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Oral lichen planus: key features of etiopathogenesis, diagnosis, and management

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Abstract

Oral lichen planus (OLP) is a chronic inflammatory autoimmune disease of unknown etiology. It is assumed that a genetic predisposition contributes to the development of the disease and influences the patient's response to various etiological factors such as autoimmune reactions to epithelial antigens, microorganisms, and stress. Immunopathogenesis is primarily driven by cell-mediated immune mechanisms, with T lymphocytes playing a central role. The clinical presentation of OLP is varied, and multiple clinical forms can occur in the same patient. OLP is categorized into six clinical types: reticular, papular, and plaque-like (hyperkeratotic variants), and atrophic, erosive, and bullous (erosive variants). The histopathological diagnosis of OLP is unique. Continuous follow-up of patients is crucial because OLP is considered an oral potentially malignant disorder (OPMD). Reported rates of malignant transformation vary, with a pooled estimate of 1.43% for OLP and 5.13% for OLP with dysplasia. Patient education plays a crucial role in treatment initiation and planning. A personalized treatment approach focuses on controlling inflammation and relieving symptoms such as pain and burning. Treatment should be individualized according to disease severity, subtype, and patient response, with constant monitoring for possible malignant transformation and comorbidities.

Keywords: etiopathogenesis, clinical features, malignant transformation, oral lichen planus, treatment

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Introduction

The first clinical description of lichen planus (LP) is attributed to Ferdinand Ritter von Hebra, who described the disease as "lichen ruber planus" in 1860 (1). The first published description of oral lichen planus (OLP) was by Erasmus Wilson in 1866, who described a white papular rash on the tongue and buccal and lower lip mucosa of a 56-year-old woman (2). This was followed in 1869 by a report of 50 further cases of OLP (3). In 1895, Louis Wickham discovered the characteristic white striae of cutaneous LP—known as Wickham's striae (1, 4)—whose bilateral appearance on the oral mucosa greatly facilitated the diagnosis of OLP. For four decades, OLP was diagnosed solely based on clinical findings until William Dubreuilh described the histopathological features in 1906 (3). Despite advances in understanding, accurate diagnosis of OLP is still a challenge even for experienced clinicians and requires careful correlation of clinical and histopathological criteria.

Oral LP is a chronic autoimmune mucocutaneous disease. Lesions on the oral mucosa can sometimes be accompanied by skin manifestations. If the mucous membranes of the esophagus, stomach, and cervix are also affected in addition to the oral mucosa, it is referred to as mucosal lichen planus. The global pooled prevalence of OLP is estimated at around 1.01%, although there are considerable geographical differences. The highest prevalence was reported in Europe (1.43%) and the lowest in India (0.49%). The prevalence increases significantly after age 40 (5) and is most commonly observed in patients 30 to 80 years old, with a higher incidence in women (1, 6).

Etiopathogenesis

OLP is characterized by an alternation of remission and relapse. A number of possible factors have been suggested, including autoimmune responses to epithelial antigens, microbial pathogens, and psychological stress (7, 8). Some etiological factors are well documented (Table 1). There is undoubtedly a link between stress and OLP, but the cause-and-effect relationship has not been established (9). Numerous studies have shown a dysregulated immune response in OLP, which is why it is considered an autoimmune disease (10). Hepatitis C virus (HCV) is the only microorganism that has been convincingly linked to OLP, but only in some geographical areas (7, 11, 12).

The immunopathogenesis of OLP is characterized by a type 1 immune response directed against exogenous or self-modified

Table 1 | **Possible causative factors.**

Causative factor	Specific data on causative factor
Autoimmune reaction	$CD8^+$ T lymphocytes attack the oral epithelium, often in response to autoantigens.
Medications (drug-induced)	Most commonly caused by gold therapy; non-steroidal anti-inflammatory drugs, antihypertensives, antimalarials, and antiretrovirals can also trigger a lichenoid rash, although the oral cavity is less commonly affected.
Dental materials (allergic reactions)	Contact allergens in dental materials such as mercury, nickel, gold, resins, and acrylates can cause oral lesions.
Viral infections	Hepatitis C virus has been associated with the development of oral lichen planus.

