

A review on various treatment modalities for oral lichen planus

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ABSTRACT

Introduction: Oral lichen planus (OLP) is a chronic inflammatory, autoimmune mucocutaneous disease. It affects the skin, mucous membrane, nails, and hair. It is seen in 1–2% of populations. It is slightly more prevalent in females than in males. Corticosteroids are the mainstay of OLP. Due to its side effects, various treatment modalities have been used to treat OLP. **Aim:** The aim of this review is to provide awareness and update on the development of the treatment of OLP. **Results:** An electronic search was conducted through many websites and databases to analyze the various treatment modalities for lichen planus. The studies were reviewed and compared. This review article summarizes the treatment modalities carried out for the OLP. **Conclusion:** OLP has been researched widely as it has been a potentially malignant disorder. Recent advancements in the treatment have been considered to reduce the side effects.

KEY WORDS: *Aloe vera*, Curcumin, Green tea, Oral lichen planus, Photodynamic therapy

INTRODUCTION

Lichenoid reactions represent a family of lesions with different etiologies with common clinical and histological appearances. Histological evidence may be used to differentiate lichenoid reactions from the other oral pathologies. Oral lichenoid reactions include:

1. Lichen planus
2. Lichenoid contact reactions
3. Lichenoid drug eruptions
4. Lichenoid reactions of graft versus host disease.

Oral lichen planus (OLP) is a chronic inflammatory mucocutaneous disease which frequently involves the oral mucosa. This disease is more prevalent in females than in males. It has been reported mostly in middle-aged patients and is rare in children. There is a confirmed relationship between cancer and OLP. However, the degree of the risk to develop into cancer could not be determined. Hence, OLP should be classified as a precancerous lesion. Many cases

of OLP are asymptomatic and identified on routine checkup.

Andreasen (1968) classified OLP into six clinical forms

- Reticular
- Papular
- Plaque-like
- Atrophic
- Erosive
- Bullous.

OLP can occur on any mucosal surface but mostly predominant in tongue and posterior buccal mucosa. Lesions may change their forms during the course of the disease. There is no cure for OLP. Studies show that a large number of agents administered to treat OLP report to be inadequate in controlling the disease.

OLP is also of special importance due to its potential to transform into a malignant disorder.^[1] The precancerous nature of OLP is still not settled, but patients must be carefully evaluated and observed. Presently, this condition is categorized as a “probable precancerous condition.”

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CLINICAL MANIFESTATIONS OF OLP

In about 15% of the patients, the cutaneous lesions may present. The skin lesions consist of pruritic erythematous papules that are flat-topped. They commonly appear in the trunk and flexor surfaces of the arms and legs. As per the patient's reports, intense scratching may relieve the itching and pain, but the trauma may aggravate the disease. This is called as Koebner phenomenon.^[2] This may also be relevant to the OLP which is exposed to the trauma due to brushing and mastication.

MANAGEMENT OF OLP

Since the etiologies of OLP are not known the therapy mainly focuses on palliative care, which involves the treatment strategies to reduce or eliminate the symptoms. Various treatment options are available for OLP. However, topical steroids are the primary treatment choice. Some studies have reported about the usage of potent steroids like clobetasol propionate in favor of steroids like triamcinolone acetonide. Topical application of cyclosporine tacrolimus and retinoids has been used as a second line of therapy. The management of OLP involves various treatment options which vary in their potency based on their action against OLP.

DIFFERENT KINDS OF TREATMENT MODALITIES FOR OLP

Corticosteroids

Corticosteroids are the first line of treatment for OLP. They act by decreasing the cell-mediated immune response thereby modulating the immune function. The topical corticosteroids are used in treating mild or moderate lesions. These include triamcinolone acetonide 0.1%, 0.05% fluocinonide, and 0.025% clobetasol propionate. Topical aqueous triamcinolone acetonide suspension is effective in relieving the erythematous lesion and ulcerations. About 0.1% aqueous triamcinolone acetonide was widely used as an oral rinse for OLP. According to a report by Vincent *et al.*^[3] 1990, this method proved to be effective in 27 patients out of 46 patients with symptomatic OLP.

