

CLINICAL REVIEW

The Importance of Recognition and Management of Oral Lichen Planus: Navigating Epidemiology, Pathogenesis, Cancer Risk, and Advances in Management

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ABSTRACT

Background: Oral lichen planus (OLP) is a chronic inflammatory disorder classified as an oral potentially malignant disorder (OPMD) due to its association with an increased prevalence and a risk of progressing to oral squamous cell carcinoma (OSCC) of up to 5%.

Methods: We conducted a comprehensive literature review to update the epidemiology, etiology, pathogenesis, diagnosis, and management of OLP using PubMed and Web of Science databases up to April 2025.

Results: OLP is a T-cell-mediated disorder, with CD4+ and CD8+ T cells playing a key role in its pathogenesis. Its multifactorial nature involves genetics, infections, immune dysregulation, allergens, stress, autoimmune diseases, and medications. Treatment options include topical corticosteroids as the first-line therapy, along with systemic medications and emerging therapies that show promise in management.

Conclusions: Regular monitoring and early intervention are crucial due to OLP's malignant potential. Further research is needed to refine treatments and assess long-term efficacy.

1 | Introduction

Lichen planus is a chronic, immune-mediated inflammatory disease that can affect skin, hair, nails, eyes, and mucous membranes. The most typical clinical feature of this disease is white hyperkeratotic striae [1, 2]. The term “lichen planus” (LP) was first introduced by the British dermatologist Erasmus Wilson, recognized since 1866 [2–4].

The oral mucosa is the most frequently affected site by lichen planus, known as oral lichen planus (OLP), representing the most common immune-mediated oral mucosal condition. It occurs in 0.5%–2% of the population, predominantly affecting

middle-aged females, and is rarely observed in children [1, 4]. OLP is characterized by remissions and flares, with various clinical presentations, including atrophic, erosive, bullous, plaque-like, and reticular forms. The gingiva, lateral and ventral tongue, and buccal mucosa are frequently affected by the reticular type, which manifests as lacy white striae. Red patches and ulcerations are the presentation of the atrophic and erosive types, respectively. While the rare bullous form of OLP appears with bullae or vesicles, resembling other oral immunobullous illnesses, plaque-form OLP presents as white plaques [5]. OLP is characterized by its potential to progress to oral squamous cell carcinoma (OSCC), which has led to its classification as an oral potentially malignant disorder (OPMD) after years of debate [6].

The risk of malignant transformation to OSCC is estimated to be between 1% and 2% [4], with the World Health Organization (WHO) identifying several factors that elevate this risk, including female gender, the erosive form of OLP, and tongue involvement [6]. Therefore, the WHO recommends lifelong follow-up for patients with OLP to facilitate early cancer detection and identify those at greater risk. Various potential triggers of OLP have been identified, including gene polymorphisms, viral infections, systemic diseases, psychological stress, oral lichenoid hypersensitivity to dental restorative materials, vaccinations, and certain medications [2, 4].

The purpose of this review is to provide an overview of the current epidemiology of OLP, indications for management, and to provide an update on advances in management.

2 | Methods

Published data from this review were identified by a search and selection database of PubMed, Web of Science up to April 2025. In addition, gray literature was also screened on Google Scholar; and results obtained from this platform were cross-verified with other sources before inclusion to ensure relevance and quality. The search criteria were set as “oral lichen planus”, “oral lichen planus pathogenesis”, “oral lichen planus diagnosis”, “oral potentially malignant disorder”, “oral squamous cell carcinoma”, and “management of oral lichen planus”. Regarding the initial list of studies, the reference lists from relevant articles, reviews, and books on the subject were obtained. Furthermore, papers that cite the initial list of studies were also reviewed, which allowed us to include more recent studies that were based on prior published reports. A total of 137 studies were included, which were grouped based on the following topics: epidemiology, etiopathogenesis, clinical features, diagnosis, and treatment. Articles including non-English publications, case reports with fewer than five cases, studies with incomplete data or poor methodological quality, and those unrelated to OLP were excluded.

3 | OLP Epidemiology

The global prevalence of OLP is estimated to be 1.01%, with higher rates in South America at 1.74%, Africa at 1.43%, Europe at 1.32%, and lower in Asia at 0.83% and North America at 0.47% [7]. Among countries, Brazil had the highest prevalence at 6.04% [8], whereas India recorded the lowest prevalence at 0.49%. OLP risk increases significantly after the age of 40 [7], and women are more frequently affected than men [9]. Recent U.S. data show that LP prevalence increases with age, reaching 0.33% in those aged 70–79, and is 1.77 times more common in women than men [10]. Detection of OLP also varies based on clinician expertise, as oral medicine and pathology specialists reported a prevalence of 1.80% compared to dentists at 0.61% and dermatologists at 0.33% [7].

