e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(3); 1728-1734

Original Research Article

Comparative Assessment of Lesion Severity, Pain Reduction, And Adverse Effects of Topical Coconut Cream and Clobetasol Propionate 0.05% in Oral Lichen Planus: A Randomized Double-Blind Clinical Trial

Upasana Pal

Assistant Professor, Department of Pharmacology, Vyas Medical College & Hospital, Jodhpur, India

Received: 11-02-2026 / Revised: 11-03-2026 / Accepted: 29-03-2026
Corresponding Author: Dr. Upasana Pal
Conflict of interest: Nil

Abstract:

Background: Oral lichen planus (OLP) is a chronic, immune-mediated mucocutaneous disorder that poses a significant therapeutic challenge due to its relapsing nature. Clobetasol propionate 0.05% ointment remains the gold standard for topical management; however, its prolonged use is associated with undesirable side effects including mucosal atrophy and secondary candidal infection. Coconut cream, rich in medium-chain fatty acids with established anti-inflammatory and immunomodulatory properties, presents a promising natural alternative. This study aimed to compare the clinical efficacy and safety of topical coconut cream versus clobetasol propionate 0.05% ointment in patients with symptomatic OLP over an eight-week period.

Methods: A double-blinded randomized controlled trial was conducted on 84 histopathologically confirmed OLP patients recruited from a tertiary dental institution. Participants were randomly allocated into two equal groups (n=42 each): Group A received topical coconut cream and Group B received clobetasol propionate 0.05% ointment, applied three times daily for eight weeks. Clinical outcomes were assessed at baseline, Week 2, Week 4, and Week 8 using the Thongprasom sign scoring criteria and the Visual Analogue Scale (VAS) for pain. Adverse effects were monitored throughout the study.

Results: Both groups demonstrated statistically significant reductions in lesion severity and pain scores from baseline. The clobetasol group achieved a greater mean reduction in Thongprasom score (69.5% vs. 60.5%, p=0.031) and VAS pain score (79.5% vs. 70.6%, p=0.018) compared to the coconut cream group at eight weeks. However, the adverse effect profile favoured coconut cream significantly (9.5% vs. 33.3%, p=0.012), with no cases of oral candidiasis or mucosal atrophy in Group A.

Conclusion: Clobetasol propionate demonstrated superior short-term efficacy in reducing OLP lesion severity and pain; however, topical coconut cream offered clinically meaningful improvement with a substantially safer adverse effect profile. Coconut cream may serve as a viable alternative in patients intolerant to corticosteroids or in mild-to-moderate presentations of OLP.

Keywords: Oral Lichen Planus; Coconut Cream; Clobetasol Propionate; Topical Corticosteroid; Randomized Controlled Trial; Natural Therapeutics.

DOI: 10.25258/ijcpr.18.3.282

Introduction

Oral lichen planus (OLP) is a chronic, non-infectious, immune-mediated inflammatory condition affecting the stratified squamous epithelium of the oral mucosa and the adjacent lamina propria. First described by Wilson in 1869, this condition holds a well-established position among the most commonly encountered oral mucosal diseases encountered in clinical dental practice. [1] It may present simultaneously or sequentially with skin, genital mucosa, scalp, and nail involvement, although oral involvement often occurs in isolation and tends to be the most clinically significant owing to its chronic, symptomatic, and potentially pre-malignant nature. [2]

The global prevalence of OLP has been variably reported, ranging from 0.5% to 2.0% of the general population, with a predilection for middle-aged women between the fourth and sixth decades of life. [3] This demographic predilection may reflect underlying hormonal or immunological differences between the sexes that render women more susceptible to antigen-driven lymphocytic activation. In terms of clinical morphology, OLP manifests across a spectrum of subtypes including reticular, papular, plaque-type, atrophic, erosive, and bullous forms. The reticular subtype, characterised by interlacing white striations known as Wickham's striae, is the most frequently

encountered variant, whereas erosive and atrophic forms tend to produce significant patient discomfort and require active therapeutic intervention. [4]