In a study conducted in 22 patients with OLP, 0.05% fluocinonide ointment in a oral base paste improved 60% of the symptoms after 2 weeks usage. Fluocinonide is an adhesive base. It is considered to be safe and effective in the treatment of OLP. In a study by Silverman *et al.*^[4] 0.025% clobetasol propionate is more effective in treating the OLP and other chronic oral lesions. It is an effective topical steroid comparing other topical and systemic drugs.

The advantage of using topical steroid is that it reduces the risks and side effects caused when using a systemic drug administration. It may cause some adverse effects such as secondary candidiasis, thinning of the oral mucosa, and discomfort on application areas. Prolonged use of potent topical corticosteroids with occlusal dressing can cause adrenal suppression. Initially, hydrocortisone hemisuccinate pellets were used in treating OLP.^[5-7] As per the studies by Cooke, 1965, they were exhibiting some relief in erosive OLP. However, Cawson,^[7] 1968, showed that only 3 out of 28 patients receiving the hydrocortisone treatment improved after 6 months of usage.

Betamethasone valerate is a more potent anti-inflammatory agent. It showed dramatic results in patients with OLP. In a double-blind study conducted by Cawson^[7] in 1968, he showed that in 8 patients out of 30 with symptomatic OLP treated with betamethasone (0.1 mg) pellets, all lesions disappeared in 1 month. Furthermore, 20 of 30 patients showed substantial improvement. Two patients failed to respond to this treatment. Similarly, Tyldesley and Harding^[8] showed that betamethasone valerate aerosol fitted with a special intraoral adaptor was an excellent treatment in the majority of 23 patients tested in a double-blind study. Greenspan *et al.*^[9] confirmed the efficacy of both betamethasone valerate aerosol and pellets in a double-blind study, noting improvement in 17 of 19 patients.

Nowadays, recent advancements showed the usage of fluorinated highly potent corticosteroids in the management of various cutaneous diseases. The anti-inflammatory action exhibited by these potent corticosteroids on the topical areas has been assessed by the vasoconstrictor assay predicting therapeutic efficacy, and it showed that these are highly potent than the aforementioned corticosteroids. Recently, their use for oral vesiculoerosive diseases, including OLP, has become popular.

A study by Conklin and Blasberg *et al.*^[10] in 1987 has demonstrated the use of clobetasol in severe cases of OLP. However, their safety and controlled studies have not been documented. Topical corticosteroids appear to be safe when applied to mucous membranes. In 1969, Lehner and Lyne^[11] measured the plasma cortisol levels before and after topical application of corticosteroids in patients with OLP. The results showed that there was no adrenal suppression in 17 patients who applied 0.4 mg of betamethasone valerate daily to their oral lesions for several months. In another study by Kutcher in 1966, triamcinolone acetonide in doses up to 480 mg was found to produce no adverse effects. However, he did not measure the plasma cortisol levels.

Plemons *et al.* displayed that the use of fluocinonide gel in patients with OLP lesions was safe and those who applied 1.5 g daily to OLP lesions for 3 weeks showed no marked adrenal suppression. However, the prolonged use of topical steroids on OLP lesions dictates regular follow-up examinations.

The adrenal suppression may be caused by the prolonged usage of a potent corticosteroid to a compromised mucosal barrier. Not all corticosteroids are safe.

According to a study conducted by Lehner and Lyne in 1969,^[11] betamethasone disodium phosphate applied to oral lesions caused adrenal suppression in 8 out of 10 patients. Betamethasone valerate aerosol in the form of Valisone can be harmful when applied to mucous membranes as per the study by Beckman, 1981.^[12] The chronic usage of topical corticosteroids can cause candidiasis in some patients. Cawson^[7] reported the occurrence of candidiasis in 4 out of 30 OLP patients undergoing treatment with betamethasone valerate. Candidiasis was reported to have developed in 3 out of 24 patients treated with clobetasol. About 31% of patients treated with triamcinolone acetonide suspension developed secondary candidiasis according to Vincent *et al.*^[3] 1990.