4 | OLP Pathogenesis

The immune-mediated response, driven by T cells primarily, defines the intricate pathophysiology of OLP. Antigen presentation

by keratinocytes stimulates CD4+ helper T cells as well as CD8+ cytotoxic T cells, which in turn produce cytokines including IL-2, IFN- γ , and TNF- α which further induce cytotoxic T-cell proliferation and cause apoptosis of basal keratinocytes, producing the characteristic epithelial changes in OLP [11–14]. Indeed, activated CD4+ and CD8+ T cells, along with a high number of Langerhans cells expressing MHC class II and hence participating in further T-cell activation, are the main cells represented in the inflammatory infiltrate of OLP lesions [13]. Additionally, Th17 T cells and other cell types produce IL-17, which is essential for eliminating bacterial and fungal infections; it can also result in inflammation and tissue damage. As a result, it could be the cause of or a result of OLP surface erosions and ulcerations [12]. Moreover, the Th1/Th2 cytokine imbalance, with elevated Th1 cytokines, enhances CD8+ cytotoxic T-cell activity, driving keratinocyte apoptosis [15]. Cytokine polymorphisms may influence whether lesions are confined to the oral cavity (IFN- γ associated) or in conjunction with skin lesions (TNF- α associated). Furthermore, dendritic cells, particularly plasmacytoid dendritic cells, produce IFN- α which can enhance cytotoxicity, activating natural killer/T cytotoxic cells or FasL-mediated apoptosis [11, 16]. Notably, IFN- γ exerts many of its effects through the JAK/STAT signaling pathway; binding to its receptor activates JAK1 and JAK2, which phosphorylate STAT proteins that translocate to the nucleus to promote transcription of genes like CXCL10. CXCL10 recruits T cells expressing CXCR-3 to the lesion site, amplifying the inflammatory response in a self-sustaining loop. This IFN- γ /CXCL10 pathway is considered a key mediator in persistent inflammation in LP [17]. Chronic OLP is exacerbated by the activation of NF- κ B and the inhibition of TGF- β signaling pathways, leading to keratinocyte hyperproliferation and epithelial hyperplasia, resulting in white lesion development [18]. OLP is ultimately characterized by tissue damage and prolonged inflammation due to a complex interaction between immunological dysregulation, overproduction of cytokines, and genetic factors (Figure 1).

5 | OLP Etiology

OLP is a multifactorial condition, with genetic predisposition as a significant factor in its development. Specific genes, including HLA-B8, -B51, -Bw57, and -Bw61, are commonly expressed in affected individuals [19]. Among the 132 genes implicated in OLP, a few key “leader genes,” including JUN, EGFR, FOS, IL2, and ITGB4, have been identified as central to pathogenesis [20]. Viral infections also play a role, with several studies highlighting potential associations between OLP and infections like varicella-zoster (VZV), which is linked to linear LP and zosteriform LP [21]. Additionally, Epstein–Barr virus (EBV), a common herpes virus found in over 90% of the population, has been related to OLP [22], with studies showing a 35% positivity rate in OLP patients compared to none in controls [23]. Raybaud et al. indicated that EBV prevalence in OLP can be as high as 74%, particularly in erosive forms [24], while Vieira Rda et al. found EBV in 75% of saliva samples from OLP patients and in 64.7% of controls, but no significant differences in clinical variations based on sample type [25]. Thirty-three studies on human papillomavirus (HPV) detection in OLP reported widely varying rates, with Kato et al. finding a prevalence of 41.5% among OLP patients, mainly for high-risk genotypes 16 and 18 [26].

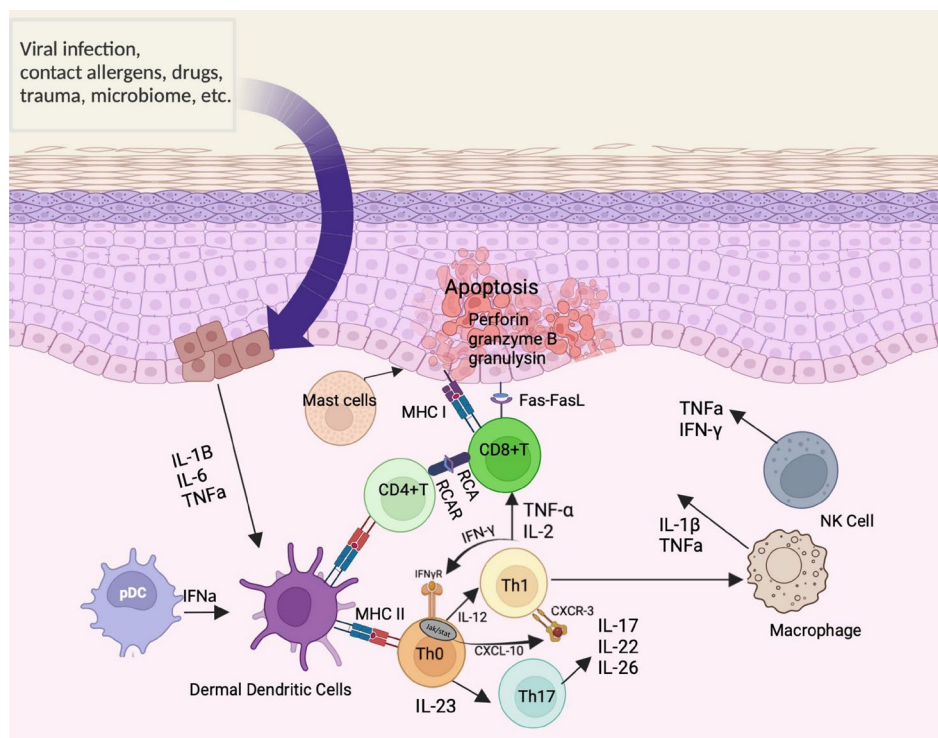


FIGURE 1 | Immunopathogenesis hypothesis of OLP. [Color figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com/doi/10.1002/hed.70012)]