The pathogenesis of OLP is fundamentally immunological in nature. Antigen-specific and non-specific mechanisms cooperate to produce the characteristic band-like lymphocytic infiltrate observed at the epithelium-connective tissue junction. Antigen-specific pathways involve the processing and presentation of unknown antigens by basal keratinocytes to CD4+ T-helper and CD8+ cytotoxic T lymphocytes, triggering keratinocyte apoptosis via Fas-FasL interactions and perforin/granzyme-B mediated pathways. [5] Non-specific mechanisms encompass mast cell degranulation and matrix metalloproteinase activation, which collectively enhance basement membrane disruption and facilitate intra-epithelial T-cell infiltration. [6] Although the precise antigenic trigger remains unidentified, proposed etiological factors include stress-induced neuroimmune dysregulation, genetic susceptibility linked to specific HLA haplotypes, hepatitis C viral infection, dental materials such as amalgam, and the use of certain medications. [7]

The symptomatic management of OLP remains a clinical challenge, particularly because the condition follows a relapsing and remitting course without a definitive cure. The principal therapeutic objective is to achieve resolution of painful erosive lesions, reduce mucosal inflammation, and prolong remission. [8] Topical corticosteroids, particularly super-potent agents such as clobetasol propionate 0.05%, are regarded as the first-line pharmacological treatment for symptomatic OLP and have been validated through multiple randomized controlled trials. [9] Clobetasol propionate exerts its anti-inflammatory effect by inhibiting the production of pro-inflammatory cytokines, reducing capillary permeability, suppressing T-lymphocyte migration, and downregulating the transcription of genes encoding interleukins and adhesion molecules. However, the chronic nature of OLP mandates prolonged treatment, which is invariably associated with complications including secondary oral candidiasis, mucosal atrophy, adrenal suppression with systemic absorption, and tachyphylaxis. [10]

In response to these limitations, substantial investigative interest has been directed toward natural, plant-derived compounds with anti-inflammatory and immunomodulatory properties as alternatives or adjuncts in OLP management. Coconut cream, derived from the expressed milk of mature coconuts (Cocos nucifera), is a rich source of medium-chain fatty acids (MCFAs), primarily lauric acid, myristic acid, and caprylic acid. These MCFAs have been demonstrated to exert potent anti-inflammatory, antifungal, antimicrobial, and wound-healing effects through multiple cellular and molecular mechanisms. [11] Lauric acid, the predominant fatty acid constituent, is converted in the body to monolaurin, which disrupts microbial lipid membranes and modulates T-cell mediated inflammatory responses. Furthermore, polyphenolic antioxidants present in virgin coconut cream scavenge reactive oxygen species and attenuate oxidative stress-driven inflammatory cascades that contribute to epithelial keratinocyte damage in OLP. [12]

Prior animal and in vitro studies have confirmed the anti-inflammatory analgesic and wound-healing properties of virgin coconut oil applied topically to mucosal and cutaneous surfaces, with favourable findings related to enhanced neovascularization, collagen synthesis, and fibroblast proliferation. [13] Despite this mechanistic rationale and the well-established safety profile of coconut-based products, no adequately powered, double-blind randomized controlled trial has previously compared topical coconut cream with clobetasol propionate for the management of OLP. The current trial was therefore designed to address this evidence gap and generate high-quality comparative data on the clinical efficacy and safety of these two topical agents in patients with histopathologically confirmed symptomatic OLP.

Materials and Methods

Study Design: This was a prospective, double-blinded, parallel-group, randomized controlled trial conducted over a period of eighteen months at a tertiary-care dental teaching institution. Ethical clearance was obtained from the Institutional Ethics Committee prior to study commencement, and the study was registered with the Clinical Trials Registry. Written informed consent was obtained from all participants in their preferred language. The trial was conducted in strict adherence to the Declaration of Helsinki and the Indian Council of Medical Research (ICMR) guidelines for clinical trials.

Eligibility Criteria: Patients presenting with clinically and histopathologically confirmed OLP, based on the modified WHO diagnostic criteria (2003), were eligible for inclusion. Additional inclusion criteria comprised: age between 18 and 70 years; active symptomatic OLP with a minimum Thongprasom sign score of ≥2; willingness to participate and complete the full 8-week treatment protocol; and no concurrent topical or systemic corticosteroid therapy for at least four weeks prior to enrolment. Patients were excluded if they had: known allergy or hypersensitivity to coconut products or corticosteroids; lichenoid drug reactions or contact hypersensitivity; pregnancy or lactation; concurrent oral candidiasis; systemic diseases

requiring immunosuppressive therapy; or a history of confirmed malignant transformation of OLP.

