Jordan Journal of Dentistry, Volume **3**, No. **3**, **2026**
DOI: https://doi.org/10.14525/JJD.v3i3.07

Jordan Journal of Dentistry

https://jjd.just.edu.jo

The Path to Understanding Oral Lichen Planus: From Etiology to Pioneering Management and Therapies: Narrative Review

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ARTICLE INFO

Article History:
Received: 27/9/2025
Accepted: 26/1/2026

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ABSTRACT

Oral lichen planus (OLP) is a chronic inflammatory mucocutaneous disorder that commonly affects the oral cavity. OLP predominantly affects middle-aged adults, with a slight female predilection. The pathogenesis involves a T-cell-mediated autoimmune response, though the exact aetiology remains unclear. Clinically, OLP presents with characteristic bilateral, symmetrical lesions, often exhibiting a reticular pattern with Wickham's striae. Other forms include erosive, atrophic, bullous, and plaque-like variants. Diagnosis relies on clinical examination, histopathology, and direct immunofluorescence. Effective management of OLP involves a multidisciplinary approach, emphasizing patient education on the chronic nature of the disease, potential triggers, and the importance of regular follow-up to monitor for complications, including the risk of malignant transformation, albeit small. This review of literature provides a comprehensive overview of OLP, covering its aetiopathogenesis, clinical manifestations, and the latest knowledge and tools to better diagnose, treat, and manage OLP, aiming to improve patient outcomes and quality of life.

Keywords: Oral lichen planus, Mucocutaneous disorder, Autoimmune, Oral mucosal disease, Malignant transformation, Disease management, Quality of life.

1. Introduction

Lichen planus (LP) is a chronic inflammatory condition that affects the skin, mouth, scalp, genitals, and nails. It is derived from the Greek "leichen" (tree moss) and Latin "planus" (flat), which describes the characteristic flat, moss-like lesions observed on the skin (1). Skin and oral mucosa are primarily afflicted (1), marked by periods of exacerbation and remission, necessitating long-term symptomatic management and attentive monitoring.

Oral lichen planus (OLP) manifests in 77% of LP patients, representing a significant subset of the disease. Notably, OLP patients may also experience cutaneous (15%) and vaginal (20%) manifestations (2). Cutaneous LP typically presents as violaceous, pruritic papules with characteristic reticular white striations known as Wickham's striae. These lesions predominantly erupt on the trunk and extremities, particularly the wrists and ankles. While often self-limiting, cutaneous LP can be persistent in some cases (3).

Genital involvement in LP varies between sexes. In females, it may manifest as erythema, erosion, white reticulated plaques, agglutination, labia resorption, or scarring. Males may develop papulosquamous, annular lesions on the glans penis. Both genders may experience associated symptoms, such as dysuria and dyspareunia (4,5).

OLP lesions, while sometimes asymptomatic, can significantly impact quality of life. Patients may experience dysphagia, dysgeusia, odynophagia, and

heightened sensitivity to certain foods (6). The chronic nature of OLP, its resistance to therapy, and the potential risk of malignant transformation necessitate lifelong monitoring, often leading to psychological distress among affected individuals (7).

2. Epidemiology

The exact prevalence and incidence of OLP remain uncertain due to the variability in clinical presentation and the lack of consistent diagnostic criteria. According to a meta-analysis (9), the worldwide prevalence of OLP is approximately 1.01%, with significant geographical differences ($p < 0.001$). Europe reports the highest prevalence at 1.43%, while India has the lowest prevalence at 0.49%. The lower prevalence in India may be influenced by tobacco-associated keratosis, which can mask the presence of OLP. This variability in reported prevalence is more likely attributable to methodological differences in sampled populations rather than an ethnic predisposition. However, a recent meta-analysis indicated that the pooled prevalence is lower among individuals of Asian origin (9).

OLP is uncommon in children, and its affected age range varies globally. The condition is more frequently observed in individuals who smoke and consume alcohol excessively (10).

