Review article

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Oral lichen planus and its recent management: A review

Iqbal M. A. [1*], Yesmin S [2], Maaisha F[3], Ibrahim S[4], Gotame P[5]

AFFILIATION:

1. **Dr. Md. Ashif Iqbal**
   Associate Professor,
   Department of Periodontology & Oral Pathology
   Update Dental College, Dhaka, Bangladesh
2. **Dr. Suraiya Yesmin**
   Lecturer
   Department of Periodontology & Oral Pathology
   Update Dental College, Dhaka, Bangladesh
3. **Dr. Fathimath Maaisha**
   Intern Doctor
   Department of Periodontology & Oral Pathology
   Update Dental College, Dhaka, Bangladesh
4. **Dr. Shaama Ibrahim**
   Intern Doctor
   Department of Periodontology & Oral Pathology
   Update Dental College, Dhaka, Bangladesh
5. **Dr. Puja Gotame**
   Intern Doctor
   Department of Periodontology & Oral Pathology
   Update Dental College, Dhaka, Bangladesh

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*** Corresponding Author**
Dr. Md. Ashif Iqbal
Associate Professor
Dept. of Periodontology & Oral Pathology
Update Dental College & Hospital,Dhaka, Bangladesh
E-mail: drasif100@gmail.com
Cell Phone: +88-01716116080
ORCID Id: http://orcid.org/0000-0002-9490-7927

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ABSTRACT:

Background: Oral Lichen Planus (OLP) is one of the most common dermatological disease which is present in the oral cavity. It is a chronic autoimmune, mucocutaneous disease that affects oral mucosa as well as the skin, genital mucosa and other sites of the body.Method: In this review study, various databases such as Google Scholar, PubMed Central, Hinari and Cochrane library were searched for articles with keywords lichen planus, oral lichen planus, premalignant lesions, management of Lichen planus. Articles were searched from January 2015 to 5th November 2020.Result: From the 34 articles obtained after reviewing the abstracts, most relevant 32articles were evaluated in this study.Conclusion: The etiology, pathophysiology, clinical presentation, histopathological features, diagnosis and various management for oral lichen planus is discussed. This article also compares the existing and the most recent treatment modalities that are available throughout the world that are discussed in the literatures. However, more intensive studies must be carried out to find the best treatments which are cost-effective in the long run.

KEYWORDS: lichen planus, oral lichen planus, premalignant lesions, Management of oral Lichen planus

INTRODUCTION:

Oral lichen planus is an auto immune chronic mucocutaneous disease[1].The prevalence rate of OLP is reported as 0.5–2.2% with a malignant transformation rate of 1.09% [2]. It usually affects middle-aged women but occurs in both sexes with a female to male ratio of 1.4:1[3-4]. Lichen planus can affect the skin, hair, scalp, esophagus, nails, genital areas and oral mucosa[3,5]. Oral lichen planus can be seen in the mucous membrane of buccal mucosa, tongue, palate and tongue[6].OLP is commonly seen as symmetrical and bilateral lesions with multifocal involvement in the oral mucosa. It can also show periods of recurrence and recover[7] .There are six clinical patterns of oral lichen planus. They are reticular, plaque-like, atrophic, erosive/ulcerative, papular and bullous form[8]. But the most common is reticular, papular and plaque forms which are usually painless and similar to other white disorders such as leukoplakia, and appears as white hyperkeratotic striae or plaques[4]. Erosive and atrophic forms can cause discomfort, pain, burning sensation and intolerance to spicy and hot food[9]. Also long lasting form of erosive OLP is associated with a significant potential for malignant transformation with an estimated risk of 0.5-2%[10].Smoking and alcohol consumption can also aggravate the chance of malignant transformation[6]. The exact pathogenesis of oral lichen planus is unclear. But some researchers suggests that- lymphocyte infiltration that triggers inflammatory responses of both the lamina propria and epithelial layer, leads to keratinocyte apoptosis in oral epithelium and cause the initiation and development of oral lichen planus[6]. Also, the etiology of oral lichen planus is unknown but it is apparent that autoimmune response, infection, hypersensitivity reactions and mental pressure can involve in its onset[9].

