SHORT REVIEW

Pharmaceutical Approaches in the Management of Oral Lichen Planus: A Short Review

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Abstract

Oral lichen planus (OLP) is a chronic inflammatory condition of the oral mucosa that mostly affects middle-aged women. It is often persistent, painful, and can interfere with normal functions such as eating and speaking. The exact cause remains uncertain, though immune dysregulation and local triggering factors are thought to play a key role. The present review summarizes the pharmacological options available for managing OLP. Corticosteroids continue to be the first-line treatment and are highly effective in controlling inflammation and providing symptomatic relief. When the disease does not respond adequately, agents such as calcineurin inhibitors, azathioprine, methotrexate, hydroxychloroquine, and sulfasalazine may be considered. In resistant or recurrent cases, adjunctive approaches such as photodynamic therapy, low-level laser therapy, and nutraceuticals, including aloe vera, have shown encouraging results in improving comfort and healing. Although many therapeutic choices exist, a complete cure remains challenging, and relapses are frequent. Further studies are required to identify safer and more targeted treatments that can offer sustained control and improve the quality of life of affected patients.

Key words: Corticosteroids, immunomodulators, nutraceuticals, oral lichen planus, photodynamic therapy

INTRODUCTION

A common chronic inflammatory disease of the oral mucosa is oral lichen planus (OLP). It affects approximately 0.5–2% of adults. It is more commonly found from the third to sixth decades of life.[1] It predominantly occurs in middle-aged women. Its etiology remains unclear, but factors such as medications, psychological stress, microbial agents, local and systemic cell-mediated hypersensitivity reactions, and dysregulated immune responses are believed to play contributory roles in its pathogenesis. All these factors ultimately lead to T-cell-mediated keratinocyte apoptosis and chronic mucosal inflammation.[2] This process induces mucosal damage and basal keratinocyte breakdown by $CD8^+$ T-cell activation, which is mediated by the production of tumor necrosis factor□α (TNF□α) and the expression of Fas ligand (CD95L). Immune signaling is further modulated by cytokine gene polymorphisms and interference RNAs, which prolong chronic inflammation in the oral epithelium.[3]

Six types of OLP, namely reticular, plaque-like, papular, atrophic/erosive, ulcerative, and bullous types, can be identified clinically. Buccal mucosa, tongue, and gingiva are most commonly affected.[4] The patient presents with complaints of burning sensations, discomfort, and pain, which cause difficulty in mastication, phonation, and maintaining oral hygiene.[5] Potential for malignant transformation is estimated between 0.5% and 2%.[6]

Despite the availability of numerous treatment modalities, pharmacologic management remains the cornerstone of therapy. It focuses on reducing symptoms, controlling symptoms, and preventing the progression of the disease. This review focuses on the pharmacological treatment strategies for OLP, emphasizing their efficacy, safety, and emerging targeted therapeutic options.

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FIRST-LINE THERAPY

The gold standard and first-line treatment for symptomatic management of OLP is corticosteroids. Their anti-inflammatory and immunosuppressive effects are caused by inhibition of T-cell proliferation and suppression of pro-inflammatory cytokines such as interleukin-2 and TNF-α.[7]

The preferable choice is a topical corticosteroid due to its high efficacy and low systemic absorption. The agents that are commonly prescribed are clobetasol, triamcinolone, betamethasone, fluocinonide, fluticasone, dexamethasone, and prednisolone. They are available as ointments, adhesive pastes, or mouth rinses and are used in concentrations ranging from 0.025% to 0.5% and are usually used 2–4 times a day. The side effects of topical corticosteroids include secondary candidiasis, mucosal atrophy, bad taste, oral dryness, and dyspepsia.[2]

For lesions that do not respond to topical treatment or are extensive, intralesional injection of corticosteroids is preferred. Intralesional injection of corticosteroids, such as triamcinolone acetonide, hydrocortisone, dexamethasone, and methylprednisolone is effective. Intralesional injections overcome the side effects of topical corticosteroids and provide high local concentration.[8]

In severe, widespread, or refractory cases, systemic corticosteroids are a useful treatment modality. Doses of 0.5 mg/day or 30–80 mg/kg/day of methylprednisolone or prednisone should be used for a short period up to 2 weeks and are tapered gradually to prevent adrenal suppression. A few mild and reversible side effects, such as facial edema, headaches, and muscle weakness, are reported in the literature.[9]

Retinoids and cyclosporine are also used as first-line treatment modalities. Retinoids inhibit the inflammatory mediators and reduce the keratinization of epithelial cells. Their use is limited as it causes mucosal irritation on topical application and systemic toxicity.[10] Cyclosporine is a calcineurin inhibitor; it suppresses the synthesis of pro-inflammatory cytokines and inhibits the formation of activated T cells. It remains an alternative for patients who do not tolerate or respond adequately to steroid therapy. However, due to its high cost and potential adverse effects such as rashes, parotid gland swelling, dyspepsia, and transient burning sensation, its routine use is not generally recommended.[11]

SECOND-LINE THERAPY

The lesions that do not respond to first-line treatment are treated with second-line therapy. These include topical calcineurin inhibitors, sulfasalazine, azathioprine, hydroxychloroquine, methotrexate, and mycophenolate mofetil. The primary objective is to modulate immune responses and reduce inflammatory cell activity.