A meta-analysis revealed that OLP patients have about seven times the odds of HPV infection compared to controls, with strong associations for HPV-16 and HPV-18, and indicated a higher prevalence of HPV in biopsies than in saliva samples [27]. Human herpesvirus 7 (HHV-7) and human herpesvirus 6 (HHV-6) have also been detected in infiltrating plasmacytoid dendritic cells within lesional skin biopsies [28, 29]. Another well-documented association is with hepatitis C virus (HCV), as LP patients are up to 13 times more likely to have HCV [30–32]. The exact mechanism is unclear but likely involves dysregulation, with CD8+ T lymphocytes specific to the virus found in LP lesions [33]. The recent COVID-19 pandemic has added another layer of complexity, with studies suggesting that COVID-19 infection or vaccination may trigger immune dysregulation, involving T-cell activation and cytokine production, potentially leading to OLP development [34–36]. A prospective observational study found that 12 out of 74 COVID-19 patients (16.2%) developed OLP. However, the lack of longitudinal follow-up and observational nature of the study underscores the need for cautious interpretation of these findings [37]. A retrospective cohort study using the TriNetX database compared 217 863 vaccinated patients to an equal number of unvaccinated patients. They found that 0.067% of vaccinated patients developed OLP within 6 days, compared to 0.027% in the unvaccinated control group. Although the risk was higher among vaccinated individuals, the overall incidence remains low, suggesting that these lesions are rare adverse events [38]. Liver health also influences OLP, with cytochrome P450 enzymes involved in metabolizing drugs that can induce or worsen the condition. Several retrospective studies proved an association between elevated serum aminotransferases and OLP lesions [39]. Furthermore, oral microbiota shifts are associated with OLP, potentially by triggering immune responses. Significant dysbiosis was found in OLP patients' saliva, with elevated levels of bacteria (*Solobacterium*,

Fusobacterium, *Porphyromonas*, *Prevotella*), fungi (*Candida*, *Aspergillus*, *Alternaria*), and viruses, along with reduced levels of *Streptococci*, *Actinobacteria*, and *Firmicutes* compared to healthy controls [40, 41]. In addition to microbial factors, OLP can be triggered by contact allergens like dental materials, including amalgam, composite resins, nickel, and gold [42, 43]. Studies have shown that stress, whether physical or psychological, can suppress the immune response through the release of catecholamines and cortisol, which in turn activate inflammatory cytokines such as TNF- α , IL-1- α , IL-6, and IL-8 [2, 44, 45]. Additionally, anxiety and depression are more common in OLP patients compared to controls, and exacerbations often coincide with anxiety episodes [2, 12]. Moreover, OLP patients are more likely to have comorbidities, including autoimmune diseases such as Sjögren syndrome, systemic lupus erythematosus (SLE), and ulcerative colitis [46–48]. A positive correlation with alopecia areata, vitiligo, and thyroid diseases (especially Hashimoto's thyroiditis) has also been confirmed [2, 49–51]. Additionally, LP is associated with a higher prevalence of obesity, dyslipidemia, diabetes, and metabolic syndrome due to chronic inflammation [52–55]. Oral Lichenoid Reactions (OLRs) are associated with various external triggers, including contact hypersensitivity to dental restorative materials (e.g., amalgam, gold, nickel), adverse reactions to certain medications, and systemic conditions such as graft-versus-host disease [56]. While OLP and OLR are clinically and histologically similar, certain distinguishing features can aid in diagnosis. Clinically, OLR lesions are often asymmetrical and localize adjacent to the offending agent, while OLP lesions are typically bilateral and symmetrical.

Histopathologically, OLRs tend to show more eosinophils, plasma cells, granulocytes, and a higher number of degranulated mast cells, while OLP is characterized by “saw-tooth” rete pegs, atrophic epithelium, and more intact subepithelial mast

cells [56–58]. These subtle differences combined with comprehensive clinical history and examination remain essential for diagnosis (Table 1).

6 | Why Treating OLP Is Important?

Treating OLP is crucial for improving patients' quality of life, as the condition can cause significant discomfort and functional impairment. OLP commonly affects the tongue, lips, gingiva, and buccal mucosa, presenting as fine white lines forming a lace-like pattern known as Wickham's striae, a pathognomonic characteristic of OLP [59].

Symptoms vary in intensity, including oral discomfort, roughness, and burning sensations, especially in erosive forms [60]. This persistent burning or tingling is often aggravated by factors such as sharp cusps, ill-fitting dentures, poor oral hygiene, chronic cheek biting, and consumption of spicy, acidic, or hot foods [3, 4, 61]. Xerostomia associated with OLP may further contribute to discomfort, dysphagia, halitosis, and increased caries risk. Additionally, lichenoid gingival inflammation can complicate periodontal status and accelerate attachment loss [4, 60]. Altered taste (dysgeusia), often described as a metallic or bitter sensation, can also affect food enjoyment [60]. Plaque and

calculus buildup can aggravate OLP, increasing the risk of gingival recession and periodontal disease progression [62]. Post-inflammatory pigmentation may present as diffuse brown or black patches [63]. In severe cases, extensive lesions can impair speech, and the chronic discomfort associated with OLP can negatively impact daily activities, contributing to psychological distress, including anxiety and depression [60].