This development of secondary candidiasis is due to prolonged contact of corticosteroids with the oral cavity, thus reducing the immune activity in those regions. This is not limited to OLP, but it causes all kinds of oral lesions. Hence, patients with chronic OLP requiring chronic corticosteroid therapy should have candidal culture performed for them before the usage and periodically while continuing the treatment.

The response to the corticosteroid treatment is varying in various patients. The topical steroids do not relieve pain or symptoms in all patients. According to a study by Silverman *et al.*^[4] 1991, the most potent corticosteroid, clobetasol propionate, failed to produce any benefit in 2 of 9 patients treated for OLP. The frequency of usage of topical corticosteroids up to 6 or 7 times a day makes compliance difficult. The optimal effects can only be achieved when they are applied between 5 and 10 times daily as reported by Silverman *et al.*^[4,5]

INTRA LESIONAL STEROID THERAPY

Chronic localized lesions can be treated with 0.20–0.40 ml of triamcinolone acetonide in the concentration of 10 mg/ml. It produces effective results. Even though it is painful, it increases the drug delivery to the affected site and minimizes the drug absorption into the system. Muscular atrophy is the common side effect associated with this treatment.

In 1967 Sleeper^[13] reported that the topical corticosteroids were inadequate in the treatment of OLP and proposed therapy with intralesional triamcinolone acetonide suspension. In his study, he concluded that seven patients who received 5–7 mg of triamcinolone injected into lesions had shown relief of symptoms within 2 weeks, and three showed complete healing of the lesions, and the remaining four showed remarkable improvement clinically. The benefits of intralesional corticosteroids are now well known and described according to Randall and Cohen.^[16] Dusek and Frick^[14] recommended using the local anesthetics like lidocaine to lessen the pain of the injections.

Zegarelli^[15] proposed the use of topical and weekly intralesional corticosteroids in combination. He conducted a study of seven patients. After 3 weeks, five patients were found to have improved 100% clinically. It is noted that remission would occur after several months in most cases. However, the recurrences were milder than the previous disease state, and it could be managed with topical agents.

There are few contraindications to intralesional corticosteroids. Atrophy of tissue and secondary candidiasis are complications that may occur locally. Three–four weekly or biweekly treatments of intralesional triamcinolone acetonide in appropriate doses is shown to have good effects on OLP and is regarded as a safe approach.

SYSTEMIC STEROID THERAPY

Systemic steroids are mostly used for moderate to severe OLP. It can also be used in cases resistant to topical therapy. The most commonly prescribed systemic steroid to manage OLP is prednisone. Systemic steroids are used in high doses and in short-course to maximize the effect on OLP lesions. It has to be ensured that the side effects should be minimized. The dosage should be determined depending on the severity of the lesion and the size of the patient.

As we are using this therapy in short terms, the risk of the hypothalamic-pituitary-adrenal axis suppression is negligible. The side effects may include insomnia, diarrhea, mood swings, nervousness, fluid retention, muscle weakness, hypertension, and decreased resistance to infection. An excellent response will be observed in the majority of patients undergoing systemic prednisone therapy. When the disease is controlled, a topical agent should be employed for maintenance.

In the study by Silverman *et al.*^[4,5] a much higher percentage of patients achieved a symptom-free state with topical corticosteroid alone, then with either systemic corticosteroid or a combination of systemic and topical corticosteroids.

Azathioprine acts as an analogous to prednisone to reduce inflammation. It lowers the doses prednisone. Possible side effects include nausea, vomiting, diarrhea, pancreatitis, bone marrow suppression, hepatotoxicity, arthralgias, and retinopathy.

All patients cannot be managed with corticosteroids alone. Topical therapy should be maintained until symptoms and clinical findings improve. The gels tend to sting and burn, but the ointments do not cause burns. The gels adhere to the oral mucosa more easily than ointments. The clinical response is same for both the gel and ointment. Non-responding ulcerations can be treated with corticosteroids. Systemic corticosteroids should be reserved for acute exacerbations characterized by multiple ulcerations or widespread disease.