Gender differences are notable, as OLP affects women more than men, with a ratio of approximately 2:1. This disparity is often linked to hormonal fluctuations and higher levels of stress (9). In contrast, cutaneous lichen planus does not demonstrate a clear sex preference (11). Interestingly, OLP frequently arises about 10 years later than cutaneous LP (9), although there exists a subset of patients who experience OLP exclusively (11).

3. Etiopathogenesis

The etiology of oral lichen planus is complex and multifactorial, involving an interplay of genetic predisposition, immune dysregulation, environmental triggers, and associations with systemic conditions. The core pathogenesis is characterized by a T-cell-mediated autoimmune response targeting basal keratinocytes, leading to liquefactive degeneration of the basal cell layer and a dense subepithelial band of lymphocytic infiltration (11).

3.1 Genetic and Immunological Basis

A genetic predisposition is supported by associations with specific HLA alleles, such as HLA-B51 and HLA-DR1 (12). This predisposition sets the stage for immune dysregulation, where current evidence suggests that in susceptible individuals, various triggers can initiate a T-helper 1 (Th1) predominant immune response (15,16). This process involves key pro-inflammatory cytokines, such as TNF-α, IL-12, and IL-23 (14). More recent research has highlighted the significant role of IL-17 in inflammation and tissue damage (13) and the abundant subepithelial mast cells, the degranulation of which contributes to the pathophysiology (12).

3.2 Triggers and Systemic Associations

The immune response can be activated by environmental factors, including certain drugs, contact allergens (e.g. metals in dental materials), stress, and infections (15,16). Among infectious agents, the association with Hepatitis C Virus (HCV) is particularly well-established, with meta-analyses confirming a significant correlation (20); the ability of HCV to infect epidermal cells may underlie this link. Other viral agents, such as certain herpesviruses and human papillomavirus, have also been investigated, though their causal role is less definitive (20,21).

Beyond infections, OLP is frequently associated with other systemic and autoimmune conditions. Thyroid dysfunction, especially hypothyroidism characterized by elevated TSH and low FT4, shows a notable prevalence in OLP patients (18). While an association with diabetes mellitus is reported, it remains less clearly defined (22). Patients with OLP also exhibit an increased risk of dyslipidemia, potentially driven by chronic inflammatory cytokines, such as TNF-α, IL-6, IL-10, and IL-4 (23), and a higher incidence of comorbid autoimmune diseases. including alopecia areata, vitiligo, and lichen sclerosus (24). Furthermore, a recent meta-analysis indicates that patients with OLP have a higher prevalence of liver diseases, including hepatitis B, HCV, cirrhosis, hepatic steatosis, and hepatocellular carcinoma, warranting clinical consideration (19).

The development and severity of OLP can thus be influenced by nutritional status, lifestyle, and occupational exposures (17). This intricate interplay between genetic susceptibility, immune pathology, external triggers, and systemic comorbidities underscores the complex nature of OLP and highlights the necessity for a comprehensive diagnostic and management approach.

4. Clinical Presentation

OLP presents in different clinical types, as shown in Table 1, related to the magnitude of the subepithelial inflammation. It can be present as either white or red lesions in a symmetrical and bilateral distribution, but reticular or papular textures serve as a hallmark (Whickham's striae). OLP exhibits six different patterns; papular, reticular, plaque-like, atrophic, erosive, and bullous. The most common are the reticular and erosive-ulcerative types (25). Desquamative gingivitis, in which the gingival epithelium is easily torn away from the underlying submucosa, is a common presentation of erosive OLP (26).

Buccal mucosa is the most common oral site (80%), followed by the tongue (65%), the lips (20%), and less than 10% in the palate and floor of the mouth. OLP lesions can exhibit vesicles and bullae. Associated skin lesions are present in 15% of OLP patients, arising most commonly on the flexor surfaces of the wrists and forearms or in conjunction with other mucosal manifestations, like gastrointestinal and genital lesions (12,27).

OLP exhibits a variable prognosis closely tied to its clinical subtype. The reticular form is often asymptomatic and carries the most favorable outlook, with studies indicating spontaneous remission in a significant proportion of cases over time (25). In contrast, the atrophic-erosive form is typically chronic, symptomatic, and characterized by periods of exacerbation and quiescence (27). This form, particularly when involving high-risk sites, like the lateral and ventral tongue, is associated with a low, but well-documented, risk of malignant transformation to oral squamous cell carcinoma (28).