Prominent lymphocyte penetration at the epithelium connective tissue interface, hyperparakeratosis, cytoid bodie, acanthosis and hydropic epithelial layer transition are some histopathologic characteristics of OLP [2, 9]

So far, OLP appears to be an incurable disease, and most clinical practice focuses on inflammation control and reduction[9,11]. Despite multiple treatment modalities, most cases of OLP appear to be more resistant to treatment, such as topical steroids(considered as the first line of treatment), systemic steroids, retinoids, calcineurin inhibitors, lasers, phototherapy, immunosuppressants, natural agents such as curcumin, aloe vera, vitamin A, laser biomodulation.Although there are many proposed treatment modalities, a permanent cure is not yet available. Unfortunately, most of the therapeutic modalities are associated with adverse effects; some of them are serious, which limits their use[11]. The main aim of this article is to combine the latest information and findings regarding oral lichen planus

ETIOLOGY

The specific etiology of oral planus lichen is uncertain. But some potential initial triggering factors which determine keratinocytes destruction are considered to be the following: such as, inducers of cell-mediated hypersensitivity, autoimmune response to local antigens, stress, and microorganisms[12].The manifestation of disease plays an important role in genetic and environmental factors ,

such as anxiety, pathogens (e.g. HPV16, HSV17), & changes in the mucosal microbioma (e.g. Candida species, various other bacteria)[1] Medical conditions such as auto immune disease, diabetes, chronic liver disease, intestinal diseases, increases cholesterol level, hypertension and infections can also cause onset of oral lichen planus[13.]

Systemic medicines can also contribute to the production of oral lichenoid reactions (OLR) and lichen planus, such as nonsteroidal antiinflammatory drugs, antihypertensives, and oral hypoglycemics. Amalgam, copper , and nickel from dental restorative content can also be related to the localization of OLR in some patients[14]

The table below briefly describes some of the etiological features of oral lichen planus.

Etiology	Description
Genetic background	Genetic polymorphism of cytokines, IFN-gamma, increase in frequency of 308A TNF-alpha allele [SKIN]
Psychosocial factors	Stress and anxiety Increase in salivary cortisol and stress
Trauma	Koebner phenomenon
Systemic reactions	HCV Hypertension, diabetic (Grinspan syndrome) Thyroid dysfunction GVHD
Oral lichenoid reactions	Dental materials systemic medications

PATHOPHYSIOLOGY

While various studies have shown that OLP is a widely recognized chronic inflammatory autoimmune response-driven disorder, the precise pathogenesis remains unknown[1,5,6,15-18]. Some researchers found that emotional stress, infection and hypersensitivity contribute to OLP onset [1,6,19]. There has also been reported genetic vulnerability by haplotypes, such as HLA – A3, -A5, -A28, -B8, -B16, -Bw35, -B7, -B18, -Aw19, -Cw8 associated with different variants of LP[1].

OLP is considered a T cell-mediated autoimmune disorder in which apoptosis of basal epithelial cells is caused by clusters of differentiation 8 (CD8)+ T cells. Its function is uncertain, but in the exacerbation and continuation of the oral lichen planus, a cytokine complex network(such as TNF- α, IFN-γ, TGF-β, IL-1, 2, 4, 5, 6, 8, 10, 12, 17, 18, and IL-22) plays an significant role[5, 14, 18, 20-22]. Other researchers suggest that the cell-mediated immune system starts with the expression of keratinocytes antigen; this step is accompanied by the movement of T cell lymphocytes directly activated by an antigen binding to the main histocompatibility complex (MHC)-1 on keratinocytes or activated CD4 + lymphocytes[13].In exchange, the activated CD8 + T cells kill the basal keratinocytes by tumor necrosis factor (TNF)-alpha, Fas-FasL-mediated or granzyme B-activated apoptosis[13]. Researchers conclude that oxidative stress influences molecules and pathways involved in the recruitment of lymphocytic infiltrates in OLP lesions and apoptosis induction, including ICAM-1, p53, TNF-alpha,NF-κB, Fas / FasL and granzyme B pathways[7, 13].Others assume that OLP 's inflammatory pathways activate T-cells to release reactive oxygen species (ROS) alone or to enhance ROS production by activating keratinocytes, causing damage to neighboring cells [7].