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antigens presented by antigen-presenting cells (APCs) such as dendritic cells (DCs) and keratinocytes. This immune activation leads to damage of the oral mucosal epithelium (13, 14). Plasmacytoid and myeloid dendritic cells, CD4+ and CD8+ T lymphocytes, natural killer (NK) cells, and mast cells are all involved in this process. Key soluble mediators in the inflammatory microenvironment—such as interferon (IFN)-γ, interleukin (IL)-12, and tumor necrosis factor (TNF)-α—play an important role in triggering the disease (13, 15). Activation of T cell–mediated immunity leads to the upregulation of cytokines, chemokines, and adhesion molecules, which together facilitate the infiltration of T cells and mast cells into the affected mucosa. This cascade ultimately leads to keratinocyte apoptosis, disruption of the basement membrane (BM), and chronic inflammation. The main effector cells are CD8+ cytotoxic T lymphocytes and CD4+ Th1-polarized T cells. However, recent evidence suggests that other T helper subsets—including Th9, Th17, and regulatory T cells—are also involved, indicating a more complex immune network underlying OLP pathogenesis (14, 16). Antigen presentation by keratinocytes and Langerhans cells activates both CD4+ helper cells and CD8+ cytotoxic T cells. Activated T helper cells secrete IL-2 and IFN-γ and thus promote the proliferation and activation of cytotoxic T lymphocytes, which in turn induce apoptosis of basal keratinocytes, a key histopathological feature of OLP. TNF-α also supports the migration of T cells from the bloodstream into the extracellular matrix of the oral mucosa (17). In addition, several cytokines have been implicated in the pathogenesis of OLP, including IL-6. A study by Glavina et al. reported a slight increase in salivary IL-6 levels in OLP patients (18).

In addition, mast cell degranulation releases cytokines, chemokines, and matrix metalloproteinases (MMPs), which further enhance T cell activation, migration, and tissue remodeling, contributing to chronicity and tissue damage in OLP (19). Recent studies have also demonstrated significant dysbiosis of the oral microbiome in OLP patients, particularly in the buccal mucosa. However, no single microorganism has been identified as specifically associated with OLP in several studies, suggesting that the functional properties of the oral microbiota—rather than its exact taxonomic composition—may play a more important role in pathogenesis. The complex interactions between host factors and the oral microbiota, as well as the functional consequences of these microbial shifts, are not yet fully understood and require further investigation (20).

It has also been shown that allergies can be the cause or trigger of OLP (21, 22). For example, contact allergens from dental restorative materials, such as nickel, mercury, resins, acrylates, gold or toothpaste, especially mint, have been found to cause OLP. To confirm possible contact allergens, an allergy patch test is recommended. However, it must be checked whether some allergens that were positive in the clinical test are relevant factors that trigger manifestations.

Clinical features

The clinical presentation of OLP is varied, and multiple forms can occur simultaneously, which can complicate the diagnostic process. A characteristic clinical sign of OLP is the presence of well-defined intertwined white lines known as Wickham's striae (1, 6). These lesions usually occur symmetrically, and they are most commonly found on the buccal mucosa, gingiva, and tongue. When the gingiva is affected, OLP usually manifests as immune-mediated desquamative gingivitis (23). OLP occurs in various clinical patterns (Table 2) and is generally classified into six subtypes: three hyperkeratotic forms—reticular (characterized by Wickham's striae), papular (Fig. 1), and plaque-like (Fig. 2)—and three erosive forms, which include atrophic, erosive, and bullous types (6, 23). Of these forms, the reticular type is the most common, and it is characterized by lacy or reticular white striae. However, its frequency may decrease over time and transition into other subtypes, particularly the erosive form. Plaque-like OLPs are characterized by homogeneous white patches, which are more common in patients with a history of tobacco use. These lesions often persist after smoking cessation. In such cases, malignant leukoplakia must be excluded due to the clinical similarity between these entities (17). Erosive OLP typically presents with multifocal, atrophic, or erythematous erosions and ulcerations. The atrophic subtype is very similar to the erosive form but shows more distinct atrophic areas on a red inflamed background. It mainly affects the gingiva and the posteroinferior buccal mucosa, especially near the second and third molars. The bullous form is extremely rare (17).