The malignant potential of OLP remains a carefully qualified concept in contemporary literature. While early studies reported transformation rates as high as 5% or more, more rigorous, long-term cohort studies have refined this estimate. A seminal systematic review by González-Moles et al. (9) concluded that the actual malignant transformation rate of OLP, when strictly diagnosed by modern WHO criteria excluding other lichenoid lesions, is approximately 1.1% over a mean follow-up of 7.5 years. The risk is not uniform; it is significantly higher in the erosive and atrophic-erythematous subtypes and strongly associated with tobacco and alcohol use. The lateral/ventral tongue is consistently identified as the highest-risk intraoral site (28). Despite variations in the clinical presentation, OLP demonstrates bilaterally in the majority of the clinical cases (29). Commonly, OLP occurs alone, but can occur as part of the cutaneous type, or infrequently can be part of the orogenital LP syndromes, including the penogingival syndrome and the vulvovaginal-gingival syndrome (4,5).

Table 1: Clinical manifestations of OLP (13)

Clinical Type and Variant	Description and Key Features	Most Common Site(s)
Reticular (Hyperkeratotic)	Most common variant. Mostly asymptomatic. Presents as a lace-like network of slightly raised gray-white lines (Wickham's striae)	Buccal mucosa (inner cheeks), bilateral. Also on tongue and gingiva
Plaque/Verrucous (Hyperkeratotic)	Appears as whitish, homogeneous, irregular patches similar to leukoplakia	Dorsal surface of the tongue and buccal mucosa
Papular (Hyperkeratotic)	Features small, discrete white papules, often with fine striae at the periphery. Frequently occurs alongside reticular lesions	Buccal mucosa, often intermingled with reticular patterns
Erosive (Erosive)	Resistant and recurrent. Characterized by redness, ulceration, and surrounding keratotic white striae. Pseudomembranes may be present. Causes a burning sensation to excruciating pain, affecting speech and eating.	Buccal mucosa, tongue, and gingiva
Erythematous/Atrophic (Erosive)	Presents as diffuse, red, atrophic lesions. Often combines features of other types, with an erythematous region encircling reticular-type white striae	Buccal mucosa and tongue
Bullous (Erosive)	Unusual form. Begins as painful blisters that rupture, leaving behind ulcerations. A positive Nikolsky's sign can occasionally be demonstrated	Buccal mucosa and gingiva. Less commonly on the tongue

5. Diagnosis

5.1 History Taking

The initial step in diagnosing OLP involves a comprehensive patient history, which is crucial for identifying the key clinical features and temporal patterns of the condition. Chronicity is a primary consideration, as OLP typically presents as a long-term condition. Patients often report alternating periods of exacerbation and quiescence, demonstrating the characteristic waxing and waning nature of the disease. The history should explore oral sensitivity, particularly to spicy or acidic foods, and mucosal discomfort, which may manifest as pain, burning sensations, or a metallic taste. Textural changes, such as mucosal surface roughness or tightness, are common complaints in symptomatic OLP patients. Associated symptoms, like dryness, redness, or swelling in the oral cavity, should also be noted (30). The progression and refinement of the diagnostic criteria for oral lichen planus (OLP) are summarized in Table 2.

6. Examination

6.1 Physical Examination

The cornerstone of diagnosis is a meticulous clinical examination. Clinicians assess the lesions for their characteristics, including distribution (unilateral or bilateral), appearance (e.g. reticular, papular, or plaque-type), and symmetry. In classic cases, lichen planus can be diagnosed clinically, but a biopsy is often performed to confirm the diagnosis, especially when it manifests as desquamative gingivitis, and to rule out other conditions (25).

6.2 Medical History

A comprehensive medical history is essential and should include assessing for potential triggers, like stress or psychological problems. Taking the history of any concurrent medical condition, such as especially HCV infection and immune-mediated disorders, is important.