Interleukin and renin has been reported to play a potential pathogenic role in autoimmune disease.

In fact in the biopsies and serum of OLP patients, elevated levels of IL-17 have been identified. IL-17 can activate different cells to release potent inflammatory molecules, such as epithelial cells , fibroblasts, and chondrocytes.In response to stimulus, oral keratinocytes are able to produce renin[6]. The activated NF-kB pathway greatly increases the production of renin, which is dramatically enhanced in the epithelial layer and lamina propria of OLP keratinocytes. Immunohistochemistry staining data showed that renin was present in cytoplasm rather than the nucleus.In the microenvironment of inflamed tissues, some recent studies indicate that renin is strongly elevated, and RAS plays a role in activating Th17 cells and increasing the production of IL-17[6]

To induce CCL-20, IL-8, and TNF-a development, exogenous IL-17 has been documented in oral keratinocytes. Previous experiments have shown that elevated levels of IL-17 mRNA and protein are found in OLP relative to unaffected samples. There may be a crucial role in OLP disease in indicating IL-17.

Interestingly, in erosive OLP patients, serum IL-17 concentrations are higher than in the non-erosive subtype, indicating a favorable association between IL-17 levels and OLP severity[6] By destroying the extracellular matrix and causing apoptosis of oral keratinocytes, over expression of renin and IL-17 in OLP can influence the onset and pathogenesis of this disease[6]

Decreased renin development has been associated with genetic or chemical involvement of the NF-kB pathway. The active NF-kB pathway is identified as mediated by pro-inflammatory cytokines(6). Chinese researchers have found that the signaling of vitamin D / VDR inhibits the NF- ¤B pathway and also improves MicroRNA-26, 27 a / b production. As a consequence, epithelial cell apoptosis and the risk of OLP lesions are decreased[6]

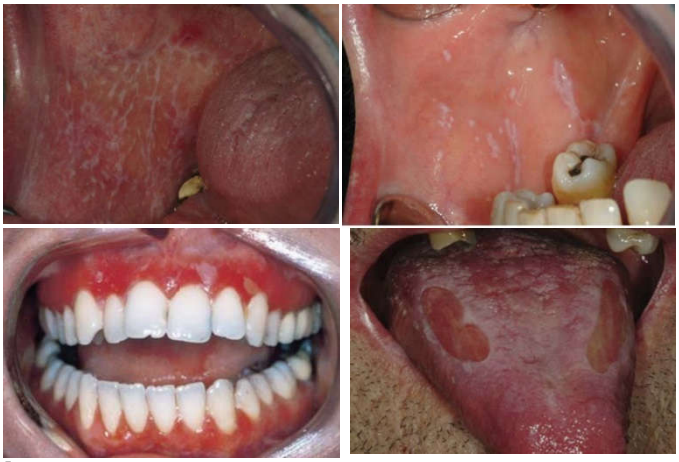
CLINICAL FEATURES:

Lichen planus is a distinct condition with normal colour, morphology and distribution, representing skin and mucosal lesions. LP affects about 0.5-2% of the general population , especially women, and occurs most frequently in middle age [1,20]Usually, numerous lesions occur in the disorder, often of bilateral and symmetric distribution.The classification of Andreasen distinguishes six OLP clinical manifestations, including reticular, plaquelike, atrophic (erythematous), erosive-ulcer and erosive[5,10]It may occur alone in the mucosa of the oral cavity, alone in the skin, concurrently in the oral cavity and skin, or in other extra-oral locations, such as the scalp, oesophagus, nails, and genitals.Erythematous lesions are frequently associatedwith reticular lesions, while both reticular anderythematous lesions are associated with erosive lesions in most cases. In addition, within the mouth, in the buccal and lingual mucosa and on the dorsal tongue, lesions are prominent [9]. Clinically, cutaneous. lichen planus is distinguished by purple, polygonal, pruritic papules on the flexor surface of the wrist,shins, trunk, and medial thighs, mostly hidden by Wickham striae [20]. In around one third of cases, a generalized incidence of oral frequently associated with discomfort and burning symptoms and can cause issues with feeding, communicating, and swallowing.Moreover with an expected risk of 0.52 percent, erosive longlasting OLP is associated with a substantial malignant transformation potential. Of all cases, there should be a differential diagnosis of burning mouth syndrome (BMS). Burning sensations and discomfort in the tongue and/or oral mucosa are characteristic signs of BMS. After ingestion of food and liquid, these symptoms may change, but are, however, constant for a duration of 4-6 months. Xerostomia, dysgeusia, metallic taste, mood swings, and variations in chemosensory perception are additional signs linked to BMS. The key distinction between OLP and BMS is that in patients with BMS , abnormal variations in the oral mucosa are not found [5] The reported incidence of malignant transformation of OLP ranged from 0 to 10 percent. A new metaanalysis has found that 1.1% of OLP lesions advance to OSCC, with a higher occurrence of cigarettes, alcohol users and hepatitis C virus infected individuals. Erosive OLP tends to be the form with the highestfrequency to advance to OSCC. Malignant transformation happens most often in lesions that are found onthe tongue.Muñoz et al. also shown that it takes an average of 5.5 years to turn OLP lesions into an existing OSCC [14]The clinical features are summerized in [Table 1.]