About two-thirds of OLP patients suffer from symptoms of discomfort (24). The severity of symptoms can vary greatly; in some individuals, symptoms are triggered only by contact with spicy or acidic foods, whereas in others they may occur spontaneously. Other symptoms may include a rough feeling of the oral mucosa, reduced elasticity, and restricted mouth opening. OLP is known to have a negative impact on patients' quality of life (QoL) (6, 18).

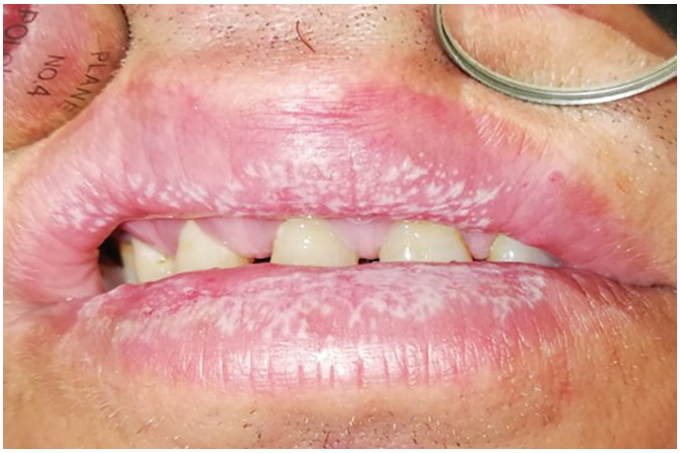

Figure 1 | Papular and reticular form of oral lichen planus on the vermilion of the lips.

Table 2 | **Clinical manifestations of oral lichen planus.**

Form	Special features
Papular or reticular	White, lacy papules or striae (Wickham's striae) in the buccal mucosa, which are asymptomatic.
Erosive or atrophic	Characterized by erosions or ulcerations with a painful red base and whitish edges, often occurring on the gums and lips.
Plaque-like	Appears as raised, thickened white patches that can be mistaken for leukoplakia.
Bullous	Characterized by the formation of blisters or bullae that may burst, leading to erosions. This form is less common but can cause considerable pain.

Diagnosis

The diagnosis of OLP can be challenging even for experienced clinicians because it is clinically and histopathologically similar to a number of other diseases. These include cicatricial pemphigoid, lichen planus pemphigoides (LPP), chronic graft-versus-host disease (cGVHD), chronic ulcerative stomatitis (CUS), oral lichenoid drug reaction (OLDR), oral lichenoid contact hypersensitivity reaction (OLCHR), lupus erythematosus (LE), proliferative verrucous leukoplakia (PVL), and oral epithelial dysplasia. Sometimes the disease can resemble certain oral diseases such as cheilitis and also oral manifestations associated with coronavirus disease (COVID-19) (25, 26).

