Updated Pathogenesis and Treatment of Oral Lichen Planus

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ABSTRACT

Background: Oral lichen planus is chronic inflammatory illness of oral mucosa caused by T-cells. It is distinguished by durations of symptomatic exacerbation & remission, & cure focuses on decreasing inflammation & providing symptomatic relief.weshow numerous concepts in pathogenesis of OLP as well as current therapy modalities.**Summary**: OLP is oral dermatosis& one of common mucosal pathologies seen by dentists. It is critical that lesion be precisely recognized & that appropriate cure be administered as soon as possible. Understanding pathogenesis of disease is critical for providing appropriate cure.

Key words: Apoptosis, autoimmune, basal keratinocytes, corticosteroids, oral lichen planus.

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Introduction

LP is permenant inflammatory skin, hair follicle, nail, & mucosa disease. Oral, genital, ocular, otic, & esophageal surfaces are all impacted, as are bladder, nasal, laryngeal, & anal surfaces in rare cases. Skin & oral mucosa were most commonly impacted areas (1).

Oral variant, known as oral lichen planus, is chronic form with relapses & remissions that necessitates long-term symptomatic therapy as well as surveillance monitoring. Approximately fifteen percent of people with oral lichen planus improve cutaneous lesions, & twenty percent develop genital lesions (2).

Cutaneous involvement was typically self-limiting, with violaceous, pruritic papules covering reticular white striae known as Wickamstriae(3).

Women genital involvement manifests as erythema, erosion, white reticulated plaques, agglutination, labia resorption, & scarring. On glans penis, men could develop annular, papulosquamous lesions. Dyuria& dyspareunia can be associated symptoms (4).

Over quarter of OLP studied cases have oesophageal involvement, with symptoms including dysphagia & odynophagia. Friable mucosa, white plaques, erythema& stricture formation can be discovered during endoscopic test (5).

Etiology

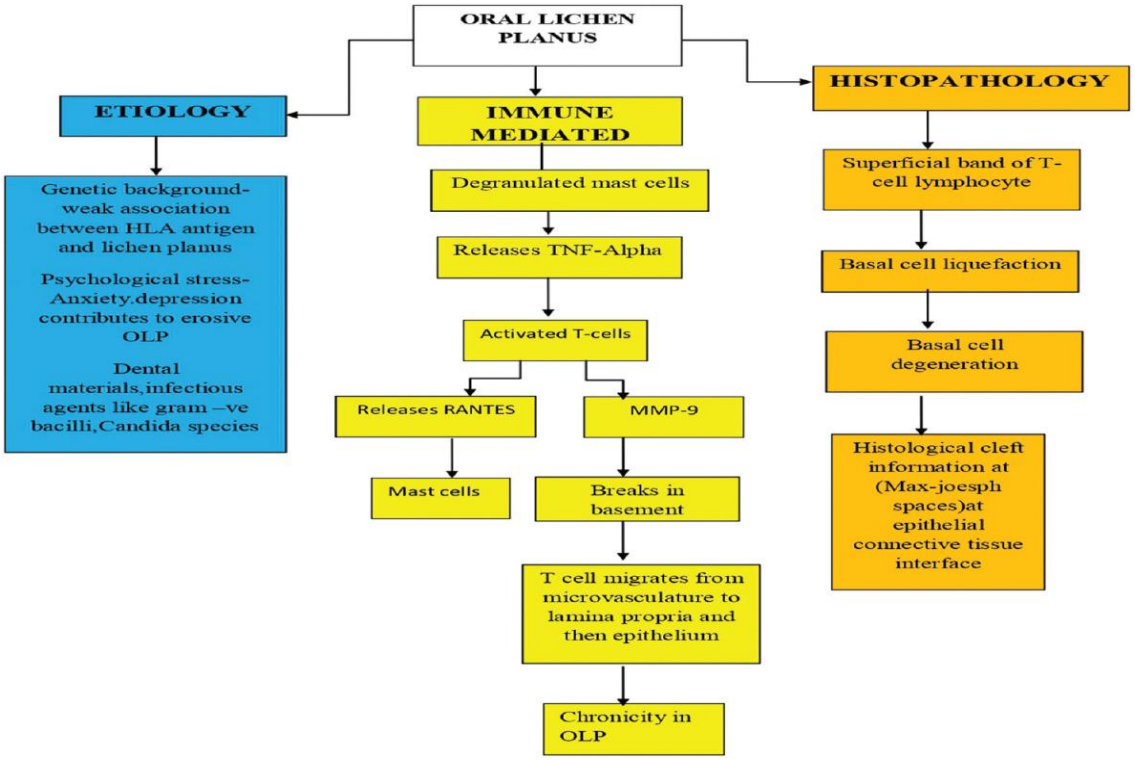

Figure 1: etiology & pathogenesis of OLP **(5)**

Many studies have been conducted to investigate aetiology& pathogenesis of OLP, & many antigen-specific & nonspecific inflammatory mechanisms were proposed to describe pathogenesis. Though often palliative, variety of therapy modalities is available, ranging from topical steroids to laser treatment (6).