6.3 Signs and Symptoms

OLP can present with a variety of clinical signs and symptoms. OLP often develops slowly in patients, who are unaware of their oral health condition. Some patients complain of oral ulcerations, red or white spots on the oral mucosa, pain, sensitivity of the oral mucosa to hot or spicy foods, and roughness of the oral lining (32). Almost two-thirds of OLP patients experience oral discomfort to some extent (33). Relapses and remissions are common in OLP; during an exacerbation, symptoms and clinical manifestations will become worse, while during a quiescence, OLP symptoms tend to decrease (32).

Stress is one factor that might exacerbate the disease's clinical manifestation (34). Sharp cusps and poorly fitting dental prostheses may be responsible for precipitating factors that impact the oral cavity, which are similar to the Koebner phenomenon, a feature of cutaneous LP in which lesions form in reaction to trauma (35).

It has also been demonstrated that OLP is worsened by the buildup of calculus and plaque (36). Advanced periodontal disease and gingival recession may eventually result from gingival OLP (37). Patients with OLP and oral lichenoid reactions (OLRs) have been reported to have diffuse dark or black oral post-inflammatory pigmentation after the spread of lichenoid lesions (38).

Table 2: Evolution of diagnostic criteria for oral lichen planus (31)

Organization	Year	Clinical Criteria	Histopathologic Criteria
World Health Organization	1978	- Multiple, often symmetric lesions - Various forms: papules, reticular, or plaque - Central papules with radiating gray-white lines - Atrophic lesions, with or without erosion - Rare bullous lesions	- Thickened ortho/parakeratinized layer in keratinized sites; thin layer in non-keratinized sites - Civatte bodies in basal epithelium and superficial connective tissue - Well-defined band-like zone - Lymphocytic infiltration in superficial connective tissue - Basal cell layer liquefaction degeneration
Modified WHO diagnostic criteria (van der Meij and van der Waal)	2003	- Bilateral, mostly symmetric lesions - Reticular pattern with gray-white lines - Erosive, atrophic, bullous, plaque-type lesions only with reticular lesions elsewhere - Other similar lesions termed "clinically compatible with" OLP	- Well-defined band-like lymphocytic infiltration in superficial connective tissue - Basal cell layer liquefaction degeneration - No epithelial dysplasia - Less clear features termed "histopathologically compatible with" OLP

American Academy of Oral and Maxillofacial Pathology	2016	- Multifocal symmetric distribution - White and red lesions in various forms (reticular/papular, atrophic, erosive, plaque, bullous) - Not exclusively in smokeless tobacco sites or adjacent to dental restorations - Onset unrelated to medication start or cinnamon product use	- Band-like or patchy lymphocytic infiltrate at epithelium-lamina propria interface - Basal cell liquefactive degeneration - Lymphocytic exocytosis - No epithelial dysplasia - No verrucous epithelial architectural change
Organization	**Year**	**Clinical Criteria**	**Histopathologic Criteria**
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7. Mimics of OLP

Lichenoid drug eruption (LDE) is a type of LP that was initially reported in 1929. The first examples of oral involvement were observed in 1949 among military soldiers in the South-West Pacific who were being treated with mepacrine, an antimalarial medicine, as a preventive measure (39).

Oral lichenoid lesions (OLLs) encompass a spectrum of conditions that clinically and histologically resemble oral lichen planus (OLP), but have distinct etiologies. Current classification recognizes four primary categories of OLL (40):

1. Lichenoid manifestations in chronic graft-versus-host disease (cGVHD)
2. Oral lichenoid contact hypersensitivity reactions (OLCHR)
3. Oral lichenoid drug reactions (OLDR)
4. Lichen planus-like lesions with atypical features.

The fourth category includes conditions, such as lichen planus pemphigoides, chronic ulcerative stomatitis, and lupus erythematosus, which share some characteristics with OLP, but lack one or more definitive clinical or histopathological features (40,41). They are indistinguishable from OLP, either clinically or histologically, which poses a challenge in the diagnosis. Examples of drugs and materials that can lead to OLDR are listed in Table 3. Table 4 presents an overview of conditions that should be considered in the differential diagnosis of OLP.