Zegarelli^[15] compared various treatment choices of corticosteroids. A slightly greater improvement was noted when all three modalities were used in individual patients. The prone to develop candidiasis was also increasing in these patients when combination therapy was used.

IMMUNOSUPPRESSANT AGENTS IN OLP

Tacrolimus

Tacrolimus is a macrolide immunosuppressant with a mechanism of action similar to cyclosporine. Tacrolimus is more potent than cyclosporine and it has better mucosal penetrating properties. Olivier *et al.*^[17] suggested that the low concentration of topical tacrolimus in distilled water produced a rapid palliating effect in patients with erosive OLP. In his study, Stoopler *et al.*^[18] reported that topical use of tacrolimus is a safe, well-tolerated, and effective therapy for OLP lesions.

Cyclosporine

Cyclosporine is an immunosuppressant and inhibits the production of lymphokines. Francès *et al.*^[19] conducted a study using cyclosporine, which resulted in improvement in all four patients treated with 100 mg of topical cyclosporine for 1 month. The blood levels of cyclosporine were relatively low in all patients. Balato *et al.*^[20] confirmed Francès *et al.*^[19] results with topical cyclosporine. Four out of 7 patients showed complete healing of all erosions, and three patients showed 40%–80% re-growth in ulcer areas.

Voûte *et al.*^[21] found that the clinical effect of cyclosporine was same as using topical corticosteroid. There was no distinct difference. Cyclosporine can be used as an alternative therapy in the control of OLP. This drug is expensive and would result in side effects such as hypertension and nephrotoxicity. This limited the usage of this drug in the treatment of OLP.

Pimecrolimus^[39]

It acts by inhibiting the T-cell activation by inhibiting the synthesis of inflammatory cytokines and mediators from mast cells. It has been used as the treatment for OLP. It has a significant anti-inflammatory activity and immune-modulatory capabilities with low systemic immunosuppressive potential.

Levamisole^[40]

It is an effective immune-modulating agent that can restore the normal phagocytic activity of macrophages and neutrophils. The levamisole is an effective drug in patients who have not responded to conventional treatments. It has an adverse effect such as nausea, vomiting, headache, and agranulocytosis.

Retinoids

In a study by Günther *et al.*^[22] they conducted an open, uncontrolled trials, Vitamin A acid was applied locally to white, reticulated lesions and concluded with good results. Patients had become asymptomatic and did not develop the erosive disease in these studies. The most widely used topical retinoid for the treatment of OLP is tretinoin. Tretinoin is a metabolite of Vitamin A.

Sloberg *et al.*^[23] reported that after 2 months of therapy with this agent, 71% of atrophic and erosive lesions “improved,” whereas the remaining 29% of lesions were “unchanged” or “worse.” Side effects included increased soreness and redness. Relapses occurred within 3 months of discontinuing therapy.

Giustina *et al.*^[24] in 1986 used 0.1% isotretinoin gel in the treatment of 20 patients with OLP. In this double-blind study, patients were instructed to apply the gel twice daily for 2 months and were evaluated. Five out of 6 patients had complete resolution with therapy. All patients who received placebo improved only after switching to therapy with active medication. Handler^[25] reported beneficial results with systemic isotretinoin for OLP.

Camisa and Allen^[26] performed a study in six patients with OLP. During the 8 weeks of treatment with isotretinoin, it is found that the patients were not completely cleared of this disease, but all patients were reported to have significant improvement of symptoms. Four out of 5 patients improved clinically. However, the authors concluded that the isotretinoin was of minimal to negligible benefit with common side effects, such as cheilitis, dry skin, headache, and rashes. According to Stuttgart^[27] 1975, the systemic use of tretinoin has beneficial results in OLP, but its toxicity and side effects limit its use.