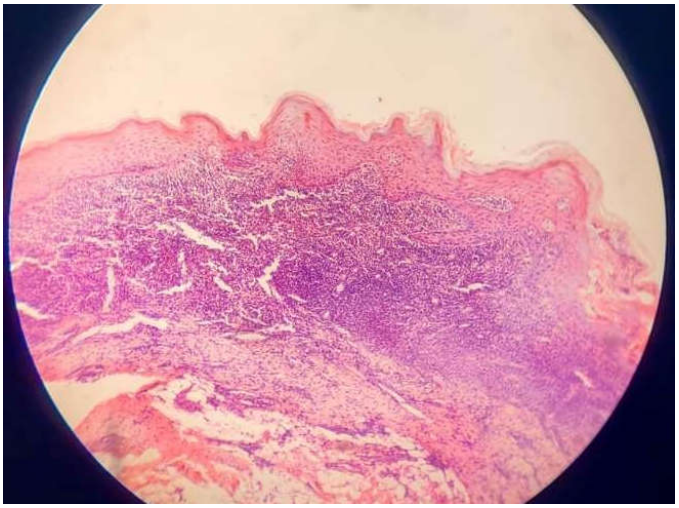
Table 1: Clinical features of oral lichen planus [22]

RETICULAR	Asymptomatic and appear as multiple papules with a network of small, raised, whitish-gray, lacy lesions referred to as Wickham striae.
EROSIVE	Erythema caused by inflammation or epithelial thinning with Wichkam striae
ULCERATIVE	Ulcerations are seen
PLAQUE LIKE	A white, homogeneous, slightly elevated, multifocal, smooth lesion.

The progression of OLP is marked by periods of recovery and exacerbation, where it will last several weeks or even months with both the signs and the symptoms. Classical cutaneous LP is self-limited and typically resolves within 6 (> 50 percent) to 18 months (85 percent). Some of the infected patients can be fully asymptomatic (about 20 percent). OLP diagnosis can be made clinically in many cases , particularly when viewed in reticular form.Leukoplakia subtype, erythroplakia, lichen sclerosis,pemphigoid lichen planus, lupus erythematosus, linear IgA syndrome, chronic ulcerative stomatitis, pemphigoid mucous membrane, and also the second stage of syphilis. A biopsy can be useful in such cases for histopathological evaluation and, if possible, for immunohistochemical analysis[1,16]



[Figure: Reticular, Plaque like , atrophic and erosive type of lichen planus from left to right and upper to lower]



Histopathology: Hyperkeratosis, basal layer vacuolization of apoptotic kertinocyes and a T cell infiltrate at the interface of the epithelium-connective tissue are the microscopic criteria for lichen planus. The epithelium undergoes progressive remodeling over time, resulting in decreased thickness and, rarely, a rete ridge resemble of saw tooth. There are growing numbers of T cells within the epithelium.

MANAGEMENT AND TREATMENT.