Sample Size: Sample size was calculated based on an anticipated 25% difference in Thongprasom score reduction between the two groups at eight weeks, with an alpha error of 0.05 and 80% power. This calculation yielded a minimum of 38 participants per group; accounting for a 10% dropout rate, 42 participants per group (n=84 total) were recruited.

Randomization and Blinding: Eligible participants were assigned to treatment groups using a computer-generated randomization sequence with sealed, sequentially numbered opaque envelopes prepared by an independent statistician. Randomization was performed in a 1:1 ratio. To achieve double-blinding, both coconut cream and clobetasol propionate ointment were prepared and dispensed in identical labelled containers coded as "Medication A" and "Medication B" by a pharmacist not involved in clinical assessment. Neither the patients nor the evaluating clinicians were aware of the treatment allocation throughout the study duration.

Interventions: Group A (Coconut Cream Group): Participants received freshly prepared, standardized topical coconut cream (25% w/w coconut milk extract in a pharmaceutical-grade cream base, containing 52% lauric acid as confirmed by gas chromatography), applied directly to the OLP lesions three times daily after meals, using a gloved finger, for 8 weeks. Patients were instructed to refrain from eating or drinking for 30 minutes after application. Group B (Clobetasol Group): Participants received topical clobetasol propionate 0.05% ointment, applied to OLP lesions in an identical manner, three times daily for 8 weeks. All patients in both groups received standardized oral hygiene instructions and antifungal prophylaxis with nystatin oral suspension 100,000 IU/mL (Group B only, as per standard protocol).

Outcome Assessment: Patients were evaluated at baseline (Week 0), Week 2, Week 4, and Week 8. The primary outcome was reduction in lesion severity measured by the Thongprasom sign scoring system (Score 0: no lesion; Score 1: mild white striae only; Score 2: white striae with erythematous area <1 cm^2; Score 3: white striae with erythematous area ≥1 cm^2; Score 4: white striae with erosive area <1 cm^2; Score 5: white striae with erosive area ≥1 cm^2). Secondary outcomes included subjective pain assessment using a 10-point Visual Analogue Scale (VAS) and documentation of adverse effects at each visit. All clinical assessments were performed by a calibrated examiner blinded to the treatment allocation.

Statistical Analysis: Data were analysed using SPSS version 21.0 (IBM Corp., Armonk, NY). The Shapiro-Wilk test assessed data normality. Within-group and between-group comparisons were performed using the Wilcoxon signed-rank test and Mann-Whitney U test, respectively. Categorical variables were analysed using the Chi-square or Fisher's exact test. Statistical significance was set at $p<0.05$, and results are expressed as mean ± standard deviation (SD).

Results

A total of 84 patients completed the study (42 per group). No dropouts occurred. The mean age in Group A was 44.3 ± 9.7 years and in Group B was 45.1 ± 10.2 years. There was no statistically significant difference between the groups with respect to age, gender distribution, disease duration, clinical subtype, or smoking status at baseline (all p-values >0.05), confirming successful randomization and homogeneity of groups (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Variable	Coconut Cream Group (n=42)	Clobetasol Group (n=42)
Age (years), Mean ± SD	44.3 ± 9.7	45.1 ± 10.2
Gender (Male/Female)	16 / 26	15 / 27
Duration of OLP (months), Mean ± SD	14.6 ± 6.3	13.9 ± 5.8
Clinical Form: Reticular (%)	15 (35.7%)	14 (33.3%)
Clinical Form: Erosive (%)	18 (42.8%)	19 (45.2%)
Clinical Form: Atrophic (%)	9 (21.4%)	9 (21.4%)
Smokers (%)	6 (14.3%)	7 (16.7%)
p-value (between groups)	>0.05 (NS)	>0.05 (NS)

The age distribution of participants in both groups is illustrated in Figure 1. The greatest representation was observed in the 41–50 year age group, with 12 patients in Group A and 13 patients in Group B, consistent with the known epidemiological predilection of OLP for middle-aged adults. The distribution pattern was similar between the two groups, further confirming baseline comparability.