Etretinate is a Vitamin A analog with a better therapeutic index than Vitamin A acid. It was found to be efficacious in the treatment of OLP by Schuppli^[28]

in 1978. The author concluding the study compelled on a fact that this therapy should become the first drug of choice in the management of erosive lichen planus.

Gorsky and Raviv^[29] reported that their results had also shown that the etretinate should become the first-line therapy for OLP. The authors showed that all patients improved in clinical signs and symptoms using etretinate. Nearly half of the patients achieved an asymptomatic state after undergoing treatment with etretinate. Due to its adverse effects, one patient withdrew from the study. The erosive lesions disappeared in these patients which is a major clinical improvement resulted using this agent. These results supported Sloberg's^[23] conclusions and his proposal of higher efficacy of etretinate in the treatment of OLP.

Side effects of etretinate included cheilitis, pruritus, desquamation of hands and feet, paronychia, and hair loss. Hence, the use of etretinate should be employed for refractory cases unresponsive to corticosteroids. There were very few cases of relapses, and erosive lesions healed on more than 4–6 months of therapy. Various researches have been employed nowadays to evaluate the effects and optimal dose of this promising new medication.

Griseofulvin

Griseofulvin in the treatment of OLP lesions has been approached only when the steroid treatment is contraindicated or when the lesions are resistant to steroids. Aufdermorte *et al.* found it to be effective in all the 3 patients he tested, whereas Bagan *et al.* did not find any improvement in 7 patients when he administered the same dosage of griseofulvin as of Aufdermorte to the 7 patients for 8 weeks.

Sehgal *et al.*^[30] reported improving results using griseofulvin therapy in cutaneous lichen planus in a double-blind study. Massa and Rogers^[31] examined 11 patient's files, in which, 6 patients showed marked improvement within 3 weeks of study. It also showed remission within 6 months of treatment. In another group of 15 patients with lichen planus, patients exhibited both cutaneous and oral lesions. However, 4 patients showed improvement of their oral lesions with griseofulvin. Hence, the author concluded with inconclusive evidence.

According to Naylor^[32] the reports showed no marked improvement in 4 patients with erosive OLP treated with griseofulvin. Until double-blind studies are performed confirming its efficacy, the use of griseofulvin in the treatment of OLP should be limited.

Dapsone^[40]

In resistant cases of erosive OLP, dapsone is an effective drug which has both anti-inflammatory and immune-modulatory effects. It is available as a gel for

topical application and systemically in tablet forms. Headache and hemolysis are the major side effects of dapsone.

Interferon^[40]

The topical application of human fibroblast interferon gel and interferon-alpha has suggested to improve erosive OLP.

Puva Therapy^[40]

Photosensitizing psoralen drugs and ultraviolet A (UV) radiation were introduced as a new therapy by Jansen *et al.* in 1987 for oral mucosal lesions. The photosensitizing drugs can either be administered systemically or applied topically before irradiation. Four psoralens are used in PUVA therapy – psoralen, methoxy psoralen (Bergapten), methoxypsoralen (methoxsalen), and trimethyl psoralen (trioxsalen). UV irradiation in combination with psoralens modulates the function of cells of the immune system.

Photodynamic Therapy

Photodynamic therapy^[33] uses a photosensitizing compound like methylene blue which is activated at a specific wavelength of laser light. It is known to destroy the targeted cell through strong oxidizers, leading to membrane lysis, cellular damage, and protein inactivation.

Diseases such as psoriasis and lichen planus are characterized by the hyper-proliferation of inflammatory cells. PDT have immune-modulatory properties which may induce apoptosis in the hyper-proliferating inflammatory cells which in turn reverses the hyper-proliferation and inflammation of lichen planus.