Beyond symptom management, OLP requires careful monitoring due to its potential for malignant transformation. In 2005, the WHO classified OLP as an OPMD due to its potential to progress to OSCC, with malignancy ratios ranging from 0.44% to 2.28% [6]. However, a recent meta-analysis updated the malignancy ratio to 1.43%, highlighting that OLP cases with epithelial dysplasia had a significantly higher rate of 5.13% [64]. This progression involves genetic and cellular changes, including reduced levels of succinate dehydrogenase enzyme B (SDHB) and DNA damage caused by inducible nitric oxide synthase (iNOS), leading to the accumulation and mutation of the p53 protein, a key marker observed in both OLP and OSCC cases [65, 66]. Patients with OLP and HCV have a 3.16 times higher risk of malignancy, with additional risk factors including female gender, older age, erosive forms, smoking, and alcohol consumption [67, 68]. The mean interval between OLP diagnosis and cancer diagnosis varies significantly, ranging from 20.8 months to

TABLE 1 | Triggering and exacerbating factors of OLP/OLR.

Medications	Viruses	Nutrition Deficiencies	Allergy
Beta-blockers	- Varicella-Zoster Virus	- Vitamin D	- Food
ACE inhibitors	- Epstein-Barr Virus	- Vitamin B12	- Metals
Diuretics	- Human Papilloma Virus	- Folic Acid	- Dental Amalgam
Antihypertensives	- Human Herpesvirus 6	- Iron	- Composite Resins
- Hydrochlorothiazide	- Human herpesvirus 7	- Vitamin A, C, and E	- Nickel
NSAIDs	- Hepatitis C Virus	Vaccines	- Gold
-	- Hepatitis B Virus	Covid-19 Vaccines	Trauma
Antibiotics	- Coronavirus SARS-CoV-2	- Tozinameran	- Rough dental surfaces
- Tetracycline	Systemic Diseases	- Spikevax	- Severe Attrition
- Sulfamethoxazole	- Hypertension	- Vaxzevria	- Decreased Vertical
Anticonvulsants	- Diabetes	- Jcovden	Dimension
- Carbamazepine	- Dyslipidemia	Hepatitis A and B	Lifestyle
Antifungals	- Thyroid disease	Vaccines	- Obesity
- Ketoconazole	- Liver dysfunction	- HAVRIX	- Stress
Antimalarials	- Graft versus host disease	- ENGERIX-B	- Insomnia
- Chloroquine	Autoimmune Diseases	Influenza Vaccines	- Betel Quid Chewing
Antiretrovirals	- Sjogren syndrome	Herpes Zoster Vaccines	
- Zidovudine	- Systemic lupus erythematosus	Covid vaccines	
Antituberculosis drugs	- Mixed connective tissue disorder	Oral Factors	
- Ethambutol	Myasthenia Gravis	- Oral Microbiome	
- Pyrazinamide	- Ulcerative Colitis	- Salivary Gland	
- Isoniazid	- Alopecia Areata	Hypofunction	
- Rifampicin	- Vitiligo	Genetic Predisposition	
Glycemic drugs	Bone Marrow Transplant		
- Tolbutamide	- Graft-versus-host disease		
- Glipizide			
- Insulin			
Statins			
- Pravastatin			
Chemotherapy			
- Immune check point inhibitors			

10.1 years, with the highest risk occurring between 3- and 6-year post-diagnosis [69]. Infectious factors, such as *Fusobacterium nucleatum* and *Candida albicans*, have been implicated in malignant transformation due to their potential production of carcinogenic compounds [70, 71].

Given these risks, regular long-term follow-up is crucial for early detection of OSCC. Follow-up intervals may range from every 2 months to annually, with annual assessments advised for reticular OLP. However, patients with dysplastic OLP should be examined more frequently, potentially every 2–3 months or if a change in symptoms is noted. If erosive changes are observed, additional biopsies may be required, and follow-up intervals should be reduced [72]. Biomarkers such as Podoplanin and ABCG2 have shown promise as risk indicators [4, 73]. Additionally, cell cycle regulators, intercellular adhesion proteins, and apoptotic biomarkers including p53, p63, and p73, serve as potential markers for the early detection of malignant transformation in OLP [74].

7 | OLP Treatment

7.1 | Corticosteroids

Corticosteroids, synthetic analogs of cortisol, are the gold standard for treating OLP due to their anti-inflammatory and immunosuppressive effects [75]. Topical glucocorticosteroids, such as triamcinolone acetonide, fluocinonide, betamethasone, and clobetasol propionate, are the first line of treatment for mild to moderate OLP lesions. High potency steroids are often required due to limited oral mucosal contact time, and if involving keratinized mucosa, such as gingivae [76]. These are typically applied as gels, creams, or rinses, as ointments are not suitable for mucosal surfaces. Topical therapies are preferred for their ability to provide local effects with minimal systemic absorption [77]. Beyond symptom management, recent research suggests that management using topical corticosteroid therapy may delay OLP's malignant transformation to OSCC by 4 years [78].

Budesonide, a high-potency topical steroid that can be compounded leading to minimize systemic effects, but prolonged use may still cause mucosal thinning, telangiectasia, and candidiasis [2, 3, 79], as Ku et al. found microbial composition changes after 4 weeks of 0.05% dexamethasone mouthwash use; potentially increasing the risk of fungal infections [80].