Table 3: Category of drugs and materials responsible for ORLs (26)

Category	Specific Causes
Antibiotics	Tetracycline
Anticonvulsants	Carbamazepine, Oxcarbazepine, Phenytoin, Valproate sodium
Antidiabetics	Chlorpropamide, Glipizide, Insulin, Tolbutamide
Antidiarrheals	Bismuth
Antifungals	Amphotericin B, Ketoconazole
Antihypertensives	Atenolol, Enalapril, Hydrochlorothiazide, Methyldopa, Metoprolol
Antimalarials	Chloroquine, Hydroxychloroquine, Quinidine, Quinine
Antimycobacterials	Aminosalicylate sodium, Isoniazid, Rifampin, Streptomycin
Antiretrovirals	Zidovudine
Chemotherapeutics	Dactinomycin, Imatinib
Immunomodulatory drugs	Gold salts, Interferon-α, Penicillamine
NSAIDs	Aspirin, Diflunisal, Ibuprofen, Indomethacin, Naproxen, Rofecoxib, Sulindac

Psychiatric drugs	Benzodiazepines, Tricyclic antidepressants, Lithium
Dental adhesives	Acrylate compounds, Eugenol
Dental metals	Beryllium, Cobalt, Copper, Chromium, Gold, Mercury, Nickel, Palladium, Silver, Tin
Other dental restoration materials	Composite, Glass ionomer, Porcelain
Flavorings	Balsam of Peru, Cinnamon (cinnamic aldehyde), Eugenol, Menthol, Peppermint, Vanillin

Table 4: Differential diagnosis of OLP, based on the clinical form (15,48)

Lesion Type	Differential Diagnosis
White reticular OLP	Candidiasis, Discoid lupus erythematosus, Chronic Ulcerative stomatitis, Leukoplakia, Morsicatio buccarum et labium (Linea alba), Mucous patches of secondary syphilis, Oral hairy leukoplakia, Lichenoid dysplasia, Squamous cell carcinoma (SCC), Poor oral hygiene
Erosive OLP	Immunobullous disorders, Connective tissue disorders, Lichenoid reactions to drugs or infections, Desquamative gingivitis, Mucous membrane pemphigoid, Pemphigus vulgaris, Systemic lupus erythematosus (SLE)

8. Diagnostic Methods

An incisional biopsy is considered the gold standard method for confirmation of the diagnosis and to rule out dysplasia and malignancy. A single tissue sample is usually sufficient for typical lesions, while multiple samples may be needed for atypical presentations (32).

a) Histopathologic Diagnosis: reveals several characteristic features that include areas of hyper-parakeratosis accompanied by thickening of the granular cell layer, and a distinctive saw-toothed appearance of the rete pegs. A key finding is the presence of "liquefaction degeneration," which refers to the necrosis of the basal cell layer. An eosinophilic band containing fibrin can be observed just below the basement membrane. Additionally, a dense subepithelial band-shaped infiltrate composed of lymphocytes and macrophages is typically present, further characterizing the disease (32,33,37).

b) Direct Immunofluorescence (DIF): This technique serves as a crucial complementary tool when clinical and histopathologic information is insufficient to confirm the diagnosis. DIF can help distinguish OLP from other autoimmune conditions, such as pemphigus vulgaris and benign mucous membrane pemphigoid, by identifying specific globular deposits of C3, fibrinogen, IgA, most commonly IgM, and IgG along with apoptotic keratinocytes (24,42,43).

c) Blood Tests: It is critical to note that no serologic test can confirm a diagnosis of OLP. However, judicious use of blood tests is recommended to rule out systemic mimics (e.g. autoimmune blistering diseases) and to identify potential triggering conditions. This is particularly relevant for hepatitis C virus (HCV) infection, which has a well-documented epidemiological association with OLP, especially in patients from endemic areas or with risk factors (19).

d) Patch Test: used as an auxiliary diagnostic method to help identify positive reactions to dental materials in cases of OLL, potentially indicating the need for replacement of dental restorations (44).