Surgical Management

Surgical treatment is employed mostly in the plaque-like lesions. The affected surface epithelium can be removed easily. It may also be recommended in the management of non-healing lesions. Surgical management^[34] should be done in the cases of the erosive and atrophic types because the surface epithelium is eroded. Cryosurgery^[35] and carbon dioxide (CO₂) laser therapies^[34] have been tried in the management of OLP lesions.^[35-40] In spite of several trials surgical treatment is not recommended due to the recurrence of inflammation. Trauma from surgical procedures may induce new lesions as proposed in the Koebner phenomenon.^[2]

CONCLUSION

OLP patients should be approached with different treatment regimens according to their conditions. The patients should be counseled depending on their chronic stage. The results of the efficacy of the

treatment modalities are inconclusive and insignificant. Corticosteroids are the most common treatment in managing the OLP lesions. As it is highly probable to transform into a malignant disorder, patients should be advised to reassess periodically.

REFERENCES

1. Thaminee S. Oral lichen planus and its malignant transformation. *J Pharm Sci Res* 2016;8:1226-8.
2. Ismail SB, Kumar SK, Zain RB. Oral lichen planus and lichenoid reactions: Etiopathogenesis, diagnosis, management and malignant transformation. *J Oral Sci* 2007;49:89-106.
3. Vincent SD, Fotos PG, Baker KA, Williams TP. Oral lichen planus: The clinical, historical, and therapeutic features of 100 cases. *Oral Surg Oral Med Oral Pathol* 1990;70:165-71.
4. Silverman S Jr., Gorsky M, Lozada-Nur F, Giannotti K. A prospective study of findings and management in 214 patients with oral lichen planus. *Oral Surg Oral Med Oral Pathol* 1991;72:665-70.
5. Chainani-Wu N, Collins K, Silverman S Jr. Use of curcuminoids in a cohort of patients with oral lichen planus, an autoimmune disease. *Phytomedicine* 2012;19:418-23.
6. Silverman S Jr., Gorsky M, Lozada-Nur F. A prospective follow-up study of 570 patients with oral lichen planus: Persistence, remission, and malignant association. *Oral Surg Oral Med Oral Pathol* 1985;60:30-4.
7. Cawson RA. Treatment of oral lichen planus with betamethasone. *Br Med J* 1968;1:86-9.
8. Tyldesley WR, Harding SM. Betamethasone valerate aerosol in the treatment of oral lichen planus. *Br J Dermatol* 1977;96:659-62.
9. Greenspan JS, Yeoman CM, Harding SM. Oral lichen planus. A double-blind comparison of treatment with betamethasone valerate aerosol and pellets. *Br Dent J* 1978;144:83-4.
10. Blasberg B, Dorey JL, Stein HB, Chalmers A, Conklin RJ. Lichenoid lesions of the oral mucosa in rheumatoid arthritis patients treated with penicillamine. *J Rheumatol* 1984;11:348-51.
11. Lehner T, Lyne C. Adrenal function during topical oral corticosteroid treatment. *Br Med J* 1969;4:138-41.
12. Beckman BI. Valisone aerosol spray contraindicated in mucous membranes. *J Am Acad Dermatol* 1981;4:233.
13. Sleeper HR. Intralesional and sublesional injection of triamcinolone acetonide for oral lichen planus. *Yale J Biol Med* 1967;40:164-5.
14. Dusek JJ, Frick WG. Lichen planus: Oral manifestations and suggested treatments. *J Oral Maxillofac Surg* 1982;40:240-4.
15. Zegarelli DJ. Treatment of oral lichen planus with topical vitamin A acid. *J Oral Med* 1984;39:186-91.
16. Randell S, Cohen L. Erosive lichen planus. Management of oral lesions with intralesional corticosteroid injections. *J Oral Med* 1974;29:88-91.