Systemic corticosteroids, such as prednisone and methylprednisolone, are reserved for severe, widespread, or recalcitrant cases of OLP that do not respond to topical treatments [81]. While effective, they pose risks such as adrenal suppression, iatrogenic Cushing's syndrome, hyperglycemia, osteoporosis, hypertension, and immunosuppression [77, 82]. Prolonged use (over 2 weeks) requires tapering to avoid adrenal suppression and potential adrenal crisis [3]. A thorough medical history is essential, as these drugs are contraindicated in patients with conditions such as stomach ulcers, diabetes, or hypertension [2, 83]. In such refractory or contraindicated cases, nonsteroidal agents like mycophenolate mofetil, sirolimus, or calcineurin inhibitors may be considered, though calcineurin inhibitors may carry a carcinogenic risk [84, 85]. Intralesional steroids like triamcinolone

acetonide can target localized ulcerations with fewer systemic effects but may cause pain and tissue atrophy [77].

7.2 | Immunosuppressants

Immunosuppressive drugs, including tacrolimus, dapsone, cyclosporine, azathioprine, and pimecrolimus, play a significant role in managing OLP by inhibiting T lymphocyte function and reducing inflammation [4, 77, 86]. Tacrolimus (0.1% ointment, twice daily for up to 4 weeks) is effective for erosive OLP but may cause transient burning, relapse after cessation, and is difficult to apply to mucosa [77, 87]. Pimecrolimus cream is more mucosa-friendly and easier to apply. Dapsone (50–150 mg/day for up to 6 months) has steroid-comparable efficacy but may cause headaches and hemolysis [4, 77]. Azathioprine offers immunosuppressive and anti-inflammatory benefits but carries risks of bone marrow suppression and liver dysfunction [88]. Cyclosporine, though useful for persistent OLP and available in compounded topical forms, is limited by systemic side effects (e.g., hypertension, gingival hyperplasia) and is ineffective as a mouthwash due to enzymatic deactivation [14]. Although effective, these immunosuppressants can cause side effects such as bad taste, nephrotoxicity, and high costs, requiring careful monitoring under specialist supervision [89].

7.3 | Retinoids

Since retinoid deficits can lead to keratinization of the oral mucosa, retinoid metabolites, which are derived from vitamin A, are used topically and systemically in the treatment of OLP. Retinoids are needed for the normal growth and proliferation of epithelial cells [90]. Common topical retinoids, including tretinoin, isotretinoin, fenretinide (0.1% gel), and tazarotene, have demonstrated efficacy in reducing reticular and plaque lesions while lowering the likelihood of recurrence post-treatment. In contrast, systemic retinoids, such as etretinate, isotretinoin, acitretin, temaroten, and tretinoin, are less frequently prescribed due to potential side effects including liver damage, teratogenicity, and cheilitis. However, temaroten can efficiently treat OLP with fewer adverse effects [4, 77, 91].

When gingival sites or palatal mucosa are involved, an enhanced effect can be achieved using occlusive vinyl appliances (medication trays) by increasing contact time and reducing the dilution of topical anti-inflammatory agents.

7.4 | Phototherapy

Photochemotherapy, particularly PUVA therapy with the photosensitizer psoralen, has proven effective in treating resistant OLP and skin LP [77]. UVA treatment without photosensitizers led to significant improvement in 87% of OLP patients; controlled studies have confirmed the efficacy of systemic psoralen for severe cases. However, side effects like nausea, dizziness, ocular symptoms, paresthesia, and oncogenic potential restrict its use to severe, unresponsive cases [92]. A study using ultraviolet B phototherapy with the TheraLight U 120-2 system in 14 patients showed promising results, with nine achieving full

remission and continued benefits for 29 weeks during maintenance treatment [93].

7.5 | Laser Treatment

Laser therapy has emerged as a promising option for treating OLP, particularly for its anti-inflammatory and antimicrobial properties [94]. CO₂ lasers, known for their thermal impact, effectively manage erosive and atrophic OLP by carbonizing and vaporizing tissue, closing blood vessels, and sterilizing incisions, making them useful in severe cases [86]. Nd: YAG lasers, with deep tissue penetration, not only provide significant pain relief but also improve clinical symptoms and control fungal strains like *Candida albicans* [95]. Additionally, Er: YAG lasers have proven effective in reducing microbial loads, such as *Porphyromonas gingivalis*, which are often elevated in OLP patients [96].

Low-level laser therapy (LLLT) now known as photobiomodulation (PBM), using varying wavelengths (400–1100 nm), has shown promise, particularly in enhancing cell metabolism without damaging tissues [77]. While some studies report PBM's ability to reduce pain and achieve lesion resolution, three studies showed significantly better improvement with topical corticosteroids compared to LLLT, with only one study demonstrating notable lesion improvement using LLLT [97, 98]. Despite these mixed results, PBM remains a viable alternative, especially for atrophic-erosive OLP, with potential analgesic, anti-inflammatory effects, and wound healing; though the lack of a standardized clinical protocol limits widespread adoption.