8.1 Recent Advances in Diagnostic Measures

Non-invasive diagnostic approaches are being explored to monitor therapy response, increase accuracy of the diagnosis and predict malignancy in LP. These techniques include dermoscopy, reflectance confocal microscopy (RCM), optical coherence tomography (OCT), diffuse reflection spectrophotometry (DRS), and ultrasonography (USG) (45).

Dermoscopy can be used to get a clearer view of the Wickham's striae, as it may occur in numerous forms, such as reticular, rounded, globular, circular, annular, or arboriform (46). Given that WS typically goes away after treatment, its presence can be taken as an indication of ongoing disease (47). Moreover, vascular features, like red spots or lines and diffuse, dotted, or clustered

hyperpigmentation, are dermoscopic indicators. Dermoscopy of LP in coloured skin is distinguished by variations in WS, including a black-hole pigmentation pattern, and the lack of vascular characteristics. It has been shown that it helps identify LP in people with dark coloured skin (48).

9. Management

Management should prioritize reassurance for asymptomatic lesions while treating symptomatic manifestations. The primary objectives are to alleviate associated symptoms and mitigate the potential risk of malignant transformation (37,43). Clinicians should pay particular attention to potential mechanical trauma or irritants, such as sharp filling margins, rough surfaces, or ill-fitting dentures. A thorough drug history is crucial to identify reversible causes of lichenoid eruptions, as discontinuation of the offending agent may be curative when feasible (37). For patients with gingival involvement, implementation of an optimal oral hygiene regimen is essential. It is important to note that the treatment of mucosal lichen planus can be challenging, often resulting in transient improvements and short-lived remissions (49). Consequently, the cornerstone of management is palliative care, aimed at minimizing pain and discomfort (50).

Topical corticosteroids are the recommended first-line treatment for OLP and are generally considered safe, although rare adverse effects, such as mucosal atrophy and candidiasis, may occur (24,51). In cases of extensive and/or erosive OLP, systemic corticosteroids may be employed to achieve rapid symptom resolution (46), Table 5 lists an overview of topical and systemic therapeutic modalities, along with their potential side effects, which are essential for guiding clinical decision-making in the management of OLP.

Table 5: Different treatment modalities for OLP (46,51)

Treatment Type	Description	Efficacy	Adverse Effects
TOPICAL			
Corticosteroids	First-line treatment for acute OLP flares; includes clobetasol propionate, triamcinolone acetonide, fluocinolone acetonide, dexamethasone, betamethasone dipropionate	No significant difference from placebo in clinical resolution; better pain resolution	Oral candidiasis, dry mouth, mucosal atrophy; systemic absorption not clinically meaningful
Calcineurin Inhibitors	Tacrolimus, pimecrolimus, ciclosporine; potent immunomodulators suppressing T-cell activity	Comparable efficacy to corticosteroids; pimecrolimus better at preventing symptom relapse	Burning sensation, dysgeusia, xerostomia, reflux; more frequent adverse events than corticosteroids
Retinoids	Vitamin A derivatives (retinoic acid, tretinoin, tazarotene, isotretinoin); modulate inflammatory mediators and reduce keratinization	Heterogeneous efficacy; improvement rates from 0 to 100%	Transient and moderate burning sensation
Rapamycin	Macrolide inhibiting mTOR activation with immunosuppressant activity	Higher remission rates with corticosteroids in short term; rapamycin led to adverse events in nearly a half of patients	Burning sensation, impaired taste
SYSTEMIC			
Corticosteroids	Prednisone, prednisolone; first-line therapy for rapid symptom relief in erosive OLP	No significant difference in remission rates compared to topical corticosteroids; rapid short-term symptom alleviation	High blood pressure, weight gain, muscle weakness, anemia, sleep disorders
Retinoids	Isotretinoin, acitretin, alitretinoin; used in refractory cases	Substantial improvement in some patients; alitretinoin showed 50% reduction in disease severity in some studies	Mucocutaneous dryness, cheilitis, musculoskeletal pain, teratogenicity, hepatotoxicity

