above 20 ng/ml is considered sufficient to prevent rickets and osteomalacia in children and adults respectively, with normal being between 30 and 100 ng/ml, deficiency between 12 and 19 ng/ml and serious deficiency < 11 ng/ml. In the present study, SVDL ≤ 20 ng/ml were assigned to the VD deficiency set, while levels ranging between 20 and 100 ng/ml were classified as VD sufficiency, while levels ≥ 100 ng/ml were considered VD toxicity.

Results: The present study included 70 Egyptian participants involving 35 OLP participants and 35 healthy volunteers. According to Kuppuswamy's socioeconomic status scale, there was a statistically significant difference between OLP and the healthy controls ($P = 0.028$); while, no significant difference was noted among different types of OLP cases ($P = 0.895$). It was found that the majority of the OLP group (45.7%) was of middle level compared to the control group (28.6%) while the majority of control group was of upper middle level (57.1%). Regarding results of the DASS-21 scale, a statistically significant greater number of patients having depression, anxiety and stress in OLP group than in control group ($P = 0.004$, 0.036, and 0.05, respectively). Moreover, there were statistically significant differences in number of patients having depression, anxiety and stress between the three types of OLP ($P = 0.05$, 0.021 and 0.011 respectively). It was noticed that severe depression occurred in 50% of erosive OLP and 50% of atrophic OLP compared to none of those with reticular type. Similarly, severe anxiety and stress occurred in 100% of participants with erosive OLP, while none of those with atrophic or reticular lesions had severe anxiety or stress.

Regarding SVDL, the mean value ± SD in OLP was 16.7 ± 5.02 ng/ml while in controls was 29 ± 8.73 ng/ml. Statistically significant greater values of SVDL were found in the healthy controls than the OLP group ($P \leq 0.001$, effect size = 0.818, and CI of -14.8 to -8.00). There was a statistically significant difference between reticular and atrophic forms of OLP ($P = 0.012$), as well as reticular and erosive types ($P = 0.029$), while there was no statistically significant difference between atrophic and erosive types of OLP ($P = 0.344$). Similarly, by comparing each type of OLP and controls, there were statistically significant differences between each type and controls ($P = 0.012$, ≤ 0.001, and ≤ 0.001) for reticular, atrophic, and erosive types, respectively. Although lower SVDL was detected in patients with dysplastic lesions (14 ± 4.32) than non-dysplastic lesions (17 ± 5.09), the difference was not statistically significant ($P = 0.187$).

When the two study groups were compared in terms of VD deficiency or sufficiency, there was a statistically significant difference between the two study groups ($P \leq 0.001$). Furthermore, it was clear that VD deficiency was more pronounced in erosive and atrophic types than reticular types, with a statistically significant difference that reflects the effect of VD deficiency on the severity of the condition ($P \leq 0.001$).

Statistical analysis was performed between some risk factors for OLP and VD deficiency and it was noticed that only two factors namely, depression and sun exposure significantly affected the number of patients having VD deficiency.
Also, there was a statistically significant direct relation between the progress of the reticular type into more severe forms, namely atrophic and erosive ($P = 0.022$ and 0.004, respectively).

A univariable binary logistic regression analysis was done for determining the risk of OLP from VD deficiency. It revealed that VD deficiency significantly contributed to the development of OLP ($p < 0.001$), with about 26 times increased likelihood of OLP among patients having VD deficiency (OR = 26.156, 95%CI = 7.083–96.593, B coefficient = 3.264, SE = 0.667). This was followed by multivariable analysis to determine the risk of OLP from VD deficiency after controlling for confounding factors which are: age, sex, socioeconomic level, diet habits, duration of sun exposure, and presence of moderate to severe depression, anxiety and stress. The results showed that VD deficiency still significantly contributing to OLP development with an increased risk to about 34 times (AOR = 34.161) after controlling confounders. The multivariable model displayed an accuracy of 85.7% and explained 62.5% of variations in the OLP disease. Higher SVDL significantly reduced the risk of having OLP (OR = 0.5, 95%CI = 0.33–0.76, $p = 0.001$).

Higher SVDLs was significantly associated with lower risk of dysplasia ($p = 0.041$). Likewise, more sun exposure was significantly associated with reduced dysplasia ($p = 0.045$). Socio-economic status, depression, age, sex and diet didn't show significant associations in this multivariable regression analysis.

Conclusion

The present study corroborates the evidence from previous reports that VD deficiency is a potential risk factor affecting the initiation of OLP and validates its association with more severe forms of the disease, including the incidence of epithelial dysplasia.

Implications for practice

Checking patients VD level who present with signes and symptoms of OLP could reduce the severity and duration of the symptomatic episodes of the condition.

REFERENCE

1. Saeed S, Choudhury P, Ahmad SA, Alam T, Panigrahi R, Aziz S, Kaleem SM, Priyadarshini SR, Sahoo PK, Hasan S. Vitamin D in the Treatment of Oral Lichen Planus: A Systematic Review. Biomedicines. 2022 Nov 17;10(11):2964.
2. Shalaby R, Nabil G, Ibrahim S, Kotb AA, Amer H, Afifi S. The role of vitamin D deficiency in the development and severity of oral lichen planus: a case-control study. Clinical Oral Investigations. 2025 Jun;29(6):1-2.