7.6 | Photodynamic Therapy

Photodynamic therapy (PDT) has an impact on OLP treatment as a new non-invasive method. It uses photosensitizers like methylene blue (MB) or toluidine blue (TB) and light to induce cell damage and lower inflammation [99]. Clinical studies indicate that PDT can alleviate pain and minimize lesions, demonstrating comparable effectiveness to topical corticosteroids and dexamethasone mouth rinse in treating OLP [100]. For example, a clinical study by Fatemeh et al. treated 11 OLP patients comparing TB-mediated PDT and topical corticosteroids. PDT was more effective up to day 21, but after 4 weeks of corticosteroid treatment on both sides, outcomes were similar. The authors suggest PDT can shorten treatment duration and serve as an adjuvant for resistant or recurrent OLP [101]. Furthermore, PDT appears to be a safe alternative or additional intervention for patients with comorbid conditions such as diabetes and hypertension [102]. However, despite its potential, the current evidence base is limited by considerable heterogeneity across studies. Variations in photosensitizer type (e.g., MB, 5-aminolevulinic acid, chlorin e6, TB), application duration, light source (diode lasers, xenon arc lamps, LEDs), wavelength (typically 600–660 nm), energy density, and treatment frequency impede standardization [99, 100, 102, 103]. Differences in lesion selection, outcome measures, and clinical type (e.g., erosive vs. reticular) further limit direct comparisons and may impact reproducibility. Additionally, while PDT

has known antimicrobial effects, its ability to disrupt deeper, more resistant biofilms remains unclear [103]. Recurrence rates are also poorly documented due to short follow-up periods, and subgroup analyses are often underpowered due to small sample sizes [100]. Therefore, while PDT demonstrates promising potential, its long-term efficacy, optimal treatment parameters, and impact on recurrence rates require further validation through larger, well-designed, and multi-center clinical trials.

7.7 | Hydroxychloroquine

Hydroxychloroquine (HCQ) has been investigated as a treatment option for OLP, particularly in refractory cases. A 1993 study reported a 90% response rate in 10 patients with erosive OLP treated with HCQ monotherapy [104]. Significant lesion reduction (63.3%) and pain relief were observed with a topical HCQ noisome gel in a placebo-controlled trial [105]. Other studies have reported more than 80% improvement [106], and moderate-to-marked improvement in 57% of patients [107]. A prospective study demonstrated a decrease in regulatory T cells after 2 weeks of HCQ use, suggesting an immunomodulatory mechanism [108], while a retrospective study also noted reduced pain and disease severity in recalcitrant cases [109]. Despite these promising results, most studies were limited by small sample sizes, retrospective designs, and lack of standardized outcomes. However, a recent multicenter randomized controlled trial (RCT) confirmed HCQ's safety and efficacy, demonstrating comparable outcomes to topical dexamethasone [110]. Nonetheless, HCQ is not the preferred treatment due to the risk of lichenoid reactions and potential side effects such as retinal damage and hyperpigmentation with long-term use [107, 111]. Although HCQ shows antibacterial, antiviral, and antifungal properties, including activity against *A. fumigatus* and *A. nidulans*, its impact on *Candida albicans* in the oral cavity remains unstudied [112].

7.8 | Platelet-Rich Fibrin

Platelet-Rich Plasma (PRP) and its advanced form, injectable Platelet-Rich Fibrin (i-PRF), have shown promising potential in managing OLP [113]. PRP is derived from autologous platelet concentrates (APCs) created by centrifuging venous blood, resulting in a fibrin clot that contains platelets and white blood cells [114]. These biocompatible products deliver high concentrations of growth factors and cytokines, promoting tissue repair and reducing inflammation, thereby providing effective pain relief for OLP symptoms [115]. Unlike corticosteroids, which can lead to adverse effects such as mucosal atrophy, PRP and i-PRF present safer alternatives with fewer side effects. Although the initial response to PRP may be slower compared to corticosteroids, its long-term outcomes are often comparable, making it suitable for patients unresponsive to conventional therapies [113, 116]. I-PRF, identified in 2001, offers advantages over traditional PRP, including faster wound healing, enhanced immune response, and the absence of anticoagulants [117]. Recent studies, including a meta-analysis by Gupta et al., have indicated that local injections of i-PRF can effectively reduce pain and lesion size, with

outcomes similar to established treatments like triamcinolone acetone. Specifically, findings from these studies highlight that i-PRF not only alleviates pain but also improves overall patient satisfaction due to its regenerative properties and minimal adverse effects [118].

7.9 | Curcumin

Curcumin, an extract from turmeric (*Curcuma longa*), is gaining attention as a potential adjunctive therapy for OLP due to its reported anti-inflammatory, antioxidant, and anti-carcinogenic properties. Historically used in traditional Indian medicine, some studies suggest curcumin may help alleviate symptoms in OLP patients, such as pain reduction and lesion improvement [119]. A systematic review described positive outcomes in trials using oral dosages of 300–600 mg three times daily for 8–12 weeks [120]. A study by Chainani-Wu et al. demonstrated that high-dose curcuminoids (6000 mg/day) can reduce OLP symptoms. However, the small sample size limited the statistical power, particularly in assessing inflammatory markers like CRP and IL-6 [121]. Additionally, in a 2015 study by Kia et al., the efficacy of 5% curcumin was compared with 0.1% triamcinolone, with complete pain resolution observed in 36% of the curcumin group and 32% of the triamcinolone group; however, the difference was not statistically significant [122]. Despite these findings, conflicting evidence exists as a study found no significant effect of curcumin on OLP [123], and other researchers have raised doubts about its overall efficacy in treating the condition [124].

7.10 | Hyaluronic Acid

Hyaluronic acid (HA) has shown potential as an effective treatment for OLP, particularly for short-term symptom relief. In a RCT of 50 patients with erosive OLP, those treated with 2% HA experienced significant reductions in pain (VAS score 1.48 vs. 7.76) and erythema (0.48 vs. 1.35) compared to the placebo group [125]. Furthermore, studies comparing HA gel to triamcinolone found both treatments equally effective in reducing pain and lesion size [126]. Though HA has demonstrated benefits in localized, short-term applications, its long-term effectiveness for OLP management remains uncertain.