17. Olivier V, Lacour JP, Mousnier A, Garraffo R, Monteil RA, Ortonne JP, *et al.* Treatment of chronic erosive oral lichen planus with low concentrations of topical tacrolimus: An open prospective study. *Arch Dermatol* 2002;138:1335-8.
18. Stoopler ET, Sollecito TP, DeRossi SS. Oral lichen planus. Update for the general practitioner. *N Y State Dent J* 2003;69:26-8.
19. Francès C, Boissic S, Etienne S, Szpirglas H. Effect of the local application of ciclosporine A on chronic erosive lichen planus of the oral cavity. *Dermatologica* 1988;177:194-5.
20. Balato N, De Rosa S, Bordone F, Ayala F. Dermatological application of cyclosporine. *Arch Dermatol* 1989;125:1430-1.
21. Voûte AB, Schulten EA, Langendijk PN, Kostense PJ, van der Waal I. Fluocinonide in an adhesive base for treatment of oral lichen planus. A double-blind, placebo-controlled clinical study. *Oral Surg Oral Med Oral Pathol* 1993;75:181-5.
22. Günther SH. Vitamin A acid in treatment of oral lichen planus. *Arch Dermatol* 1973;107:277.
23. Hersle K, Mobacken H, Sloberg K, Thilander H. Severe oral lichen planus: Treatment with an aromatic retinoid (etretinate). *Br J Dermatol* 1982;106:77-80.
24. Giustina TA, Stewart JC, Ellis CN, Regezi JA, Annesley T, Woo TY, *et al.* Topical application of isotretinoin gel improves oral lichen planus. A double-blind study. *Arch Dermatol* 1986;122:534-6.
25. Handler HL. Isotretinoin for oral lichen planus. *J Am Acad Dermatol* 1984;10:674.
26. Camisa C, Allen CM. Treatment of oral erosive lichen planus with systemic isotretinoin. *Oral Surg Oral Med Oral Pathol* 1986;62:393-6.
27. Stüttgen G. Oral vitamin A acid therapy. *Acta Derm Venereol Suppl (Stockh)* 1975;74:174-9.
28. Schuppli R. The efficacy of a new retinoid (Ro 10-9359) in lichen planus. *Dermatologica* 1978;157 Suppl 1:60-3.
29. Gorsky M, Raviv M. Efficacy of etretinate (Tigason) in symptomatic oral lichen planus. *Oral Surg Oral Med Oral Pathol* 1992;73:52-5.
30. Sehgal VN, Abraham GJ, Malik GB. Griseofulvin therapy in lichen planus. A double-blind controlled trial. *Br J Dermatol* 1972;87:383-5.
31. Massa MC, Rogers RS 3rd. Griseofulvin therapy of lichen planus. *Acta Derm Venereol* 1981;61:547-50.
32. Naylor GD. Treating erosive lichen planus with griseofulvin: A report of four cases. *Quintessence Int* 1990;21:943-7.
33. Mostafa D, Tarakji B. Photodynamic therapy in treatment of oral lichen planus. *J Clin Med Res* 2015;7:393-9.
34. Horch HH, Gerlach KL, Schaefer HE. CO₂ laser surgery of oral premalignant lesions. *Int J Oral Maxillofac Surg* 1986;15:19-24.
35. Loitz GA, O'Leary JP. Erosive lichen planus of the tongue treated by cryosurgery. *J Oral Maxillofac Surg* 1986;44:580-2.
36. Saawarn N, Shashikanth MC, Saawarn S, Jirge V, Chaitanya NC, Pinakapani R, *et al.* Lycopene in the management of oral lichen planus: A placebo-controlled study. *Indian J Dent Res* 2011;22:639-43.
37. Chainani-Wu N, Madden E, Lozada-Nur F, Silverman S Jr. High-dose curcuminoids are efficacious in the reduction in symptoms and signs of oral lichen planus. *J Am Acad Dermatol* 2012;66:752-60.
38. Patil S, Khandelwal S, Sinha N, Kaswan S, Rahman F, Tipu S. Treatment modalities of oral lichen planus: An update. *J Oral Diagn* 2016;1:47-52.
39. Lavanya N, Jayanthi P, Rao UK, Ranganathan K. Oral lichen planus: An update on pathogenesis and treatment. *J Oral Maxillofac Pathol* 2011;15:127-32.
40. Gujjar P, Zingade J, Patil S, Hallur J. Recent update on treatment modalities of oral lichen planus-a review. *IJSS Case Rep Rev* 2015;2:40-4.

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