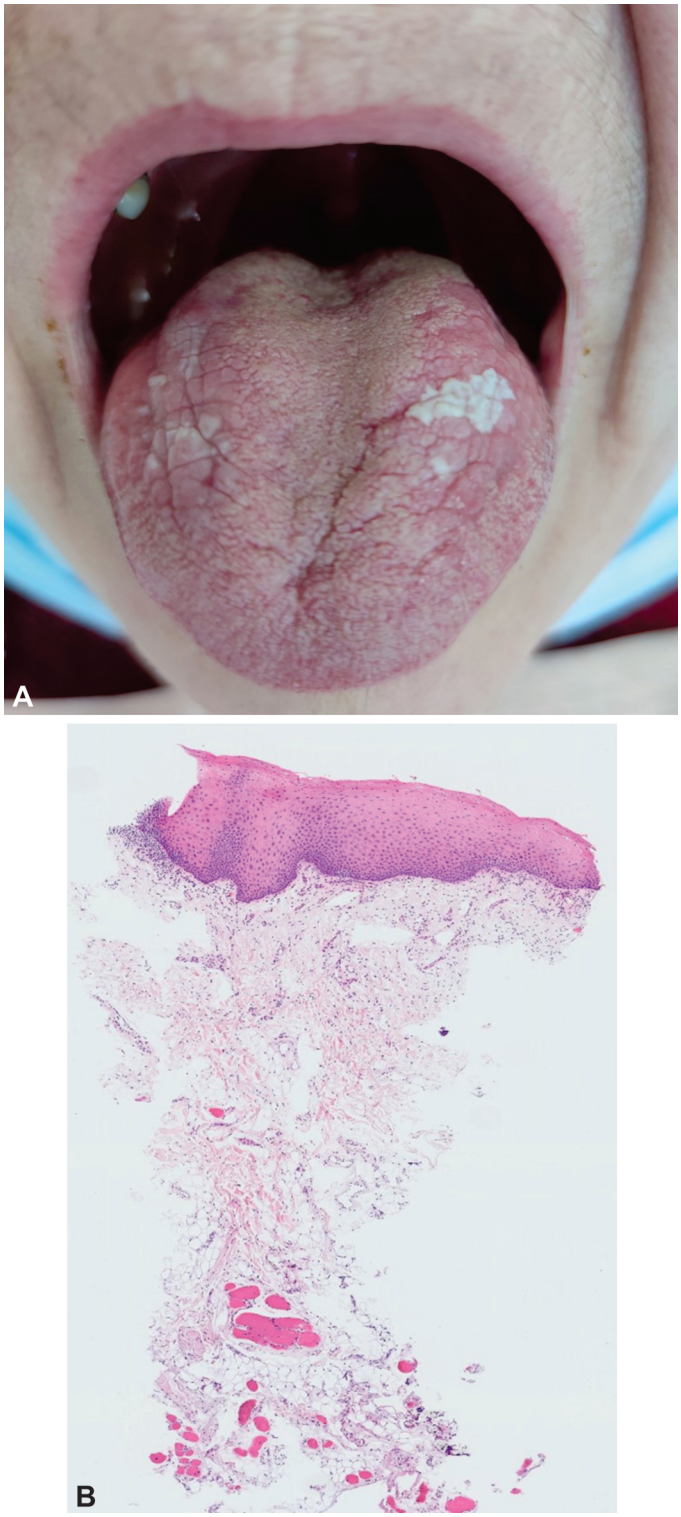

Figure 2 | Oral lichen planus (OLP): a) plaque form of OLP on the mucosa of the dorsum of the tongue, b) histopathological findings: thick squamous epithelium with pronounced acanthosis and dense band-like infiltrate of CD8+ T lymphocytes infiltrating the basal epithelial layer (hematoxylin & eosin, 40×).

In 1978, the World Health Organization (WHO) Center for Precancerous Lesions established the first histopathological criteria (27). These criteria include the presence of orthokeratosis or parakeratosis with varying epithelial thickness. Occasionally, characteristic "sawtooth" rete ridges can be observed. Colloid bodies (also known as Civatte bodies) may occur in the basal cell layer or in the superficial lamina propria. Other features include liquefaction degeneration of the basal cell layer and the presence of a narrow band of eosinophilic material in the BM zone. A dense, well-defined, band-like infiltrate consisting mainly of lymphocytes is typically confined to the superficial lamina propria (27).

In 2003, Van der Meij and van der Waal introduced changes to the original WHO criteria (28). The revised histopathological criteria retain some elements of the WHO guidelines, such as the presence of the aforementioned inflammatory infiltrate composed mainly of lymphocytes, together with liquefactive degeneration of the basal epithelial cell layer. The authors emphasize that a definitive diagnosis of OLP requires confirmation that epithelial dysplasia is not present. They recommend that pathologists should use the term "histopathologically compatible with" OLP in cases in which the histological features are not clearly defined. The authors also point out the inherent subjectivity of such judgements, which must be carefully considered when interpreting them.