Management of OLP should be done in a systematic manner. The first step is to develop a history-based diagnosis, clinical evaluation and complex histo-pathology testing, direct immunofluorescence (DIF), indirect immunofluorescence (IIF), skin patch testing [21].The second stage is to advise the patient of the following: OLP is a persistent condition with predicted flare-up times and symptom-free periods; each patient will have variations in disease activity[21].The targets of therapy are to relieve unpleasant signs, eradicate ulcerative lesions, decrease the risk of oral cancer, prolong symptom-free times and promote proper oral and dental hygiene[21]. Owing to its recalcitrant disposition and also its idiopathic etiology, multiple drugs will need to be tried. At present, topical and systemic immuno- suppressants are typically administered to relieve clinical symptoms, but most of the cases of OLP tend to be more persistent and more resistant to treatment despite of many treatment modalities like topical and systemic steroids, anti-inflammatory coating gels, topical calcineurin inhibitors, retinoids and immunosuppressants. While some of these leave significant side effects such as dysgeusia, tachyphylaxis, oral mucosa thinning, adrenal suppression ,systemic absorption and secondary candidiasis up on long term management [3,5,6,10,11]. Hence numerous studies are being carried out throughout the world to find alternative treatments for OLP using non-pharmacological approaches and other therapeutic strategies.Topical steroids are considered as the first line of treatment as well as the gold standard in OLP treatment for decades[3,5,6,10,11]. In mild cases of OLP, topical steroids can be used but systemic corticosteroids must be given in cases where topical corticosteroid doesn't work, such as in recalcitrant, erosive and erythematous OLP. Here, 40-80 mg of prednisolone is prescribed for 5-7 days[5]. Some immunosuppressants and immunomodulatory agents are indicated in cases of contraindications for systemic steroids (breast-feeding, herpetic diseases, glaucoma, breastfeeding, HIV, asthma, diabetes mellitus, or hypertension): calcineurin inhibitors (cyclosporine, tacrolimus, pimecrolimus), mycophenolate mofetil, efalizumab[21]. Cyclosporine can be used as a mouth rinse. However due to its high cost, it should be reserved for recalcitrant cases. Tacrolimus is a more powerful inhibitor of calcineurin that can be used in the treatment of recalcitrant and erosive OLP safely and efficiently[5,21]. It has an immunosuppressive effect similar to cyclosporine and can readily penetrate into the mucosa. It is highly advised to use it two times/day (ointment of Protopic 0.1%) [21] for a limited period of time because of its ability to facilitate malignant transformation in long term use[21]When steroids are contraindicated, Dapsone, a steroid-sparing medication with reduced side effects (usual adult dose of 100 mg a day for 3 months), is an important medicine in erosive OLP[21]. In case of isolated plaques and non healing erosive OLP, surgical excision is recommended[5]. In clustered erosive and symptomatic OLP, free gingival grafts were used, indicating a complete eradication of the disease after 3.5 years[5] Micronutrients, like antioxidants, modify the role of the immune system and are perceived appealing substitutes of negligible side effects while handling OLP[11].Oral lichen planus being hypothesized to be an autoimmune disease, several researches have been carried out to treat lichen planus by micronutrients including antioxidants that modify the immune system function. Such studies involve the impact of vitamins A, D, E [3,11]

Vitamin A : Topical retinoids like tretinoin, isotretinoine or fenretinide have been reported to induce transient reversal of white striae in OLP. However, relative to topical cortocosteroids, topical retinoids are usually less effective since they are associated with side effects such as cheilitis and elevated serum triglyceride and liver enzyme levels[5,21]

Vitamin D: One study showed that subjects who took vitamin D supplements in addition to the routine treatment improved the clinical appearance of the lesion in the 1st week and completely disappeared on a period of 4 weeks. In another study of OLP treatment, 3 groups of patients were put on psychiatric counseling and vitamin D along with topical cortecosteroid. The result of this study showed that patients receiving vitamin D supplements with or without the psychiatric counseling, improved their symptoms[7].

Vitamin E : In another clinical trial, adjunctive systemic use of vitamin E with topical triamcinolone acetonide adhesive paste has demonstrated positive results without any side effects[11] .