7.11 | Vitamin D

Vitamin D is emerging as a valuable supplementary treatment in managing OLP due to its immunomodulatory and anti-inflammatory properties. Systematic reviews indicate that vitamin D supplementation, especially when combined with steroid therapies, significantly improves OLP symptoms compared to placebos or steroids alone. This is likely due to its regulation of keratinocyte proliferation and differentiation, which are crucial for mucosal health. Studies have found that patients receiving vitamin D, with or without psychological counseling, show significant improvements in symptoms [127]. A larger study involving 450 OLP patients also revealed that those supplemented with vitamin D experienced notable benefits [128].

7.12 | Periodontal Treatment

Oral care plays a crucial role in managing OLP, especially considering its intricate relationship with chronic periodontitis (CP), the leading cause of tooth loss in adults driven by bacterial infection and immune responses. Key risk factors for OLP progression, such as poor oral hygiene, stress, and dietary habits, emphasize the importance of periodontal health in OLP management [129]. Studies indicate a mutually reinforcing effect between OLP and CP, with significant improvements in OLP lesions observed following periodontal treatment [130]. Moreover, systemic risk factors like smoking, diabetes, and obesity can exacerbate both periodontal and OLP conditions. Smoking enhances inflammatory responses in OLP lesions and may increase cancer risks due to reduced macrophage expression [131]. Therefore, maintaining proper oral health is essential to reduce the severity of OLP lesions. Future attention to the oral microbiome is needed. Patients should avoid abrasive toothpaste and hard-bristle toothbrushes to minimize discomfort, and any restorations or appliances irritating should be adjusted. While asymptomatic patients with reticular OLP may not require treatment, erosive and atrophic forms necessitate intervention, and all forms of OLP require follow-up [85]. Addressing both the disease state and patient behaviors is vital for effective recovery.

7.13 | Surgery

OLP is an immune-mediated disorder, meaning that surgical excision and cryosurgery, which physically remove lesions without targeting the underlying immune processes, do not provide effective long-term management. As a result, lesions frequently recur after surgery. Cryosurgery often results in recurrence, with new lesions forming on healing wounds and more intense symptoms during the recovery process. Gingival grafts and free soft tissue grafts have also been utilized to treat localized erosive OLP lesions and may have a limited role in OLP-associated gingival recession [4].

The primary goal of OLP treatment is to reduce local inflammation, prevent recurrence, and reduce the potential for malignant transformation [132, 133]. Early detection of malignant progression is critical. Asymptomatic reticular lesions typically require only monitoring for changes. Management, therefore, should focus on treating atrophic and erosive lesions, alleviating symptoms, and minimizing the risk of cancer (Figure 2).

8 | Cost-Effectiveness and Accessibility Considerations

Optimal OLP therapies should be cost-efficient, effective, and have low toxicity. A systematic review compared efficacy and costs of available interventions used to treat OLP by analyzing 70 randomized clinical trials; it found that clobetasol remains the preferred option over fluocinonide, triamcinolone, and tacrolimus, due to its efficacy and lower cost (~\$4.12–4.54/g vs. tacrolimus at \$4.96/g) [134]. A pilot trial also demonstrated that both 0.1% tacrolimus and 0.05% clobetasol in orabase formulations