An immunosuppressant, azathioprine, has been efficiently used in the management of pemphigoid and has shown favorable results in erosive OLP when administered orally for a period of 3–7 months at 1–2 mg/kg/day. Nausea, vomiting, and diarrhea, along with leukopenia, pancytopenia, and drug-induced hypersensitivity syndrome, are some side effects of azathioprine.[12]

Calcineurin inhibitors, tacrolimus and pimecrolimus, are both useful in patients who do not respond to corticosteroids. T-cell activation and cytokine release are both inhibited by their action. Tacrolimus 0.03–0.1% or pimecrolimus 1% are topically applied for 4–8 weeks twice daily and reduce erythema and ulcers noticeably. A significant burning sensation is caused at the application site during the initial application phase. Recent research indicates that short-term use is safe and well-tolerated when patients are regularly monitored, although early studies raise concern about potential malignant transformation.[13]

A folate antimetabolite, methotrexate, has anti-inflammatory actions and has shown a response in patients with refractory OLP. It can be administered orally or subcutaneously at a dose of 10–15 mg/week. Elevation of liver enzymes, decrease in leukocyte count, and alopecia are some of its severe complications.

The U.S. Food and Drug Administration approved the antirheumatic drug sulfasalazine, which is commonly used in inflammatory diseases and autoimmune conditions. It can be used systemically and topically at a dose of 2.5 g/day and 30 mg/5 mL for 6–8 weeks, respectively, for effective results. Mild gastrointestinal disturbances are its side effects.[14]

An anti-malarial agent popularly used for immunomodulatory effects is hydroxychloroquine sulfate. Its effective and safe dose is 200–400 mg/day. It might cause ocular toxicity, hence ophthalmologic evaluation is required in long-term use.[15]

Mycophenolate mofetil suppresses lymphocyte proliferation; it is an inhibitor of inosine monophosphate dehydrogenase. It is considered a substitute for azathioprine due to its safety profile and fewer hematologic complications. It can be used to manage refractory erosive lesions.[2]

THIRD-LINE THERAPY

For patients with refractory or recurrent OLP that do not respond to conventional corticosteroid and immunosuppressive therapy, numerous advanced pharmacological and adjunctive modalities have been explored. Third-line therapy is used. Low-level laser therapy (LLLT), photodynamic therapy (PDT), and nutraceuticals are included in third-line therapy.

LLLT is a non-invasive and non-pharmacological treatment for OLP. Helium neon (633 nm), ruby (694 nm), and argon

(488 and 514 nm) are used to stimulate fibroblasts and the epithelial cells, and thus promote wound healing.[16] A minimally invasive approach that does not leave scars is PDT. Photosensitizers such as methylene blue and toluidine blue are commonly used to destroy the proliferated inflammatory cells. After application of photosensitizers for 10 min, the area is exposed to light at a wavelength of 630–660 nm for approximately 2 min. PDT has shown equivalent effectiveness to topical corticosteroids with minimal side effects, including transient erythema or burning.[17]

Aloe vera gel, lycopene, and topical tocopherol are nutraceuticals used as supportive third-line treatment for managing OLP due to their antioxidant and anti-inflammatory properties. Among these, *A. vera* gel has shown efficacy comparable to triamcinolone acetonide 0.1%, significantly reducing pain and mucosal inflammation with excellent tolerability. It may be used alone in mild cases or as an adjunct to corticosteroids in erosive forms of OLP, offering a safe and well-accepted complementary approach to conventional therapy.[2]

CONCLUSION

OLP is a chronic inflammatory condition of the oral mucosa that often requires long-term management and follow-up. The main goal of treatment is to relieve symptoms, control inflammation, and prevent recurrence or malignant transformation. Corticosteroids are the most effective and the choice of drugs for initial management. In patients where lesions do not respond, immunomodulators such as methotrexate, azathioprine, and calcineurin inhibitors are useful substitutes. Third-line therapies such as LLLT, PDT, and nutraceuticals offer supplementary relief, especially for patients with refractory or resistant lesions. Although these newer approaches show promising results, more research is needed to establish their long-term effectiveness. A better understanding of disease mechanisms may lead to targeted and individualized therapies.

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