There are some alternative non-pharmacological treatments available such as PRP, PDT, laser, and ozone therapy[5,21] recently emerged as a new and futuristic therapeutic modality, specially in severe cases and patients who are not reacting to conventional treatments[5]

In PRP(platelet rich plasma) patients own plasma is used for the treatment. PRP showed to be effective in decreasing the symptoms and improvement in clinical signs of OLP, which was resistant to conventional therapy[3]. A case report of erosive OLP which showed resistance to conventional therapy was efficiently treated by PRP. There was significant reduction in patient's symptoms and the clinical presentation of the lesion after 1st week and the lesion was completely regressed in terms of size and inflammation by the 4th week. On the follow up visit after 6 months, no recurrence of the lesion was observe[3] .The effect of PRP gel with cyclosporine mouthwash and retinoic acid lotion on various OLP phenotypes was contrasted in a pilot study. They concluded that PRP could be used in the erosive form, which proved to be successful once a week when implemented[3].

In Photodynamic therapy, a photosensitizing compound (methylene blue), triggered by laser light at a certain wavelength, is used in photodynamic therapy to kill the targeted cell using powerful oxidizers that trigger cell destruction, membrane lysis and protein inactivation, and has been successfully used to relieve OLP symptoms in adult patients [5,23].A systematic review of the effectiveness of (PDT) in symptomatic OLP treatment, however, revealed contradictory findings. Two trials, for instance, documented comparable efficacy of PDT and corticosteroids[24,25].

Laser treatment tends to be more effective in treating painful erosive OLP. In contrast with topical super-potent corticosteroid numerous experiments have documented the effect of laser therapy on erosive OLP, including the use of 980 nm laser diode [9,13,] carbon dioxide laser evaporation , pulsed diode laser biostimulation with 904 nm pulsed infrared rays, low-dose 308 nm laser and UV rays. Their consequence is the degradation of the superficial epithelium (containing protein denaturation of the target keratinocytes); in addition, the diode laser also kills the underlying connective tissue along the epithelium with the inflammatory compound[13]. While several studies have reported positive findings, the efficacy of laser therapy in OLP has yet to be proven[9]

Use of ozone (O3) has also gained a lot of interest in treating OLP. O3 is a very strong antioxidant that is being used as a disinfectant and a germicidal agent used in medical purposes. It also increases blood circulation and has healing effect[10,23,26].A case-controlled study showed that Compared to the group treated with corticosteroids alone, lesion size, Thongprasom score and discomfort dramatically reduced in the group treated with ozone and topical corticosteroids[10]. This was supported by another two studies carried out by Mostafa et al and Bayer et al (23,26). Ozonized water seems to be effective as an adjunct therapy, in combination with topical corticosteroids, for the treatment of eOLP[10]

Other natural agents like, lycopene, cur-cumin, purslane and aloe vera have also been assessed for management of OLP with varying outcomes[3,11]

CONCLUSION.

Oral lichen planus is a chronic inflammatory condition of uncertain etiology. Patients who have a history of chewing tobacco, areca nut chewing, smoking and alcohol consumption should be monitored constantly as there is a higher chance of the lesion to undergo malignancy specially in case of atrophic and erosive type. Depending on the form and severity of the lesion and doctor's own preference, the treatment could be non-surgical or surgical. However, more intensive studies must be carried out to find the best treatments which are cost-effective in the long run.

REFERENCES:

1. Ioannides D, Vakirlis E, Kemeny L, Marinovic B, Massone C, Murphy R, et al. European S1 guidelines on the management of lichen planus: a cooperation of the European Dermatology Forum with the European Academy of Dermatology and Venereology. Journal of the European Academy of Dermatology and Venereology. 2020 Jul 1;34(7):1403-14. https://doi.org/10.1111/jdv.16464 PMid:32678513
2. Talungchit S, Buajeeb W, Lerdtripop C, Surarit R, Chairatvit K, Roytrakul S, et al. Putative salivary protein biomarkers for the diagnosis of oral lichen planus: A case-control study. BMC Oral Health. 2018 Mar 13;18(1). https://doi.org/10.1186/s12903-018-0504-8 PMid:29534707 PMCid:PMC5851270
3. Shaik S, Jyothi PN, Kumar BV, Suman S v, Praveen KS, Sravanthi M, et al. Platelet Rich Plasma a New Prospective in Treatment of Recalcitrant Erosive Lichen Planus-A Case Report. Vol. 3, International Journal of Research and Reports in Dentistry. 2020.
4. Iocca O, Sollecito TP, Alawi F, Weinstein GS, Newman JG, de Virgilio A, et al. Potentially malignant disorders of the oral cavity and oral dysplasia: A systematic review and meta-analysis of malignant transformation rate by subtype. Vol. 42, Head and Neck. John Wiley and Sons Inc.; 2020. p. 539-55. https://doi.org/10.1002/hed.26006 PMid:31803979
5. Mester A, Lucaciu O, Ciobanu L, Apostu D, Ilea A, Campian RS. Clinical features and management of oral lichen planus (OLP) with emphasis on the management of hepatitis c virus (HCV)-related OLP. Vol. 18, Bosnian Journal of Basic Medical Sciences. Association of Basic Medical Sciences of FBIH; 2018. p. 217-23. https://doi.org/10.17305/bjbms.2018.3133 PMid:29984679 PMCid:PMC6087560
6. Ge X, Xie H, Nguyen T, Zhao B, Xu J, Du J. Renin Promotes STAT4 Phosphorylation to Induce IL-17 Production in Keratinocytes of Oral Lichen Planus. iScience. 2020 Apr 24;23(4).