Orallichenplanus is chronic inflammatory response mediated by T cells that affects oral mucosa. Other factors, like trauma& stress, appear to play role in exacerbating OLP symptoms (7).

Several researchers have found strong link among Hepatitis C virus infection & OLP in certain geographic areas, including Japan, Mediterranean region, & metropolitan population of United States (8).

Epidemiology

Global occurrence of oral lichen planus is estimated to be around two percent. It affects twice as many females as males & is most commonly diagnosed among 5th & 6th decades of life, even if it can affect children & young adults (9).

Pathogenesis

OLP is T-cell-mediated autoimmune illness wherein auto-cytotoxic CD8+ T cells kill oral epithelial basal cells (10).

Lichen planus antigen is unidentified, but it could be self-peptide, creating lichen planus true autoimmune illness. Keratinocyte antigen expression & unmasking at future lesion place can be caused by systemic drugs, contact allergens in dental restorative materials & toothpastes, mechanical trauma, bacterial & viral infection, & unknown agent (11).

Heat shock proteins were upregulated in OLP & werebelieved to be antigens, however their overexpression could also be common final route connecting number of exogenous

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agents in pathogenesis of OLP. Heat shock proteins expressed by oral keratinocytes could be auto-antigenic in OLP in this context. Susceptibility to OLP could be caused by dysregulated HSP gene expression in stressed oral keratinocytes & by incapability to control immune response following self-HSP recognition that ispossible because of reduced immune response(12).

According to non-specific mechanism, mast cell activation causes release of pro-inflammatory mediators like proteases & increasing of matrix metalloproteinases. This causes T cell infiltration of superficial lamina propria, basement membrane disruption, & keratinocyte apoptosis (13).

Chronic nature of OLP is caused by activation of nuclear factor kappa B& inhibition of changing growth factor control way (TGFβ -/Smad), which results in hyperkeratosis & presence of distinct white lesions. Genetic polymorphisms in first intron of IFNγ promoter gene were proposed as risk factors for evolving OLP (14; 15).

Histopathology

Attendance of band of lymphocytic inflammatory infiltrate in subepithelial connective tissue, hydropic degeneration of basal layer, & lack of epithelial dysplasia are histological criteria. If 3 of above criteria are met, lesion is regarded as typical lichen planus from histological standpoint; if 1 of histological criteria is not met, lesion is regarded histologically compatible with lichen planus(16).

It was later pointed out that several of these features are not unique to OLP. As result, American Academy of Oral & Maxillofacial Pathology recently suggested new set of diagnostic histopathologic criteria in attempt to exclude many of lichenoid mimics & enhance diagnosis accuracy. following criteria are now included in proposed criteria: band-like zone of lymphocytic infiltrate at epithelium-lamina propria interface, basal cell layer liquefaction, lymphocytic exocytosis, & lack of epithelial dysplasia & verrucous epithelial architectural changes (17).

Direct immunofluorescence analysis of samples from OLP studied cases reveals fibrinogen deposition in shaggy pattern straight basement membrane zone, but no immunoglobulins or complements. Because histologic outcomes were not particular to OLP, thorough history with clinical relationship is required toreach definitive diagnosis (18).

Living basal keratinocyte maintains basement membrane's normal integrity by secreting collagen four &laminin five inside epithelial basement membrane zone. Keratinocytes, in turn, necessitate cell survival signal deduced from basement membrane to avoid onset of apoptosis. Apoptotic keratinocytes were no longer capable of performing this function, resulting in basement membrane disruption (19).

Hepatitis c virus infection & oral lichen planus

Over ninety controlled researches from around world have found epidemiological evidence that Hepatitis C Virus can be etiologic factor in OLP. Association appears to be widespread in Southern Europe, Japan, & United States. Although, countries with greatest HCV occurrencearticle negative & non-significantassociations, implying that LP-HCV association maynot be described solely by greater incidence in population (20).