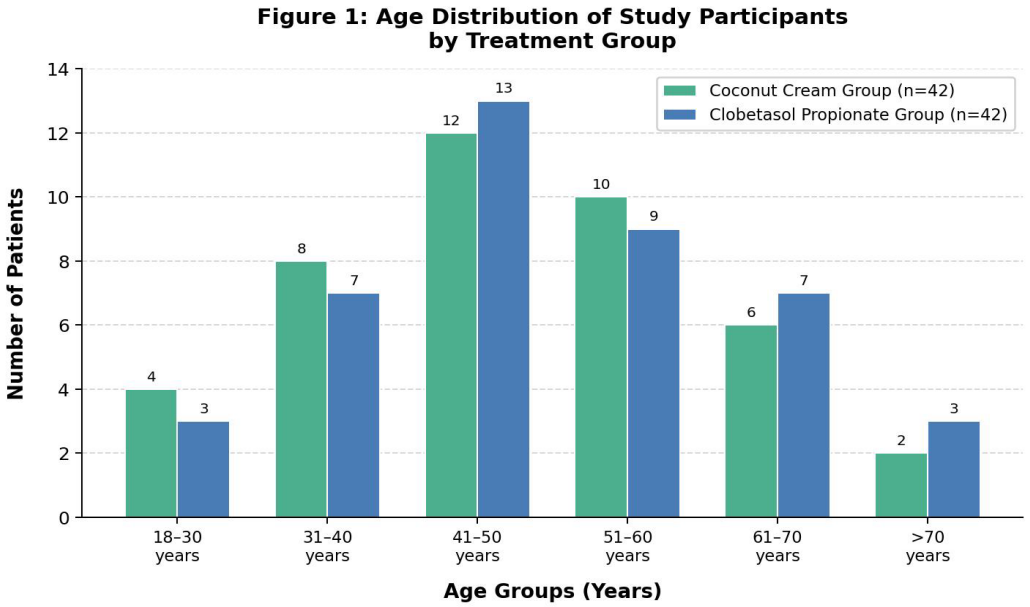

Figure 1: Age Distribution of Study Participants by Treatment Group

Both groups showed a progressive and statistically significant reduction in Thongprasom sign scores from baseline to Week 8. At eight weeks, the mean score reduction was 69.5% in Group B (clobetasol) versus 60.5% in Group A (coconut cream), with a statistically significant between-group difference (p=0.031). Intergroup differences became apparent from Week 2 onwards (Table 2).

The Thongprasom score results indicate that while both agents produced meaningful clinical improvement in OLP lesion severity, clobetasol propionate demonstrated a statistically greater reduction at all post-baseline time points. Nevertheless, the coconut cream group achieved more than a 60% mean improvement in clinical sign score by Week 8, which represents a clinically relevant therapeutic response. The progressive within-group improvement in both arms was highly significant ($p<0.001$, Wilcoxon signed-rank test), suggesting a robust therapeutic effect for both agents over the eight-week treatment period. Subjective pain scores were significantly reduced in both groups over the treatment period. The clobetasol group achieved a mean VAS reduction of 79.5% compared to 70.6% in the coconut cream group at Week 8, with a statistically significant intergroup difference (p=0.018). The most notable rate of pain reduction was observed between baseline and Week 4 in both groups (Table 3).

Table 2: Comparison of Thongprasom Sign Scores Between Groups Across Assessment Points

Evaluation Point	Coconut Cream (Mean ± SD)	Clobetasol (Mean ± SD)	Difference (Mean)	p-value
Baseline (Week 0)	3.62 ± 0.74	3.57 ± 0.81	0.05	0.784 (NS)
Week 2	2.91 ± 0.63	2.64 ± 0.58	0.27	0.048*
Week 4	2.14 ± 0.71	1.76 ± 0.64	0.38	0.016*
Week 8	1.43 ± 0.59	1.09 ± 0.52	0.34	0.009**
% Reduction from Baseline	60.5%	69.5%	9.0%	0.031*

Table 3: Comparison of Visual Analogue Scale (VAS) Pain Scores Between Groups Across Assessment Points