Hydroxychloroquine (HCQ)	Immunomodulatory agent primarily used in cutaneous lupus erythematosus.	Effective in 70%-100% of patients; considered among first-line systemic treatments for refractory OLP	Minor gastrointestinal and neuromuscular symptoms; rare long-term effects (e.g. retinopathy)
Conventional Immunosuppressives	Methotrexate, azathioprine, mycophenolate mofetil; used for severe cases	Methotrexate showed substantial improvement in several reports; azathioprine effective in most patients	Gastrointestinal symptoms, hepatotoxicity, hematotoxicity, risk of neoplasia
Thalidomide	Enhances immunosuppressive T-cell activity; used for refractory erosive OLP	Complete or partial healing of erosive lesions reported in several cases	Teratogenicity, peripheral neuropathy, drowsiness, rashes
Biologics	TNF-alpha inhibitors and IL inhibitors (e.g. secukinumab, ustekinumab); used for refractory cases	Mixed results; secukinumab showed improvement but no superiority over placebo in trials	Not specified in detail
Oral Targeted Therapies	Apremilast and JAK inhibitors (e.g. tofacitinib); targeting inflammatory pathways	Reports of improvement in refractory OLP; JAK inhibitors showed efficacy in single case reports	Adverse effects led to treatment discontinuation in some patients

9.1 Recent Advances in Therapeutic Options

A systematic review conducted by Shazina et al. in 2022 showed a significant improvement in OLP symptoms in patients who received vitamin D supplements in addition to traditional steroid therapy or a placebo. However, more studies with bigger sample sizes are needed to confirm these findings (52). To add, targeting the mast cells in OLP could be a useful therapeutic approach (16). Another recent study has evaluated the effect of injectable platelet rich fibrin in the treatment of lichen planus. Those concentrates are derived from the patient's blood and are full of platelets and growth factors. They are commonly used in oral surgery, periodontal treatment and implantology. They are known to stimulate tissue regeneration, reduce inflammation, and enhance wound healing (53).

Promising outcomes have been shown in recent research on the use of Janus kinase (JAK) inhibitors to treat lichen planus (LP), where 73.3% of LP cases treated with those drugs had partial or full remission, according to a systematic review (54). Benefits of JAK inhibitors are quick symptom relief, frequently in a period of days or weeks. They are also well tolerated by patients without experiencing any serious adverse effects and promising results were seen in patients who had not responded to any other treatments (55,56).

Additionally, targeting of the IL-23/IL-17 axis, which has a central role in the pathogenesis, was shown to result in improvement of cutaneous LP and/or OLP (57). Other newly emerging treatment modalities to manage OLP are under investigation and these include; topical aloe vera, biologics, low intensity laser, photodynamic therapy and oral curcuminoids (58-60).

9.2 Monitoring and Follow-up

Long-term management of OLP requires regular follow-up due to its chronic nature and the potential for malignant transformation, with the risk estimated at 0.4% to 5% (40). Erosive and atrophic types, especially on high-risk sites, like the buccal mucosa, tongue, and floor of the mouth, have a higher likelihood of transformation, particularly in patients with dysplasia, tobacco use, and immunosuppression (61). While the optimal frequency of monitoring remains undefined due to insufficient evidence, current practice suggests annual clinical evaluations for most cases, with more frequent assessments (every 2-6 months) for erythematous, erosive, or atrophic forms that carry a higher risk of malignant progression (62).

10. Conclusions

In conclusion, this clinical review has provided a comprehensive overview of OLP, encompassing its etiology, clinical presentation, and management strategies. The complex nature of OLP, characterized by its chronic course and diverse manifestations, underscores the importance of a specialized approach to patient care. Healthcare professionals, including doctors and dentists, play a crucial role in the early recognition and appropriate management of this condition. This paper has outlined essential guidelines for diagnosis and treatment of OLP, focusing on symptom alleviation and

prevention of complications and acute flares. The document also highlights novel therapies, and targeted approaches, which may offer hope for patients with refractory disease. This comprehensive summary aims to equip healthcare professionals with the necessary knowledge to effectively recognize, diagnose, and manage the various manifestations of OLP, ultimately improving patient outcomes and quality of life.

Conflict of Interests

The authors have no conflict of interests.

Funding Information

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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