Recent evidence suggests link among hepatitis C virus-related CLD & oral lichen

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planus. HCV is hepatotropic, single-stranded RNA virus that was linked to extrahepatic manifestations & autoimmune diseases such as LP, polyarteritisnodosa, & erythema nodosum. In numerous Indian states, prevalence of HCV infection between overall populations varies from 0.1 to 7.9percent. Previous research from our country found no link among HCV infection & OLP (21).

Recent concepts in treatment

Oral lichen planus has no known cure. Primary aim of therapy is to decrease inflammation & relieve symptoms. First-line cure for OLP is topical corticosteroids, which may be applied like adhesive gel & used as mouth rinse. Topical agents are chosen over systemic agents because they are more effective & have fewer side effects. Whereas triamcinolone acetonide gel is commonly used to cure OLP studied cases, greater potency corticosteroids like clobetasol propionate arerealat providing symptomatic relief (22)

To ensure adequate contact time with oral mucosa, studied cases are instructed to dry oral mucosa before applying topical gel & to abstain from eating & drinking for thirty minute after application. Utilizing dexamethasone mouth rinses is especially beneficial for studied cases who have widespread oral lesions & when lesions are not easily available for topical gel application (23).

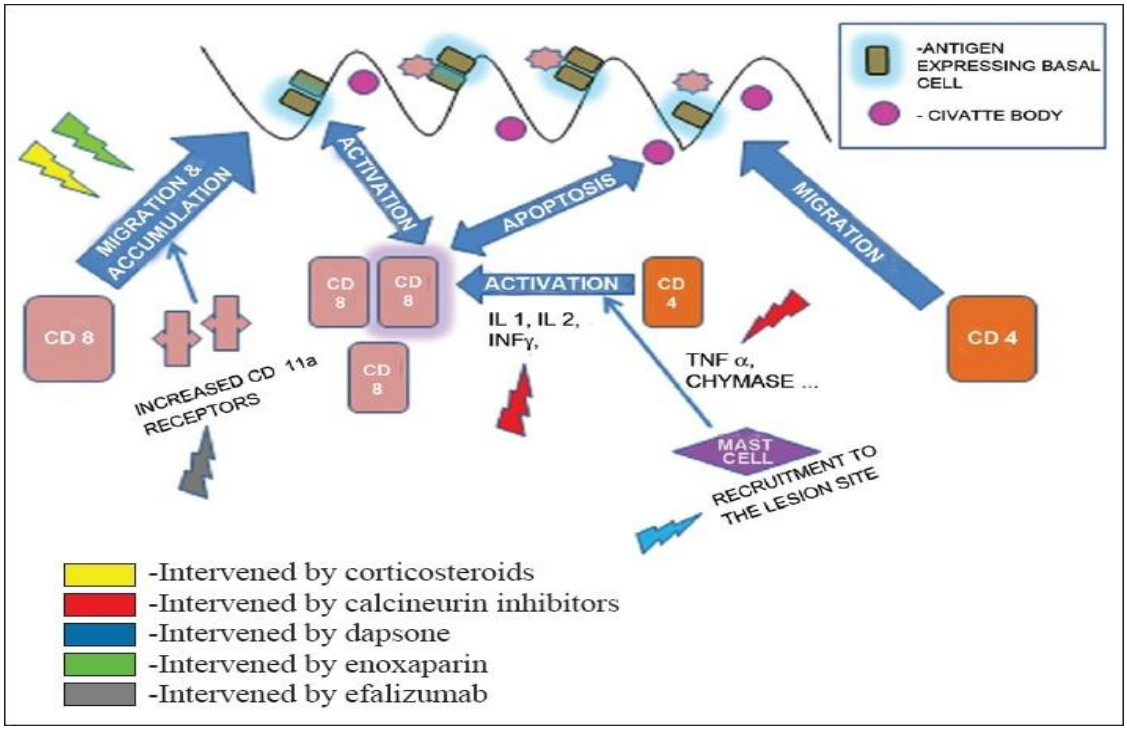

Figure 2: Depending on drug properties in oral lichen planus, proposed locations of action **(23)**

Corticosteroids

These are drugs that are most commonly used to treat OLP.Studied cases with severe forms of OLP are given great-potency &superpotent corticosteroids in the form of mouthwashes &intralesional injections. Long-term use of topical steroids mayconsequence in secondary candidiasis, which needs antifungal cure(24).While using superpotent steroids such as clobetasol, risk of tachyphylaxis& adrenal insufficiency is strong, particularly when used for extended duration. Systemic corticosteroids are used only in

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cases of recalcitrant erosive & erythematous LP where topical treatments failed. Drug of choice is systemic prednisolone, but it must be used at lowest probabledose for shortest amount of duration (forty–eighty mg for five–seven days) (25).