Evaluation Point	Coconut Cream (Mean ± SD)	Clobetasol (Mean ± SD)	Difference (Mean)	p-value
Baseline (Week 0)	6.42 ± 1.23	6.38 ± 1.17	0.04	0.876 (NS)
Week 2	4.78 ± 1.11	4.22 ± 0.98	0.56	0.021*
Week 4	3.12 ± 0.94	2.47 ± 0.87	0.65	0.002**
Week 8	1.89 ± 0.76	1.31 ± 0.69	0.58	0.001**
% Reduction from Baseline	70.6%	79.5%	8.9%	0.018*

The VAS pain data confirm that topical application of coconut cream produced clinically meaningful analgesia in OLP patients, though the degree of relief was modestly inferior to that achieved with clobetasol propionate. The within-group changes in both arms were statistically significant at all time points ($p<0.01$). Importantly, no patient in Group A reported worsening of pain during the treatment

period, whereas two patients in Group B experienced transient exacerbation of mucosal burning during the initial two weeks, likely attributable to the vehicle formulation.

The adverse effect profile differed markedly between the two groups. A total of 14 adverse events were recorded in Group B (clobetasol) compared to only 4 in Group A (coconut cream), representing an overall adverse effect rate of 33.3% versus 9.5%, respectively (p=0.012). Oral candidiasis occurred exclusively in Group B (n=5, 11.9%), while mucosal atrophy was documented in 3 patients (7.1%) in Group B. No participant in Group A developed either of these corticosteroid-related complications (Table 4). No serious adverse events, allergic reactions, or systemic toxicity were reported in either group.

The adverse effect data unequivocally demonstrate the superior safety profile of topical coconut cream relative to clobetasol propionate ointment in this study population. The absence of oral candidiasis and mucosal atrophy in the coconut cream group is a particularly clinically significant finding, as these are among the most common and clinically troublesome complications of prolonged topical corticosteroid use in oral mucosal disease. The relatively low rate of adverse events in Group A affirms the tolerability and biocompatibility of coconut cream as a topical oral medicament.

Table 4: Adverse Effects Profile Across Treatment Groups

Adverse Effect	Coconut Cream Group n (%)	Clobetasol Group n (%)
Oral Candidiasis	0 (0.0%)	5 (11.9%)
Burning Sensation (mild)	3 (7.1%)	4 (9.5%)
Taste Alteration	1 (2.4%)	2 (4.8%)
Mucosal Atrophy	0 (0.0%)	3 (7.1%)
Allergic Reaction	0 (0.0%)	0 (0.0%)
Total Adverse Events	**4 (9.5%)**	**14 (33.3%)**
p-value	–	0.012*

Discussion

This double-blinded randomized controlled trial compared the therapeutic efficacy and safety of topical coconut cream against clobetasol propionate 0.05% ointment in patients with histopathologically confirmed symptomatic OLP over an eight-week treatment period. The results confirm that both agents are clinically effective in reducing lesion severity and pain in OLP; however, clobetasol propionate demonstrates statistically superior reductions in both Thongprasom sign scores and VAS pain measures. Conversely, the adverse effect profile of coconut cream was significantly more favourable, with a near-fourfold lower rate of adverse events compared to the corticosteroid arm.

The superior efficacy of clobetasol propionate observed in this trial is consistent with the existing body of literature. A landmark randomized controlled trial by Carbone et al. demonstrated substantial improvement in both symptoms and clinical signs of OLP following topical clobetasol use, with significant reductions in lesion area and pain intensity. [9] Similarly, Thongprasom et al. reported that topical corticosteroids, particularly super-potent agents, produced the most clinically meaningful improvement in erosive and atrophic OLP subtypes compared to placebo. [10] Comparable outcomes were reported by Ribeiro et al. in a randomized trial comparing laser phototherapy with topical clobetasol 0.05%, in which clobetasol achieved statistically significant sign and symptom reduction at Day 30 despite eventual relapse at follow-up visits. [11] The mechanistic basis for this efficacy lies in clobetasol's potent glucocorticoid receptor-mediated suppression of cytokine gene transcription, including TNF-α, IL-1β, and IL-6, which are pivotal mediators of the pathological T-lymphocyte infiltration in OLP. [12,13,]