The microscopic diagnosis of OLP is difficult, partly due to the different histopathological features. The presence of hyperkeratosis in OLP varies depending on the biopsy site and clinical form. Reticular, papular, and plaque variants often show hyperkeratosis, whereas the atrophic and erosive forms usually do not show hyperkeratosis. One of the most characteristic microscopic features of OLP is a dense band-like mononuclear lymphocytic infiltrate in the superficial lamina propria. However, macrophages and DC subsets may also be present (29). The intensity of the inflammatory infiltrate can vary considerably, possibly reflecting the degree of disease activity and influenced by previous therapeutic interventions. The clinical presentation of the OLP, the anatomical location, or concomitant inflammatory processes may influence the presence of different subsets of inflammatory cells. In erosive forms, additional neutrophil-rich inflammation due to ulceration may alter the histological appearance. Gingival lesions often contain a mixed inflammatory infiltrate that includes plasma cells, indicating the coexistence of gingivitis or periodontitis, often associated with dental plaque or calculus. Another feature of OLP recognized in both the original (1978) and revised (2003) WHO diagnostic criteria is liquefactive degeneration (27, 28). However, this feature is not unique to OLP but can also be observed in biopsy specimens of cGVHD, LE, OLDR, and OLCHR (30, 31). The presence of Civatte bodies (colloidal, hyaline, or cytoid bodies) is a feature that supports the diagnosis of OLP, but they can also be observed in LE, OLDR, and cGVHD (32). The presence of "sawtooths" supports the diagnosis of OLP but is not considered a definitive diagnostic feature. Although eosinophils are generally not present in OLP, they are commonly found in cicatricial pemphigoid and are frequently seen in contact hypersensitivity reactions, and so their presence is a potential indicator of OLCHR.

A recently proposed set of diagnostic criteria for OLP includes both clinical and histopathological features and builds on previous recommendations. Clinically, OLP is characterized by bilateral, often symmetrical, white lesions with or without concomitant

erosions, ulcerations, or desquamative gingivitis. In addition to the previously described microscopic features of OLP, which include the absence of epithelial dysplasia, the latest diagnostic criteria explicitly refer to the absence of verrucous epithelial architecture. The integration of these clinical and histological features is essential for the definitive diagnosis of OLP and for differentiation from other oral mucosal diseases with overlapping lichenoid features (33).

Although OLP has a broad spectrum of clinical manifestations, its histopathological diagnosis (HPD) is unique. Microscopic features of OLP include hyperparakeratosis, hyperorthokeratosis, and their combination; cytoid (Civatte) bodies; hydropic degeneration of the basal layer of the epithelium; and, most importantly, a dense, band-like inflammatory lymphocytic infiltrate in the lamina propria. Other findings include epithelial expansion, which is referred to as a "sawtooth" due to its appearance, atrophy, acanthosis, homogeneous eosinophilic deposits at the interface between the epithelium and connective tissue, and ulceration. Melanosis and melanin incontinence associated with melanophages can also be seen in biopsy specimens, especially in individuals with darker skin (27, 34).

Direct immunofluorescence (DIF) is an additional diagnostic tool that can help with the final diagnosis. It is particularly helpful in differentiating OLP from autoimmune bullous diseases with the clinical picture of desquamative gingivitis (35). The characteristic immunofluorescence pattern of OLP is tuft-like deposits of fibrinogen along the BM in the absence of immunoglobulin (with the exception of immunoglobulin-coated cytoid bodies) and complement (36). However, fibrinogen has also been detected in the BM of other potentially malignant and malignant oral lesions, suggesting that this finding is not exclusive to OLP (37). Although DIF increases the cost of diagnosis, it may be necessary when clinical and histopathological evidence alone is insufficient. In contrast, indirect immunofluorescence (IIF) gives negative results and is not considered a useful diagnostic test for OLP.