To cure persistent erosive OLP, intralesional corticosteroid injections can be used. Oropharyngeal candidiasis is side effect of topical corticosteroids. If required, clinicians could consider adjunctive treatment with topical/systemic antimycotics(26). Systemic corticosteroids were used to cure recalcitrant OLP that has failed to respond to topical treatments, serious OLP with widespread ulceration & erythema, & lichen planus with extra-oral involvement impacting numerous locations. As lesions heal or symptoms enhance, dosage & frequency of use can be lowered (27). Corticosteroids were mainstay of OLP management; however, other modalities such as mycophenolatemofetil, made significant contributions to disease's cure (28).

Other immune-suppressants &immune-modulatory agents

Calcineurin inhibitors

Cyclosporine, tacrolimus, &pimecrolimus are immunosuppressive drugs that inhibit calcineurin. These medications are known as calcineurin inhibitors (29).

Cyclosporine

Cyclosporine, calcineurin inhibitor, is immunosuppressant commonly used in post-allogenic organ transplantation to suppress studied case's immune system (30). In OLP, cyclosporine is used topically with adhesive bases or like mouth rinse. Even so, solution is expensive & must only be used in most stubborn cases of OLP. Systemic absorption is extremely low (31).

Tacrolimus

Tacrolimus, calcineurin inhibitor, is steroid-free topical immunosuppressive agent used to cure atopic dermatitis. It is ten-onehundred times more potent than cyclosporine & has larger percutaneous absorption. It was used in obstinate OLP cases. Streptomyces tsukubaensis produces this substance, which belongs to macrolide family (32). Tacrolimus has an immunosuppressive effect similar to cyclosporine, but it has larger ability to penetrate mucosa. Most common side effect noted is burning sensation; relapses of OLP after cessation were noticed (33).

Pimecrolimus

Novel treatments that are impactful & cause less morbidity are required. Anti-inflammatory action of calcineurin inhibitors provides rationale for using these topical agents in OELP studied cases, & many open-label researches using topical tacrolimus demonstrated efficacy. Few case reports & one recent comparative research have suggested that one percent pimecrolimus cream is impactful in OELP. Pimecrolimus absorption across human mucosa has not been thoroughly studied. Its use on OELP ulcerative lesions could result in significant systemic levels of drug (34).

Retinoids

Topical retinoids with immunomodulating properties, like tretinoin, isotretinoin, &fenretinide, were shown to be impactful in OLP. White striae may be reversed with topical retinoids, however impacts could be transient. Systemic retinoids were used with variable degrees of success in cases of serious lichen planus(35).

Dapsone

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Dapsone, an antimicrobial drug is also proved to have immunomodulatory properties.Dapsone is a widely used and economic drug which is used to treat leprosy. Dapsone is successfully used in therapy of lichen planus in few studies(36).

Mycophenolates

Mycophenolic acid was reintroduced into dermatological medicine after being used to cure psoriasis. It was used to cure serious cases of OLP because it is well-tolerated immunosuppressive drug used in organ transplantation. Mycophenolates are both expensive & effective when used long term (37).

Efalizumab

It is recombinant humanised monoclonal antibody that is used to cure psoriasis like immunosuppressant.

Efalizumab, monoclonal antibody to CD11a, binds to this adhesion molecule & improves OLP by decreasing T lymphocyte activation & trafficking. In vitro researches of mononuclear cells in OLP revealed that anti-CD11a antibodies reduced migration by peripheral blood mononuclear cells by sixty percent. It is given like subcutaneous injection once week. It is currently effective cure for plaque psoriasis (38).

Non-pharmacological modalities

- **PUVA treatment**

Several OLP studied cases are resistant to all available treatments. In cure of lichen planus, photodynamic treatment was used as alternative way. Local UVB phototherapy could be impactful therapy option for erosive OLP(39).

- **Photodynamic treatment**

Photodynamic treatment is method that employs photosensitizing compound,PDT was for some success in oncology, most notably in head & neck tumours. PDT has been shown to have immunomodulatory impacts & to encourage apoptosis in hyperproliferating inflammatory cells discovered in psoriasis & lichen planus. This has potential to opposite lichen planushyperproliferation& inflammation (40).

- **Laser treatment**

Lasers have lately been used in cure of oral lichen planus as they enhance wound healing efficacy & remove potential side effects caused by drugs. In OLR studied cases, diode laser (940 nm) is very effective in providing symptomatic relief of burning sensation (41).