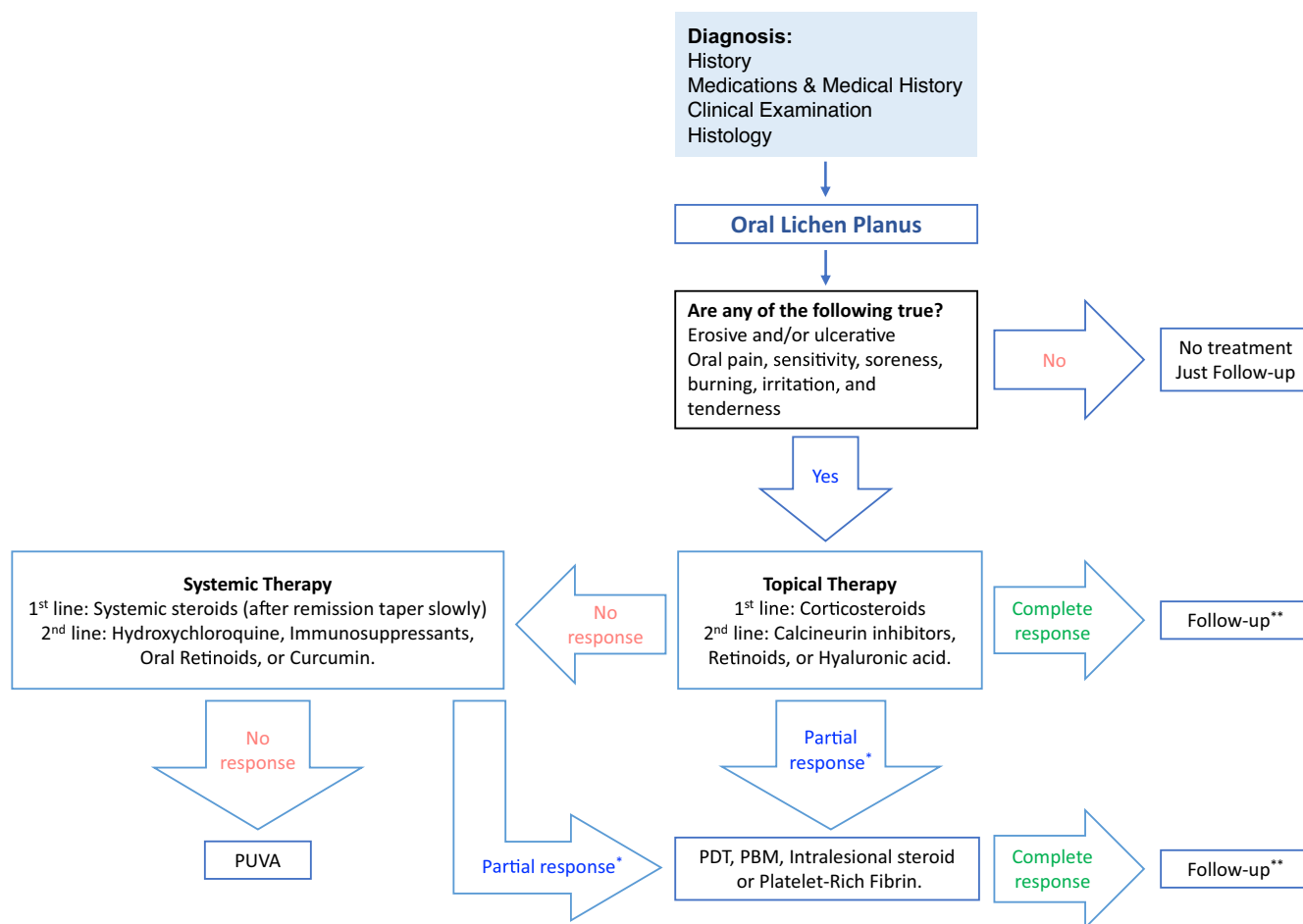


FIGURE 2 | Treating OLP recommendations. *A partial response referred to a 50%–79% decrease in active skin lesions from the baseline [132]. ** Roughly 50% of patients experience relapse, defined as the recurrence of active lesions after complete response, within 4–17 weeks [133]. [Color figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com)]

were effective in improving clinical symptoms in symptomatic OLP patients. However, due to its lower cost, clobetasol may be preferred as a first-line treatment, while tacrolimus may serve as an alternative for steroid-refractory cases [134]. The potential for systemic absorption and impact on blood glucose should be noted prior to prescribing. In the setting where systemic absorption should be avoided, budesonide may be considered or other approaches provided.

Intralesional steroids, though effective, are more invasive and costly due to procedural fees. For example, intralesional betamethasone showed a greater reduction in erosive area than 40 mg/mL triamcinolone; though pain relief was similar between groups [135].

Systemic steroids require more complex dosing schedules and carry an increased risk of side effects. Although the total cost varies depending on duration and frequency of administration, a 0.5 mg tablet of betamethasone costs around \$0.58, while dapsone, another systemic option, costs about \$1.92 per 100 mg tablet and has shown better efficacy compared to topical triamcinolone, tacrolimus, and retinoids [76].

PDT may offer long-term cost-benefit advantages over medication management due to fewer sessions and possible home

device use. A recent RCT compared PDT and topical clobetasol for treating OLP and found both to be effective, with PDT resulting in greater lesion reduction (79.88% vs. 56.3%) and a higher complete response rate (79% vs. 62%) at 3 months. However, clobetasol remains more accessible and affordable, despite a higher relapse rate [136].

In a case-control study comparing PDT and LLLT, PDT was more effective than LLLT in modulating local inflammation (65% vs. 31% reduction in salivary IL-4); whereas LLLT showed greater pain relief (50% vs. 33.3%) [137].

9 | Conclusion

OLP is a multifactorial and increasingly common condition with significant implications for patient quality of life and a potential risk for malignant transformation. Routine monitoring and testing are usually sufficient for asymptomatic reticular OLP. OLP is not a surgically managed condition. For mild to moderate symptomatic OLP, topical corticosteroids are usually chosen as first-line treatment due to their cost-benefit ratio with continuing follow up. To treat acute exacerbations, persistent, and considerably deteriorating OLP lesions, low, brief doses of oral systemic steroids may be considered; however, long-term use should be

avoided. For the treatment of persistent OLP, extensive lesions, and patients who are either unresponsive to topical corticosteroids or in those at risk of oral candidiasis, topical calcineurin inhibitors and vitamin A derivatives may be used. Vitamin A derivatives may have an additional role to play in suppression of plaque-like change and dysplasia risk. Even though there is evidence that PDT sessions may be more effective and have fewer adverse effects than other treatment approaches, further study is needed. For resistant cases, combination of therapies with different mechanisms of action should be considered. Educating patients on eliminating potential carcinogens, treating erosive lesions, and maintaining a nutritious diet may also reduce cancer risk. Regular cancer screenings, biopsies when indicated due to tissue change, and a multidisciplinary medical approach that may include dental practitioners, oral medicine specialists, dermatologists, rheumatologists, and other specialists ensures comprehensive care and early intervention, essential in managing this OPMD. The development of immunomodulatory agents that target particular cytokines implicated in the LP immune response is a promising development. However, larger RCTs are required to confirm their long-term safety and effectiveness.

Author Contributions

J.B.E. and F.S. identified the need for review, developed the outline, and reviewed and edited the search strategy. E.A. conducted the literature search, created the figures and table, and drafted the original manuscript. J.B.E. and F.S. contributed to the critical revision of the manuscript. All authors read and approved the final manuscript.

Ethics Statement

The authors have nothing to report.

Consent

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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