The final diagnosis of OLP is based on the patient's medical and dental history, the clinical oral examination, and the histopathological findings. In cases of uncertainty or ambiguity, it is recommended that an oral medicine specialist and a pathologist engage in an active discussion. Histopathological criteria, such as those proposed by van der Meij and van der Waal, require the absence of epithelial dysplasia (28, 38). Because PVL may have clinical and histopathological features similar to OLP, this recently proposed set of diagnostic criteria also includes the absence of verrucous epithelial architecture. When interpreting the histopathological findings, the clinician must bear in mind that the presence of eosinophils or a perivascular lymphoplasmacytic infiltrate in the deeper layers of the lamina propria generally excludes OLP. Clinicians need to be aware that the diagnosis of OLP can be difficult and that the diagnostic process should not be considered complete after a single biopsy. Follow-up is important to monitor response to therapy and any changes in the clinical picture. Repeat biopsies for HPD and/or DIF will help the clinician make an accurate and definitive diagnosis. If the clinical appearance of the lesions, such as the development of verrucous changes, differs from the typical OLP features, further biopsies are warranted. As part of these new diagnostic criteria, it is recommended that a checklist be used to help the clinician identify and collect the data necessary for the diagnosis of OLP. The list includes anamnestic data such as history of organ transplant/cGVHD, LE (systemic/discoid), HCV and/or other liver disease, tobacco use (in any form), medications, and products containing cinnamon (food, chewing gum, or toothpaste), as well as clinical information on oral lichenoid lesions (OLL) (38). The completed list should be sent to the pathology specialist together with the biopsy sample.

Malignant transformation

Fritz Williger published the first documented clinical case of malignant transformation of OLP in 1924 (39). The reported rates of malignant transformation in OLP range from 0.4% to 12.5%. A recent systematic review estimates the pooled rate of malignant transformation to be 1.43% for OLP overall and 5.13% for cases with dysplasia (7, 40–42). In the Global Oral Health Programme of 2005, the WHO classified OLP as a premalignant condition (43). OLP is considered an oral potentially malignant disorder (OPMD), which is why these patients should be followed up (44). Established risk factors for malignant transformation of OLP lesions into oral squamous cell carcinoma (OSCC) include bad habits (smoking and alcohol), clinical appearance of the lesions (erythematous and heterogeneous lesions), topography of the lesions (tongue margins), HCV infection, and chronic kidney disease (CKD) (42, 45, 46). OSCC accounts for about 90% of all malignant tumors in the head and neck region and was considered a disease of older age (between 60 and 70 years). Recently, it has become increasingly common in younger age groups that are not exposed to the main risk factors. The disease develops in patients with a genetic predisposition and known environmental factors such as smoking, immunosuppressive drugs, chronic inflammation, certain viruses, and a diet low in fresh fruit and vegetables (47–49).

Association with other diseases

OLP is an immune-mediated disease and can therefore be associated with the occurrence of other diseases. Diabetes mellitus (DM) and hepatitis are considered potential diseases that may increase the risk of developing OLP. Studies on the association between DM and OLP have produced conflicting results. Studies show a variable prevalence of DM in OLP patients in different populations, ranging from 9% to 85% (50, 51), and a significant association between non–insulin-dependent DM and OLP (52). Studies by Saini et al., Ansar et al., and Bagewadi et al. show no association between DM and OLP (53–55).

The association between OLP and chronic liver disease (CLD) is well known, whereas the association with HCV infection remains controversial, but it has apparently been demonstrated in Japan and southern Europe (56). This can be explained by the higher incidence of HCV infection in the Japanese and Mediterranean populations (56). The association between HCV infection and OLP is based on the following facts: 1) antibodies against oral epithelial cells have been found in HCV-positive individuals, and 2) HCV infection can activate cytokines involved in pathogenesis (57). Data from the literature do not suggest an etiopathogenetic role of HCV in the transition from OLP to OSCC. Several studies report an increased prevalence of human papillomavirus (HPV) infection in OLP, particularly the high-risk types HPV16 and HPV18, with higher detection rates in erosive subtypes, suggesting a possible role in malignant transformation (58–62). However, this association is inconsistent across different populations and studies, and some results suggest that the association is more likely to be coincidental than causal, emphasizing the need for further prospec-

tive cohort research (63). In addition, OLP has been associated with a higher prevalence of psychological comorbidities, such as depression, anxiety, and stress. It is therefore recommended that dentists be vigilant for these conditions in OLP patients to ensure timely and appropriate referral for psychological support (64).