By causing protein denaturation, all lasers damage superficial epithelium having target keratinocytes. Little researches that have been documented display lots of promise, & yet their efficacy has yet to be confirmed. There is no treatment for OLP; purpose of cure for symptomatic studied cases is palliation (42).

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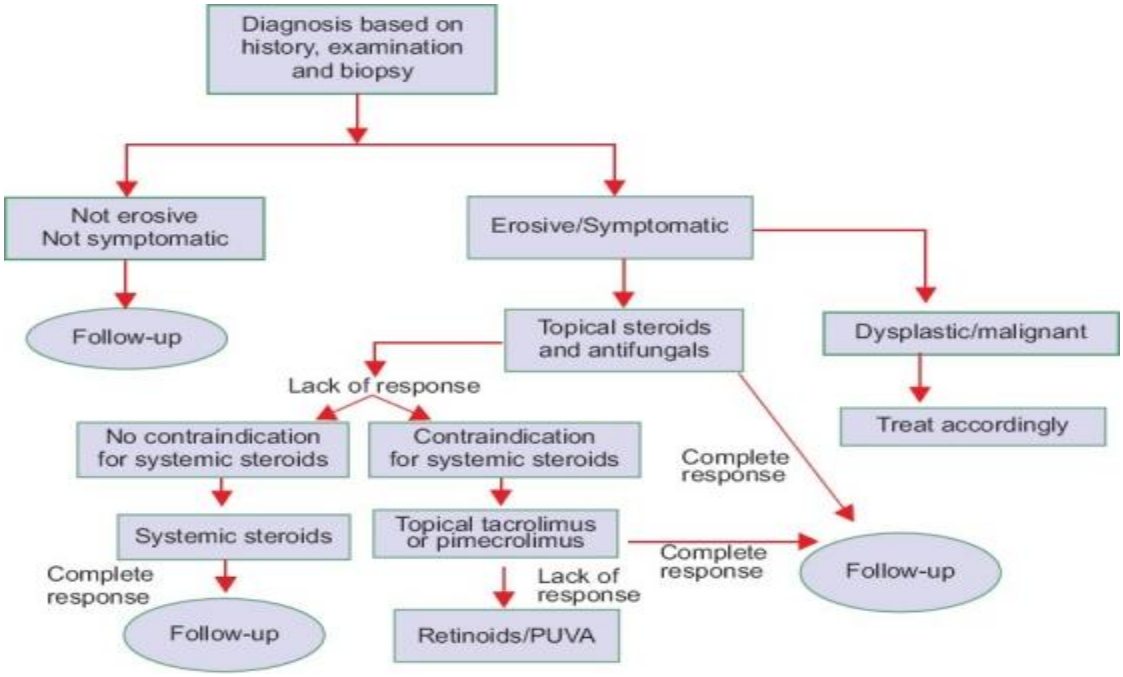

Figure 3: Protocol/algorithm for therapy of oral lichen planus (6)

American Academy of Oral Medicine (43)

1. Studied cases with OLP or OLL must be supervised by experienced clinician on regular basis for possible malignant & premalignant lesions, & any suspicious regions must be biopsied.
2. Studied cases must be counselled about low however potentially raised risk of oral cancer at time of diagnosis, so as to they understand that periodic tests are required if studied cases are asymptomatic & their symptoms are managed.
3. AAOM heavily supports development of multicenter, prospective researches of malignancy & OLP to better understand risk of oral cancer improving in studied cases with OLP &OLL, as well as subsets of studied cases who are most at risk of developing oral cancer.

Deterrence & studied case Education

Studied cases who have been diagnosed with OLP must be notified that therapy is not curative and is only intended to provide symptomatic relief. Change in lifestyle, like avoiding acidic & spicy foods, can help alleviate symptoms(44).

To emphasise importance of long-term clinical follow-up, it is essential to highlight chance of malignant transformation. Regular studied case self-monitoring can be helpful in detecting any suspicious modifications, like persistent oral ulcers & growths (45).

Improving Performance of Healthcare Teams

Oral lichen planus can be difficult to diagnose because studied cases can show non-specific clinical & histologic characteristics that overlay with number of situations. To arrive at accurate diagnosis, thorough history taking & clinical & histopathological test are required. Studied cases with OLP are typically cared for by dentist or oral surgeon (46).

Conclusion

Accurate clinical & histopathological test of Oral lichen planus will result in definitive diagnosis. Oral lichen planus cure must be tailored to the individual. There is no such thing as universal

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therapy protocol. It is essential to find recent clinical trials in order to cure oral lichen planus effectively. Early detection & treatment will aid in prevention of improvement of oral squamous cell carcinoma.

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