Despite its clinical superiority, clobetasol propionate is associated with well-characterised adverse effects that limit its utility in long-term management. In the present study, oral candidiasis occurred in 11.9% of patients in the clobetasol group, while 7.1% developed mucosal atrophy. These findings closely align with those reported by Soria et al. in a randomized trial comparing clobetasol formulations, where secondary candidal infection was among the most common complications necessitating antifungal prophylaxis. [14,15] Mucosal atrophy, another recognised complication of topical super-potent steroids, reflects disruption of keratinocyte proliferation and collagen synthesis resulting from prolonged glucocorticoid exposure. Such complications not only impose additional therapeutic burden on the patient but also raise concerns regarding the safety of sustained use in a condition with a recognized potential for malignant transformation, estimated at approximately 1% over time. [16,17,18]

The findings from the coconut cream arm of this trial are both novel and encouraging. The 60.5% reduction in Thongprasom sign score and 70.6% reduction in VAS pain score, while statistically inferior to clobetasol, represent clinically meaningful outcomes. The therapeutic activity of

coconut cream in this context can be attributed to the pharmacologically active medium-chain fatty acid constituents, particularly lauric acid, which accounts for approximately 49–52% of the total fatty acid composition. Lauric acid, upon absorption, is converted to monolaurin, a monoglyceride with well-documented immunomodulatory, anti-inflammatory, and antimicrobial properties. [17] In vitro studies have demonstrated that lauric acid inhibits the LPS-stimulated production of pro-inflammatory cytokines including IL-1β and TNF-α in macrophages, through suppression of the NF-κB signalling pathway, a mechanism that parallels, albeit less potently, the anti-inflammatory actions of corticosteroids. [12]

From a wound-healing perspective, the topical application of virgin coconut oil has been shown to accelerate dermal and mucosal wound healing through enhancement of neovascularization, proliferation of fibroblasts, and synthesis of pepsin-soluble collagen. [13] These properties are mechanistically relevant to OLP management, where regeneration of the disrupted basement membrane and restoration of the epithelial barrier are central to clinical improvement. Furthermore, the polyphenolic antioxidants present in coconut preparations, including ferulic acid and p-coumaric acid, mitigate reactive oxygen species-mediated oxidative damage to keratinocytes, which is increasingly recognised as a contributory mechanism in OLP pathogenesis and progression. [7] The absence of oral candidiasis in the coconut cream group is particularly noteworthy and may be attributed to the intrinsic antifungal properties of lauric acid and caprylic acid against Candida albicans, providing a therapeutic advantage over corticosteroids that suppress local mucosal immunity and predispose to fungal colonisation. [19,20]

From a clinical translational standpoint, the findings of this trial have several practical implications. For patients with mild-to-moderate OLP presentations, particularly those who are elderly, immunocompromised, or intolerant to corticosteroids, topical coconut cream may offer a safe and effective primary therapeutic option. In patients with erosive or severe OLP where rapid pain control is paramount, clobetasol propionate may still be preferred as initial therapy, with coconut cream potentially playing a role in maintenance therapy or as an adjunct to reduce corticosteroid exposure. Future studies should explore the effects of prolonged treatment beyond eight weeks and evaluate long-term relapse rates, quality-of-life outcomes, and histological changes in response to coconut cream therapy. The development of standardised pharmaceutical coconut cream preparations with defined fatty acid concentrations will be essential for reproducibility and regulatory consideration of this agent as an approved treatment for OLP.

Several limitations of this study warrant acknowledgment. First, the observation period was restricted to eight weeks, which may not adequately capture the relapsing nature of OLP or long-term remission rates. Second, the standardised topical coconut cream used was a freshly prepared formulation and may not be commercially reproducible without pharmaceutical standardization. Third, the study was conducted at a single centre, potentially limiting generalizability to broader populations with different dietary backgrounds or comorbidities. Finally, histological assessment of tissue changes following treatment was not performed, which would have provided additional mechanistic insights into the biological effects of coconut cream on the oral mucosal epithelium.

Conclusion

This randomized controlled trial demonstrates that while clobetasol propionate 0.05% ointment achieves superior short-term clinical efficacy in reducing OLP lesion severity and pain, topical coconut cream produces a clinically meaningful therapeutic response with a significantly safer adverse effect profile. The absence of corticosteroid-associated complications such as oral candidiasis and mucosal atrophy in the coconut cream group underscores its potential as a natural, well-tolerated alternative in OLP management. Coconut cream may be considered a first-line option for mild-to-moderate OLP and in patients where prolonged corticosteroid use is contraindicated. Larger multi-centre trials with extended follow-up periods are warranted to validate these findings.