Treatment and management

Patient education is extremely important before starting and planning treatment. Patients should be informed in detail about the etiology of the disease, the clinical features, and the specifics of OLP treatment. They should be informed that there is an individualized therapeutic response, that a longer treatment duration is required, and that several successive different treatment regimens must be applied until adequate disease control is achieved. The therapeutic approach aims to control the inflammation and associated symptoms (pain and burning sensation). Patients should be made aware of the importance of follow-up and the link between OLP and oral cancer. The diagnosis can be very difficult even for an experienced clinician and is confirmed by histopathology. This is important for the correct therapeutic approach. The treatment of OLP depends on the form and the symptoms present. Non-erosive forms of OLP are usually asymptomatic and therefore do not require treatment. Symptomatic lesions in erosive forms of OLP require treatment, but patients should be advised that the clinical picture and symptoms of OLP will undoubtedly worsen again after the symptoms subside and treatment will be required again.

A broad spectrum of treatment options is available (Table 3). These include topical, perilesional, and systemic corticosteroids; topical retinoids; topical calcineurin inhibitors; doxycycline; hydroxychloroquine; azathioprine; mycophenolate mofetil; methotrexate; dapsone; apremilast; levamisole; thalidomide; biologics (such as efalizumab, etanercept, alefacept, rituximab, secukinumab, guselkumab, and JAK inhibitors); photodynamic therapy; low-level laser therapy; topical aloe vera; and oral curcuminoids (1, 17, 65, 66). Corticosteroids remain the first-line therapy, with topical and perilesional formulations favored for non-erosive and mildly erosive forms and systemic corticosteroids reserved for severe or refractory erosive OLP. Topical 0.1% tacrolimus is also an effective option for milder forms. Depending on the response to therapy, other immunosuppressive or immunomodulatory agents may be used. Accompanying measures such as maintaining optimal oral hygiene, treating oral candidiasis, and screening for hyposalivation are crucial for successful treatment (17, 67). Some evidence suggests that oxidative stress plays a role in pathogenesis (68). Antioxidants such as vitamins are commonly used because vitamins A and E inhibit lipid peroxidation of cell membranes, and vitamin C plays a role in stabilizing collagen structure and also helps in the regeneration of vitamin E (69). Curcumin is also an antioxidant that increases salivary and serum levels of vitamins A, E, and C (69). Vitamin D supplementation has also been shown to improve symptoms due to its anti-inflammatory and immunomodulatory properties (70). Overall, treatment should be individualized according to the severity and clinical subtype of OLP and the patient's response, with constant monitoring for possible malignant transformation and comorbidities.

Conclusions

OLP is considered a chronic inflammatory autoimmune disease with a completely unclear and complex etiopathogenetic mechanism. It is caused by genetic and environmental factors (dysregulated immune response, viruses, and stress). The diagnosis of OLP is a diagnostic challenge even for an experienced clinician and should be based on a detailed history (medical and dental), a clinical oral examination, and an HPD. OLP is an OPMD associated with oral cancer, and patients should be informed of these important facts to motivate them to follow up. Before starting treatment, patients should receive detailed information about the etiology and clinical picture of OLP as well as the individual therapeutic response (summarizing the most important information about OLP in the leaflet is recommended). The therapeutic approach must be individualized (with corticosteriod as first-line therapy) and aimed at controlling inflammation and symptoms (burning and pain).

Table 3 | **Treatment options.**

Topical therapy	Systemic therapy	Other measures
• Topical corticosteroids	• Oral corticosteroids	• Good oral hygiene
• Topical calcineurin inhibitors (e.g., cyclosporine, tacrolimus)	• Oral retinoids (e.g., acitretin or isotretinoin)	• Avoidance of irritants such as spicy or acidic foods
• Topical retinoids	• Immunosuppressants (e.g., methotrexate, azathioprine, mycophenolate mofetil)	• Smoking cessation
	• Hydroxychloroquine	• Vitamin D supplementation

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