https://doi.org/10.1016/j.isci.2020.100983
PMid:32213463 PMCid:PMC7093809

7. Paria Motahari, Fatemeh Pournaghi Azar, Arefeh Rasi. Role of Vitamin D and Vitamin D Receptor in Oral Lichen Planus: A Systematic Review. Ethiopian Journal of Health Sciences [Internet]. 2020 Jul 1;30(4). Available from: https://www.ajol.info/index.php/ejhs/article/view/199904 https://doi.org/10.4314/ejhs.v30i4.17
8. Qataya PO, Elsayed NM, Elguindy NM, Ahmed Hafiz M, Samy WM. Selenium: A sole treatment for erosive oral lichen planus (Randomized controlled clinical trial). Oral Diseases. 2020 May 1;26(4):789-804. https://doi.org/10.1111/odi.13285
PMid:31975475
9. Ge X, Yuan L, Wei J, Nguyen T, Tang C, Liao W, et al. Vitamin D/VDR signaling induces miR-27a/b expression in oral lichen planus. Scientific Reports. 2020 Dec 1;10(1). https://doi.org/10.1038/s41598-019-57288-9
PMid:31942011 PMCid:PMC6962379
10. Veneri F, Bardellini E, Amadori F, Conti G, Majorana A. Efficacy of ozonized water for the treatment of erosive oral lichen planus: a randomized controlled study. Medicina oral, patologia oral y cirugia bucal. 2020 Sep 1;25(5):e675-82. https://doi.org/10.4317/medoral.23693
PMid:32683383 PMCid:PMC7473429
11. Abdeldayem E, Mohamad W, Shaker O, Ali S. Effect of adjunctive systemic vitamin E on clinical parameters and salivary total antioxidant capacity in symptomatic oral lichen planus patients: Randomized controlled clinical trial. Advanced Dental Journal. 2020 Jan 1;2(1):24-33. https://doi.org/10.21608/adjc.2020.22386.1046
12. Parlatescu I, Tovaru M, Nicolae CL, Sfeatcu R, Didilescu AC. Oral health-related quality of life in different clinical forms of oral lichen planus. Clinical Oral Investigations. 2020 Jan 1;24(1):301-8. https://doi.org/10.1007/s00784-019-02951-8
PMid:31098713
13. Cassol-Spanemberg J, Rodríguez-de Rivera-Campillo ME, Otero-Rey EM, Estrugo-Devesa A, Jané-Salas E, López-López J. Oral lichen planus and its relationship with systemic diseases. A review of evidence. Journal of Clinical and Experimental Dentistry. 2018 Sep 1;10(9):e938-44. https://doi.org/10.4317/jced.55145
PMid:30386529 PMCid:PMC6203921
14. Uma Maheswari T, Chaudhary M. Management of oral Lichen Planus based on the existing clinical practice guidelines. Journal of Indian Academy of Oral Medicine and Radiology [Internet]. 2020;32(3):284. Available from: http://www.jiaomr.in/text.asp?2020/32/3/284/296587 https://doi.org/10.4103/jiaomr.jiaomr_55_20
15. Gabriella D, Klemens R, Xiao-hui RF, Corinna B, Eva H. Effect of personality traits on the oral health-related quality of life in patients with oral lichen planus undergoing treatment. Clinical Oral Investigations. 2020; https://doi.org/10.1007/s00784-020-03561-5
PMid:32929623
16. Robledo-Sierra J, van der Waal I. How general dentists could manage a patient with oral lichen planus. Medicina Oral Patologia Oral y Cirugia Bucal. 2018 Mar 1;23(2):e198-202. https://doi.org/10.4317/medoral.22368
PMid:29476684 PMCid:PMC5911349
17. Mehrbani SP, Motahari P, Azar FP, Ahari MA. Role of interleukin-4 in pathogenesis of oral lichen planus: A systematic review. Medicina oral, patologia oral y cirugia bucal. 2020 May 1;25(3):e410-5. https://doi.org/10.4317/medoral.23460
PMid:32134902 PMCid:PMC7211366
18. Peng Q, Yang JY, Zhou G. Emerging functions and clinical applications of exosomes in human oral diseases. Vol. 10, Cell and Bioscience. BioMed Central Ltd.; 2020. https://doi.org/10.1186/s13578-020-00424-0
PMid:32489584 PMCid:PMC7245751
19. de Lima SLG, de Arruda JAA, Abreu LG, Mesquita RA, Ribeiro-Rotta RF, Mendonça EF, et al. Clinicopathologic data of individuals with oral lichen planus: A Brazilian case series. Journal of Clinical and Experimental Dentistry. 2019;11(12):1-11. https://doi.org/10.4317/jced.56379
PMid:31824590 PMCid:PMC6894913
20. Mardani M, Torabi Ardakani S, Dastgheib L, Hamidizadeh N, Author C. Serum Levels of IL-22 in Patients with Oral Lichen Planus and Cutaneous Lichen Planus. Journal of Dentistry. 2020 Jan 11.
21. Rotaru D, Chisnoiu R, Picos A, Picos A, Chisnoiu A. Treatment trends in oral lichen planus and oral lichenoid lesions (Review). Experimental and Therapeutic Medicine. 2020 Oct 14;20(6):1-1. https://doi.org/10.3892/etm.2020.9328
PMid:33123228 PMCid:PMC7588785
22. Angelin D, Nair B. Comparative evaluation of survivin expression in leukoplakia, lichen planus, and oral squamous cell carcinoma: An immunohistochemical study. Journal of Cancer Research and Therapeutics. 2020 Apr 1;16(3):569-74. https://doi.org/10.4103/jcrt.JCRT_421_19
PMid:32719269
23. Al-Maweri SA, Ashraf S, Kalakonda B, Halboub E, Petro W, AlAizari NA. Efficacy of photodynamic therapy in the treatment of symptomatic oral lichen planus: A systematic review. Vol. 47, Journal of Oral Pathology and Medicine. Blackwell Publishing Ltd; 2018. p. 326-32. https://doi.org/10.1111/jop.12684
PMid:29350426
24. Bakhtiari S, Azari-Marhabi S, Mojahedi SM, Namdari M, Rankohi ZE, Jafari S. Comparing clinical effects of photodynamic therapy as a novel method with topical corticosteroid for treatment of Oral Lichen Planus. Photodiagnosis and Photodynamic Therapy. 2017 Dec 1;20:159-64. https://doi.org/10.1016/j.pdpdt.2017.06.002
PMid:28669793
25. Maloth KN, Velpula N, Kodangal S, Sangmesh M, Vellamchetla K, Ugrappa S. Photodynamic therapy - A non-Invasive treatment modality for precancerous lesions. Journal of Lasers in Medical Sciences. 2016;7(1):30-6. https://doi.org/10.15171/jlms.2016.07
PMid:27330695 PMCid:PMC4908982
26. Bayer S, Kazancioglu HO, Acar AH, Demirtas N, Kandas NO. Comparison of laser and ozone treatments on oral mucositis in an experimental model. Lasers in Medical Science. 2017 Apr 1;32(3):673-7. https://doi.org/10.1007/s10103-017-2166-1
PMid:28190112