References

1. Wilson E. On lichen planus. J Cutan Med. 1869; 3:117–32.
2. Scully C, Carrozzo M. Oral mucosal disease: lichen planus. Br J Oral Maxillofac Surg. 2008;46(1):15–21.
3. Gupta S, Jawanda MK. Oral lichen planus: an update on etiology, pathogenesis, clinical presentation, diagnosis and management. Indian J Dermatol. 2015;60(3):222–9. [PMID: 26120146]
4. Ismail SB, Kumar SK, Zain RB. Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation. J Oral Sci. 2007;49(2):89–106. [PMID: 17634721]
5. Roopashree MR, Gondhalekar RV, Shashikanth MC, George J, Thippeswamy SH, Shukla A. Pathogenesis of oral lichen planus – a review. J Oral Pathol Med. 2010;39(10):729–34. [PMID: 20923445]

6. Kurago ZB. Etiology and pathogenesis of oral lichen planus: an overview. Oral Surg Oral Med Oral Pathol Oral Radiol. 2016;122(1):72–80. [PMID: 27260276]
7. Nogueira PA, Carneiro S, Ramos-e-Silva M. Oral lichen planus: an update on its pathogenesis. Int J Dermatol. 2015;54(9):1005–10. [PMID: 26147778]
8. Carrozzo M, Thorpe R. Oral lichen planus: a review. Minerva Stomatol. 2009;58(10):519–37.
9. Carbone M, Goss E, Carrozzo M, Castellano S, Conrotto D, Broccoletti R, et al. Systemic and topical corticosteroid treatment of oral lichen planus: a comparative study with long-term follow-up. J Oral Pathol Med. 2003;32(6):323–9.
10. Thongprasom K, Carrozzo M, Furness S, Lodi G. Interventions for treating oral lichen planus. Cochrane Database Syst Rev. 2011;(7):CD001168.
11. Intahphuak S, Khonsung P, Panthong A. Anti-inflammatory, analgesic, and antipyretic activities of virgin coconut oil. Pharm Biol. 2010;48(2):151–7. [PMID: 20645831]
12. Nevin KG, Rajamohan T. Effect of topical application of virgin coconut oil on skin components and antioxidant status during dermal wound healing in young rats. Skin Pharmacol Physiol. 2010;23(6):290–7. [PMID: 20523108]
13. Ahmad HI, Li H, Al-Rawi SS, Majid ASA, Al-Habib OA, Xia X, et al. Angiogenic and wound healing potency of fermented virgin coconut oil: in vitro and in vivo studies. Am J Transl Res. 2017;9(12):5235–44.
14. Ribeiro AS, Tessari MB, Menezes SA, Menezes TO, Neto AS, Figueredo CM. Efficacy of laser phototherapy in comparison to topical clobetasol for the treatment of oral lichen planus: a randomized controlled trial. Photomed Laser Surg. 2014;32(8):447–55. [PMID: 24887747]
15. Soria A, Agbo-Godeau S, Taïeb A, Frances C. Treatment of refractory oral erosive lichen planus with topical rapamycin: 7 cases. Dermatology. 2009;218(1):22–5.
16. Thongprasom K, Luangjarmekorn L, Sererat T, Taweesap W. Relative efficacy of fluocinolone acetonide compared with triamcinolone acetonide in treatment of oral lichen planus. J Oral Pathol Med. 1992;21(10):456–8.
17. Lodi G, Carrozzo M, Furness S, Thongprasom K. Interventions for treating oral lichen planus: a systematic review. Br J Dermatol. 2012;166(5):938–47.
18. Eisen D, Carrozzo M, Bagan Sebastian JV, Thongprasom K. Oral lichen planus: clinical features and management. Oral Dis. 2005;11(6):338–49.
19. Al-Hashimi I, Schifter M, Lockhart PB, Wray D, Brennan M, Migliorati CA, et al. Oral lichen planus and oral lichenoid lesions: diagnostic and therapeutic considerations. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol. 2007;103(Suppl):S25.e1–12.
20. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol. 2009;45(4–5):309–16.